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MOBILITY DECISIONS, ECONOMIC DYNAMICS AND EPIDEMIC

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Abstract. In this paper we propose a theoretical model including a susceptible-infected-recovered-dead (SIRD) model of epidemic in a dynamic macroeconomic general equilibrium framework with agents' mobility. The latter affect both their income (and consumption) and their probability of infecting and of being infected. Strategic complementarities among individual mobility choices drive the evolution of aggregate economic activity, while infection externalities caused by individual mobility affect disease diffusion. Rational expectations of forward looking agents on the dynamics of aggregate mobility and epidemic determine individual mobility decisions.

The model allows to evaluate alternative scenarios of mobility restrictions, especially policies dependent on the state of epidemic.

We prove the existence of an equilibrium and provide a recursive construction method for finding equilibrium(a), which also guides our numerical investigations.

We calibrate the model by using Italian experience on COVID-19 epidemic in the period February 2020 - May 2021. We discuss how our economic SIRD (ESIRD) model produces a substantially different dynamics of economy and epidemic with respect to a SIRD model with constant agents' mobility. Finally, by numerical explorations we illustrate how the model can be used to design an efficient policy of state-of-epidemic-dependent mobility restrictions, which mitigates the epidemic peaks stressing health system, and allows for trading-off the economic losses due to reduced mobility with the lower death rate due to the lower spread of epidemic.

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1. Introduction

We propose an integrated assessment model, denoted by ESIRD, encompassing a susceptible-infected-recovered-dead (SIRD) model of epidemic and a dynamic macroeconomic general equilibrium model of economy, where mobility choices of forward looking agents affect both income (and consumption) and the spread of epidemic. A calibrated version of the model illustrates the possibilities to use the model to design an efficient policy of state-of-epidemic-dependent mobility restrictions.

Pandemic crisis has shown that sudden drops in individual mobility have a substantial negative consequences on aggregate income and consumption (OCDE, 2020). The decrease of individual mobility along COVID-19 crisis has been the joint outcome of individual decisions, caused by the diffusion of infection, and of containment measures imposed by national authorities (lockdown, curfew, etc.). In turn, a reduction in individual mobility brings down individual income (Huang *et al.*, 2020) as well as epidemic dynamics, being higher individual mobility associated to a higher probability of infecting and being infected (Nouvellet *et al.*, 2020). Therefore, entangled externalities and "general equilibrium" effects are at work; more precisely, individual mobility decisions display i) strategic complementarities with mobility choice of other agents, because the marginal impact on individual income of individual mobility is increasing in the aggregate mobility (Bulow *et al.*, 1985; Cooper & John, 1988); and, ii) negative externalities on contagion dynamics, because of agents in their mobility choices internalize the risk of being infected but not the effect of infecting other people (Bethune & Korinek, 2020).[1] So far, within the recent dynamic micro-funded epidemiological-economic literature (see, e.g., Eichenbaum *et al.*, 2020a; Toxvaerd, 2020; Jones *et al.*, 2020) no contribution has characterized in a dynamic macroeconomic general equilibrium model the optimal mobility choices in presence of strategic complementarities in production and negative externalities on contagion dynamics, the first step in a more adequate evaluation of policies reducing individual mobility for contrasting COVID-19 epidemic.

In the model we focus on short-term mobility. It should be meant as the daily/weekly activities of mobility observed in labour market, i.e. commuting, movements by car, track, bus, train, etc., generally observed in the economy. This mobility also includes movements for the activity of consumption, both for purchasing goods and services both for leisure.

Epidemic dynamics is driven by a generalized version of the SIRD model where the average number of contacts per person per time is endogenous, as well as the transition rate, i.e. the flow of new infected, and depends on the mobility choices of agents.

Agents maximize an inter-temporal discrete time utility function taking into account consumption and mobility costs. Their choice of mobility for working (respectively for consuming) depends on their state (susceptible, infectious or recovered), the aggregate level of economic activity, the current and future policies on mobility restrictions, and on their future utility,

[1]Another possible source of externality, the healthcare congestion, is analysed by Jones *et al.* (2020).

which, in turn, depends on the probabilities of being infected in the future and on the future dynamics of economy. In each period aggregate economic activity (consumption) depends on the state of the epidemic and on the individual mobility choices.

We set the agent's problem as a game with a continuum of players in a finite state space (the four states of agents). The notion of equilibrium is basically borrowed – even if re-elaborated – from Jovanovic & Rosenthal, 1988 (see their Definition 4.2) and shown to be equivalent to the notion of Nash equilibrium (Proposition 4.3). We provide an existence result (Theorem 4.5), and then propose an algorithm to identify an equilibrium (see Section 4.3 and Theorem 4.6).

The model can be seen as a discrete time, finite state, infinite horizon Mean Field Game (MFG). The latter studies the behavior of Nash equilibria in differential games as the number of agents becomes large. There is extensive recent research activity on MFGs starting from the pioneering works of Lasry and Lions (Lasry & Lions, 2006a,b, 2007). In the large population limit, one expects to obtain a game with a continuum of agents where, like in our case, the effects on the decision of any agent from the actions of the other agents are experienced through the statistical distribution of states. Since perturbations from the strategy of an agent do not influence the statistical states' distribution, the latter acts as a parameter in each agent's control problem. The passage to the limit is still a difficult theoretical topic and it is out of the scope of this paper (see, e.g., Carmona & Delarue, 2018).

We calibrate the model by using Italian experience on COVID-19 epidemic in the period February 2020 - May 2021. Numerical explorations of model under different configurations of state-of-epidemic-dependent mobility restrictions highlight the presence of a trade-off between economic losses and fatalities due to pandemic, i.e. of a pandemic possibilities frontier as in Kaplan *et al.* (2020) and Acemoglu *et al.* (2020). However, we argue that policy evaluation should take into account two additional directions, the first relate to the share of susceptible at the end of period of evaluation, which can favor a fresh outbreak of epidemic in the future without an efficient vaccine; and the social feasibility of prolonged mobility restrictions (Vollmer *et al.*, 2020).

Our paper makes four main contributes to literature. The first is to the epidemiological-macroeconomic literature, which has recently received a burst from the COVID-19 outbreak. Its main goal is to produce integrated assessment models, where the economic dynamics complements epidemiological models. In particular, a strand of literature focuses on optimal policy problem from a planner's perspective without modeling individual behavior (see, e.g., Alvarez *et al.*, 2020; Piguillem *et al.*, 2020; Moser & Yared, 2020; Atkeson, 2020), while another one considers forward-looking agents and market determination of good and factor prices, as in Eichenbaum *et al.* (2020a), Toxvaerd (2020), Jones *et al.* (2020) and Kaplan *et al.* (2020). With respect to these contributions we explicitly consider agents' (short-term) mobility. There are several good reasons for doing this: (i) in the epidemiological literature mobility is (not surprisingly) identified as the key variable in containing the epidemic (Nouvellet *et al.*, 2020);

(ii) mobility is an easily measurable variable and many datasets are actually freely available (e.g. GSM mobility data, Google Mobility Trends, and Apple Mobility Lab); therefore, it is ideal for bringing the model to the data; and, (iii) since mobility was/is the primary focus of several restrictive policies imposed by governments, in the proposed framework it is particularly natural to evaluate past and future policies on mobility restrictions. Focusing on mobility implies, as already argued, taking into account non-market interactions among individual choices; this leads to an another element of novelty in our epidemiological-macroeconomic model: the presence of strategic complementarities in individual decisions. The latter introduces substantial difficulties in the mathematical study of the model, which we start addressing proving the existence of a Nash equilibrium.

The second contribution is on methodological side. We have discussed above that our model belongs to the class of discounted infinite horizon, discrete time, finite state space MFG. So far, to the best of our knowledge our model does not fall into the classes already studied in the literature. In particular, Gomes *et al.* (2010), Doncel *et al.* (2019), Hadikhanloo & Silva (2019), and Bonnans *et al.* (2021); Wiecek (2020) deal with MFG in discrete time and finite state space. However, Hadikhanloo & Silva (2019) and Bonnans *et al.* (2021) consider only finite horizon problems; Gomes *et al.* (2010) (and similarly Wiecek (2020)) consider infinite horizon problems of ergodic type or with entropy penalization, where the dependence of the agents' utility from the choices of the other agents is more regular than in our model, and this allows them to prove existence and uniqueness of equilibrium. Finally, Doncel *et al.* (2019) consider an infinite horizon MFG, but where agents' cost does not depend on the strategies of the other agents, which instead happens in our model for the presence of strategic complementarities. Hence, our result of existence of an equilibrium and the verification type theorem are to be considered a novelty.

We also contribute to the theoretical economic literature focusing on the endogenous determination of the infection rate and the reproduction rate of an epidemic (Avery *et al.*, 2020). Infection rate depends on a large number of aggregate factors (climate, geography, health system, etc.), but also crucially revolves on individual choices. To endogenize infection rate has been proposed several approaches, among which a purely epidemiological approach as Fenichel (2013) and a behavioral approach (see Engle *et al.*, 2020a and Bisin & Moro, 2021). Farboodi *et al.* (2020), Toxvaerd (2020), and Eichenbaum *et al.* (2020a) are instead more in line with our approach, developing a settings where forward-looking individuals chose their actions facing a epidemic-economic trade-off. However, no paper directly models mobility choices of individuals taking into account strategic complementarities and negative externalities in an infinite horizon general equilibrium setting for explaining the dynamics of infection rate along the pandemic. The advantage of our approach are evident in the interpretation of results, allowing for directly correlating mobility and infection rate, and in the possibility to bring the model to data.

The final contribution is to the literature looking at the effect of epidemics diffusion on mobility (see, e.g, Meloni *et al.*, 2011 and Nouvellet *et al.*, 2020 for an epidemiological perspective). In particular, Engle *et al.* (2020b) and Simonov *et al.* (2020) look at the effects of governmental suggestions and Fox News messages respectively; while Goolsbee & Syverson (2021) and **?** try to disentangle the effects of individual decisions (auto-segregation) and governmental restrictive policies on mobility during the COVID-19 crisis. Our model provide a perfect candidate theoretical framework to analyse the observed mobility behavior since it characterizes in a dynamic general equilibrium setting individual decisions both in a policy-free regime (auto-segregation) or, alternatively, in a regime where restrictive measures on mobility are introduced. More important, our contribution provides a theoretical framework to evaluate restrictive policies going beyond the simple trade-off economic losses/fatalities as prospected in Kaplan *et al.* (2020), Acemoglu *et al.* (2020), and Gollier (2020). It makes it possible, for instance, to take into account in the evaluation other key dimensions regarding the social feasibility of policies, the fragility of post-lockdown situations with a high risk of fresh outbreaks, and the sustainability of health systems (see, in particular, Sections 5 and 6). It also allows to give an answer to the provocative question posed, among others, by Cochrane (2020) on the viability of a containment policy based only on self-confinement of individuals free of any governmental restrictions on mobility. At least for the Italian experience in 2020, our model suggests that a policy only based on self-confinement would have resulted in a peak prevalence of nearly six million infected people (see Section 6), which corresponds to a need of about four hundred thousand of beds in hospitals. This would have been unsustainable for a country having, in February 2020, about 190,000 beds in hospitals, most of them already occupied by patients with COVID-19 independent pathologies.

The paper is organized as follows: Section 2 presents the model, Section 3 focuses on the agent's optimization problem while Section 4 provides a recursive construction method for equilibrium(a). Section 5 calibrates the model to Italian data; Section 6 uses the model to investigate the effects of policies aiming at mitigating epidemic and their effects on economic activity; Section 7 concludes.

2. The epidemiologic-economic dynamic model

We consider an infinite horizon discrete time world with a continuum set of agents, whose individual actions do not modify the evolution of the global epidemic state. This is, roughly speaking, the typical setting that is used in the Mean Field Games theory, as discussed in the Introduction.

As in the classical SIRD framework (Chowell *et al.*, 2016), at each time period, the disease status k of an agent can be: susceptible ($k = S$); infected ($k = I$); recovered ($k = R$); and died ($k = D$). We then denote the set of possible disease status by $\mathbb{K}$, i.e.

$$\mathbb{K} := \{S, I, R, D\}\,.$$

2.1. **Mobility strategies and epidemic dynamics.** We want to model how the classes of disease, i.e., the shares of population with different disease status, evolve over time following the standard SIRD model *without vital dynamics* (newborns are not considered) according to the mobility choices of the classes. We assume that, at any time $t \in \mathbb{N}$, the agents of each epidemic class behave in the same way. Hence, we introduce the set

$$\mathcal{A} = \Big\{\Theta = (\Theta_p, \Theta_c) : \mathbb{N} \times \mathbb{K} \to [0,1]^2 \text{ s.t. } \Theta(\cdot, D, \cdot) = (0,0)\Big\}, \tag{1}$$

where $\Theta_p(t,k)$ and $\Theta_c(t,k)$ represent, respectively, the mobility rate (whose maximal value is w.l.o.g. normalized to 1) for production and for consumption chosen by the agents belonging to the class of disease $k \in \mathbb{K}$ at time $t \in \mathbb{N}$.

Consider the set $\mathcal{P}(\mathbb{K})$ of probability distributions on $\mathbb{K}$ and let $\mu(t) \in \mathcal{P}(\mathbb{K})$ represent the epidemic state at time $t \in \mathbb{N}$, i.e. $\mu(t)(\{k\})$ represents the shares of population in disease status $k \in \mathbb{K}$. The evolution of the epidemic depends on the mobility rates chosen by the agents, i.e. on Θ, as follows:

$$\mu(t+1) = [Q^{\Theta(t,\cdot)}(\mu(t))]^T \mu(t), \tag{2}$$

where $[\cdot]^T$ denotes transposition,

$$Q^{\Theta(t,\cdot)}(\nu) := \begin{pmatrix} 1-\tau^{\Theta(t,\cdot)}(\nu) & \tau^{\Theta(t,\cdot)}(\nu) & 0 & 0 \\ 0 & 1-\pi_R-\pi_D & \pi_R & \pi_D \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix}, \quad \nu \in \mathcal{P}(\mathbb{K}),$$

where $\pi_R, \pi_D \in (0,1)$ are two exogenous constants such that $\pi_R + \pi_D < 1$ and

$$\tau^{\Theta(t,\cdot)}(\nu) := \beta_P \nu(I) \Theta_p(t,I) \Theta_p(t,S) + \beta_C \nu(I) \Theta_c(t,I) \Theta_c(t,S)$$

where $\beta_P, \beta_C > 0$ are given constants such that $\beta_P + \beta_C < 1$. More explicitly, using the notation

$$\mu(t,k) := \mu(t)(\{k\}),$$

$$\begin{cases} \mu(t+1,S) = \mu(t,S)\big(1 - \beta_P \mu(t,I)\Theta_p(t,I)\Theta_p(t,S) + \beta_C \mu(t,I)\Theta_c(t,I)\Theta_c(t,S)\big), \\ \mu(t+1,I) = \mu(t,S)\big(\beta_P \mu(t,I)\Theta_p(t,I)\Theta_p(t,S) + \beta_C \mu(t,I)\Theta_c(t,I)\Theta_c(t,S)\big) \\ \qquad\qquad -\mu(t,I)(1-\pi_R-\pi_D), \\ \mu(t+1,R) = \mu(t,I)\pi_R + \mu(t,R), \\ \mu(t+1,D) = \mu(t,I)\pi_D + \mu(t,D). \end{cases}$$

The following remark explain the interpretation of the above equations.

Remark 2.1 *Denoting by $s(t), i(t)$, respectively, the percentage of population susceptible and infected at time t, we have $s(t) = \mu(t,S)$, $i(t) = \mu(t,I)$. Hence*

$$\Delta s(t) := s(t+1) - s(t) = \mu(t+1,S) - \mu(t,S) = -\beta(t) s(t) i(t)$$

where

$$\beta(t) := \beta_P \Theta_p(t, I)\Theta_p(t, S) + \beta_C \Theta_c(t, I)\Theta_c(t, S)$$

plays the role of the transmission rate of disease in the classical SIRD model. In our model it depends on the mobility choices of the infected and susceptible agents $\Theta_p(t, I)$, $\Theta_p(t, S)$, $\Theta_c(t, I)$ *and* $\Theta_c(t, S)$.

Remark 2.1 shows how our approach micro-funds the value of $\beta(t)$ (the average number of contacts sufficient for transmission of a person per unit of time), the key parameter of SIRD model. It allows on the one hand to distinguish the probability of contracting the virus in the workplace or by consuming and, on the other hand, it makes explicit the dependence of the probability of infection on the movements made to participate to the two activities.

It should be noted that the dependence of β on movements is "quadratic". This fact is quite natural since the probability of contracting the virus increases both because of the increased mobility of the agent i and for the increased mobility of other agents. Since in the model agents'behaviors are only determined by individual utilities, this will generate a first channel of strategic complementarity (with aggregate negative effects) in the mobility choices of agents. Agents will not completely internalize the aggregate contagion effects of their mobility choices and will have the tendency to move (and therefore spread the virus) "too much" with respect to the optimal level.

2.2. **Income and mobility.** We suppose that the income of agent depends on three elements: (i) her/his health conditions; (ii) her/his mobility choices to work; and, (iii) the general state of the economy. More precisely, at a generic period $t \in \mathbb{N}$, the income $Y(t, k)$ of the representative agent in the state $k \in \{S, I, R\}$, when the epidemic is in the state $\mu(t)$ and she undertakes the production mobility choice $\vartheta_p \in [0, 1]$, is given by

$$Y(t, k, \vartheta_p) = Z(t)\left(A_0(k) + A_1(k)\vartheta_p\right), \tag{3}$$

where

$$A_0(k) = \begin{cases} a_0^{SR} & \text{if } k \in \{S, R\} \\ a_0^{I} & \text{if } k = I \end{cases} \qquad A_1(k) = \begin{cases} a_1^{SR} & \text{if } k \in \{S, R\} \\ a_1^{I} & \text{if } k = I \end{cases} \tag{4}$$

and

$$Z(t) := \phi\Big(\mu(t, S)\Theta_p(t, S),\ \mu(t, I)\Theta_p(t, I),\ \mu(t, R)\Theta_p(t, R)\Big), \tag{5}$$

where $\phi : [0, 1]^3 \to (0, \infty)$ is non-decreasing in all the components and such that $\phi(0, 0, 0) = \varepsilon > 0$. We assume that

$$0 < a_0^I \leq a_0^{SR} \text{ and } 0 \leq a_1^I \leq a_1^{SR}, \tag{6}$$

where the second inequalities reflect the fact that that healthy (susceptible or recovered) agents are more productive than infected. Notice that in the agent's income Y, the only term depending on μ and Θ is Z.

2.3. **Consumption and mobility.** The price faced by the agent for the consumption good depends on her/his (consumption-related) mobility choice ϑ_c and it is given by:

$$P(t,\vartheta_c)=\frac{1}{P_0+P_1\vartheta_c}, \tag{7}$$

where

$$P_0,P_1\geq 0. \tag{8}$$

Abstracting from saving, at the generic time period, the consumption of the agent in the disease state k when the epidemic is in the state $\mu(t)$ and she undertakes the production-consumption choices $\vartheta=(\vartheta_c,\vartheta_c)$ is then given by

$$C(t,k;\vartheta) \;=\; \frac{Y(t,k,\vartheta_p)}{P(t,\vartheta_c)}=Z(t)\left(A_0(k)+A_1(k)\vartheta_p\right)\left(P_0+P_1\vartheta_c\right).$$

2.4. **Mobility costs .** We introduce now the mobility costs of agents. We assume that moving is costly. In particular, at a generic time period, the cost of the agent in the disease state k when the epidemic state is $\mu(t)$ to move with intensity ϑ_p (respectively, ϑ_c) in the labour market (respectively, in the consumption market) is

$$\gamma_p\left(k,\mu(t)\right)\vartheta_p\quad\Big(\text{respectively,}\quad \gamma_c\left(k,\mu(t)\right)\vartheta_c\;\Big), \tag{9}$$

where $\gamma_p:\mathbb{K}\times\mathcal{P}(\mathbb{K})\to(0,\infty)$ (respectively, $\gamma_c:\mathbb{K}\times\mathcal{P}(\mathbb{K})\to(0,\infty)$). We assume that:

$$\gamma_p(R,\nu)\leq\gamma_p(S,\nu)\leq\gamma_p(I,\nu),\qquad \gamma_c(R,\nu)\leq\gamma_c(S,\nu)\leq\gamma_c(I,\nu),\quad\forall\nu\in\mathcal{P}(\mathbb{K}). \tag{10}$$

2.5. **Agents' utility.** The utility at time t of the agent in the disease state $k\in\mathbb{K}$, undertaking the actions $\vartheta=(\vartheta_p,\vartheta_c)\in[0,1]^2$ is $u(t,k,\vartheta)$, where $u:\mathbb{N}\times\mathbb{K}\times[0,1]^2\to\mathbb{R}$ with $u(\cdot,D,\cdot)\equiv 0$ and, for $k\in\{S,I,R\}$,

$$u(t,k,\vartheta):=\ln\Big[Z(t)\left(A_0(k)+A_1(k)\vartheta_p\right)\left(P_0+P_1\vartheta_c\right)\Big]-\gamma_p\left(k,\mu(t)\right)\vartheta_p-\gamma_c\left(k,\mu(t)\right)\vartheta_c-M, \tag{11}$$

where $M\in\mathbb{R}$ is the constant utility of state dead, which "normalizes the utility of nonsurvival to zero" (Rosen, 1988, p. 2). In the expression of the utility the dependence on time only arises through $Z(t)$ and $\mu(t)$, hence we can write it as

$$u(t,k,\vartheta)=U(k,Z(t),\mu(t),\vartheta),$$

where

$$U:\mathbb{K}\times(0,\infty)\times\mathcal{P}(\mathbb{K})\times[0,1]^2\to\mathbb{R},$$

$$U(k,z,\nu,\vartheta)=\ln\Big[z\left(A_0(k)+A_1(k)\vartheta_p\right)\left(P_0+P_1\vartheta_c\right)\Big]-\gamma_p\left(k,\nu\right)\vartheta_p-\gamma_c\left(k,\nu\right)\vartheta_c-M. \tag{12}$$

Variables μ and Θ only arises in the terms Z and (γ_p,γ_c). Z depends on μ and Θ through (5), while μ depends on μ_0 and Θ through (2). Hence, we find useful to emphasize the dependence of u on μ_0 and Θ by writing $u^{\mu_0,\Theta}$.

3. The agent's optimization problem

To define and study the equilibria in the above setting, we first look at the optimization problem of an agent who may possibly deviate from the behavior of the others. Fix the initial epidemic distribution $\mu_0 \in \mathcal{P}(\mathbb{K})$ and the overall agents' behavior $\Theta \in \mathcal{A}$. Then, let $\mu(t)$, $t \in \mathbb{N}$, be evolving according to (2) with initial state $\mu(0) = \mu_0$. We assume, as it is natural in the context of a continuum of agents, that the mobility choices of the agent do not modify the evolution of μ. Hence, we consider an agent who takes for given μ_0 and Θ — hence, $\mu(\cdot)$, since her choices do not affect the evolution of μ — and deviates from Θ maximizing her/his expected total inter-temporal utility. Since we rely on Dynamic Programming, we let the initial time and state vary. Hence, we assume that the agent starts at time $t_o \in \mathbb{N}$ in the state $k_o \in \mathbb{K}$, where $(t_o, k_o) \in \mathbb{N} \times \mathbb{K}$, and that she chooses her strategies (in the closed-loop sense) in the set

$$\mathcal{A}_{\rm ag}(t_o) := \Big\{\theta = (\theta_p, \theta_c) : \{t_o, t_o+1, ...\} \times \mathbb{K} \to [0,1]^2 \text{ s.t. } \theta(\cdot, D) = (0,0)\Big\}. \tag{13}$$

The feedback strategies depend on the present time and state of the agent – the knowledge of μ and Θ is hidden in the dependence on time of θ. Given a discount factor $1-\rho \in (0,1)$, the expected total inter-temporal utility is given by taking the discounted sum over time of the instantaneous utility introduced above in (11):

$$J^{\mu_0,\Theta}(t_o, k_o; \theta) := \mathbb{E}\left[\sum_{t=t_o}^{\infty} (1-\rho)^{t-t_o} u^{\mu_0,\Theta}(t, K^\theta(t), \theta(t, K^\theta(t)))\right], \tag{14}$$

where the epidemic state of the agent $K^\theta(t)$ evolves randomly as follows. First, $K^\theta(t_o) = t_o$ and at each time $t \geq t_o$ the state at time $t+1$, i.e. $K^\theta(t+1)$, is determined by the transition kernel introduced below when the generic variable $\vartheta \in [0,1]^2$ is substituted by $\theta(t,S)$:

$$\mathcal{P}^\vartheta(t) := \begin{pmatrix} p^\vartheta_{SS}(t) & p^\vartheta_{SI}(t) & p^\vartheta_{SR}(t) & p^\vartheta_{SD}(t) \\ p^\vartheta_{IS}(t) & p^\vartheta_{II}(t) & p^\vartheta_{IR}(t) & p^\vartheta_{ID}(t) \\ p^\vartheta_{RS}(t) & p^\vartheta_{RI}(t) & p^\vartheta_{RR}(t) & p^\vartheta_{RD}(t) \\ p^\vartheta_{DS}(t) & p^\vartheta_{DI}(t) & p^\vartheta_{DR}(t) & p^\vartheta_{DD}(t) \end{pmatrix} = \begin{pmatrix} 1-\tau^\vartheta_{\rm ag}(t) & \tau^\vartheta_{\rm ag}(t) & 0 & 0 \\ 0 & 1-\pi_R-\pi_D & \pi_R & \pi_D \\ 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 1 \end{pmatrix}.$$

where

$$\tau^\vartheta_{\rm ag}(t) := \beta_P \mu(t,I)\Theta_p(t,I)\vartheta_p + \beta_C \mu(t,I)\Theta_c(t,I)\vartheta_c.$$

$\tau^\vartheta_{\rm ag}(t)$ is obtained by $\tau^{\Theta(t,\cdot)}$ substituting $\Theta(t,S)$ with ϑ, i.e. the probability of infection of the susceptible agent changes due her choice of deviating from $\Theta(t,S)$. Summarizing K^θ is the controlled Markov chain whose transition kernel at each time $t \geq t_o$ is $\mathcal{P}^{\theta(t,K^\theta(t))}(t)$, i.e. this matrix gives the transition probability of the state of the agent from time t to time $t+1$.[2] The agent aims at maximizing $J^{\mu_0,\Theta}(t_o,k_o;\theta)$ over $\theta \in \mathcal{A}_{\rm ag}(t_o)$. Define the value function of the agent as

$$V^{\mu_0,\Theta}(t_o,k_o) = \sup_{\theta \in \mathcal{A}_{\rm ag}(t_o)} J^{\mu_0,\Theta}(t_o,k_o;\theta).$$

[2]When $\theta = \Theta$, then the dynamics of μ is, as expected, the Fokker-Planck equation associated to the random dynamics of the agent.

It is well known, by the Dynamic Programming Principle, that the value function is a solution (possibly not unique) to the so called Bellman equation, which is written as follows (with unknown v):

$$v(t_o,k_o)=\sup_{\vartheta\in[0,1]^2}\sum_{k\in\mathbb{K}}p^{\vartheta}_{k_ok}(t_o)\big[u^{\mu_0,\Theta}(t_o,k_o,\vartheta)+(1-\rho)v(t_o+1,k)\big]. \tag{15}$$

4. Equilibrium: existence and recursive construction

4.1. **Definition of equilibrium.** Let us give the definition of Nash equilibrium for the continuum of agents' game.

Definition 4.1 (Nash equilibrium) *Let $\mu_0\in\mathcal{P}(\mathbb{K})$. A Nash equilibrium (for our continuum of agents' game) starting at μ_0 is a map $\overline{\Theta}^{\mu_0}\in\mathcal{A}$ such that, calling $\overline{\mu}(\cdot)$ the solution to (2) associated to $\overline{\Theta}^{\mu_0}$ and defining*

$$\overline{\theta}(t,k):=\overline{\Theta}^{\mu_0}(t,k),\qquad (t,k)\in\{t_o,t_o+1,...\}\times\mathbb{K},$$

we have

$$J^{\mu_0,\overline{\Theta}^{\mu_0}}(t_o,k_o)=V^{\mu_0,\overline{\Theta}^{\mu_0}}(t_o,k_o). \tag{16}$$

Inspired by Jovanovic & Rosenthal, 1988 we also give the following definition that will turn out to be equivalent to the latter one.

Definition 4.2 (Equilibrium) *Let $\mu_0\in\mathcal{P}(\mathbb{K})$ be a given initial distribution of the disease at time $t=0$. An equilibrium starting from μ_0 is a couple $(v^{\mu_0},\overline{\Theta}^{\mu_0})$, with $v^{\mu_0}:\mathbb{N}\times\mathbb{K}\to\mathbb{R}$ and $\overline{\Theta}^{\mu_0}\in\mathcal{A}$, such that:*

(i) *v^{μ_0} is bounded and satisfies for every $(t_o,k_o)\in\mathbb{N}\times\mathbb{K}$ the dynamic programming equation*

$$v(t_o,k_o)=\sup_{\vartheta\in[0,1]^2}\sum_{k\in\mathbb{K}}p^{\vartheta}_{k_ok}(t_o)\big(u^{\mu_0,\overline{\Theta}^{\mu_0}}(t_o,k_o,\vartheta)+(1-\rho)v(t_o+1,k)\big). \tag{17}$$

(ii) *Calling $\overline{\mu}(\cdot)$ the solution to (2) with initial datum μ_0 and with $\Theta=\overline{\Theta}^{\mu_0}$, for all $(t_o,k_o)\in\mathbb{N}\times\mathbb{K}$, the couple $\overline{\Theta}^{\mu_0}(t_o,k_o)\in[0,1]^2$ is an optimizer in the right hand side of (17).*

Proposition 4.3 *The two above definitions are equivalent.*

Proof. (a) Let $\mu_0\in\mathcal{P}(\mathbb{K})$, let $(v^{\mu_0},\overline{\Theta}^{\mu_0})$ be an equilibrium in the sense of Definition 4.2, and let $(t_o,k_o)\in\mathbb{N}\times\mathbb{K}$. Then, define

$$\overline{\theta}(t,k):=\overline{\Theta}^{\mu_0}(t,k),\qquad (t,k)\in\{t_o,t_o+1,...\}\times\mathbb{K},$$

By standard verification arguments in optimal control, it is clear that, since v^{μ_0} is bounded, it coincides with the value function of the agent and the control $\overline{\theta}\in\mathcal{A}_{\mathrm{ag}}(t_o)$ is optimal for the agent when the other agents follow the same feedback strategy. Hence, (16) is verified showing that $\overline{\Theta}^{\mu_0}$ is a Nash equilibrium in the sense of Definition 4.1.

(b) Let $\mu_0\in\mathcal{P}(\mathbb{K})$ and let $\overline{\Theta}^{\mu_0}$ be a Nash equilibrium in the sense of Definition 4.1 and consider the couple $(V^{\mu_0,\overline{\Theta}^{\mu_0}},\overline{\Theta}^{\mu_0})$. By the dynamic programming principle, $V^{\mu_0,\overline{\Theta}^{\mu_0}}(t_o,k_o)$ satisfies (17), so part (i) of Definition 4.2 is satisfied. Part (ii) of the same definition is satisfied by (16). □

4.2. **Existence of equilibria.** The main result of this subsection is the proof of the existence of an equilibrium. We shall make use of the following fixed point theorem.

Theorem 4.4 (Tikhonov's fixed point Theorem) *Let $\mathcal{V}$ be a locally convex topological vector space, let $\mathcal{Q} \subseteq \mathcal{V}$ be a nonempty compact convex set, and let $F : \mathcal{Q} \to \mathcal{Q}$ be a continuous function. Then F has a fixed point.*

Theorem 4.5 *An equilibrium exists for each $\mu_0 \in \mathcal{P}(\mathbb{K})$.*

Proof. Fix $\mu_0 \in \mathcal{P}(\mathbb{K})$. Consider the space of sequences

$$\mathcal{V} := \Big\{ q = (q_R, q_I, q_S, q_D) : \mathbb{N} \to \mathbb{R}^4 \Big\}$$

endowed with the topology of pointwise convergence. The latter is a locally convex topological vector space since the topology is induced by the family of seminorms

$$\mathbf{p}_t(q) = |q(t)|_{\mathbb{R}^4}, \quad t \in \mathbb{N},$$

where $q(t)$ is the $t-$th component of q. Then, consider

$$\mathcal{Q} := \Big\{ q = (q_R, q_I, q_S, q_D) : \mathbb{N} \to [0,1]^2 \times [0,1]^2 \times [0,1]^2 \times \{0\} \Big\} \subset \mathcal{V}.$$

$\mathcal{Q}$ is convex and, by Tikhonov's compactness Theorem, it is compact in $\mathcal{V}$. We consider the one-to-one correspondence $\mathcal{M} : \mathcal{Q} \to \mathcal{A}$ defined by

$$(\mathcal{M}q)(t,k) \equiv q_k(t), \quad (t,k) \in \mathbb{N} \times \mathbb{K}.$$

Let

$$F : \mathcal{Q} \to \mathcal{Q}, \qquad F(q)(t_o, k_o) := (\hat{\theta}_p(t_o, k_o; q), \hat{\theta}_c(t_o, k_o; q)), \qquad (t_o, k_o) \in \mathbb{N} \times \mathbb{K}.$$

where $(\hat{\theta}_p(t_o, k_o; q), \hat{\theta}_c(t_o, k_o; q))$ is the unique the maximizer over $[0,1]^2$ of

$$\vartheta \mapsto \sum_{k \in \mathbb{K}} p^{\vartheta}_{k_o k}(t_o) \big[u^{\mu_0, \mathcal{M}(q)}(t_o, k_o, \vartheta) + (1-\rho) V^{\mu_0, \mathcal{M}(q)}(t_o + 1, k) \big].$$

Clearly, if $\overline{q}$ is a fixed point of F, then $(V^{\mu_0, \mathcal{M}(\overline{q})}, \mathcal{M}(\overline{q}))$ is an equilibrium according to Definition 4.2. Fix $(t_o, k_o) \in \mathbb{N} \times \mathbb{K}$; given a sequence $(q^n) \subset \mathcal{Q}$ converging to $q \in \mathcal{Q}$, we have, $V^{\mu_0, \mathcal{M}(q_n)}(t_o, k_o) \to V^{\mu_0, \mathcal{M}(q)}(t_o, k_o)$. Consequently, by strict concavity and regularity of $u^{\mu_0, \mathcal{M}(q)}$, we also have the convergence

$$(\hat{\theta}_p(t_o, k_o; q_n), \hat{\theta}_c(t_o, k_o; q_n)) \to (\hat{\theta}_p(t_o, k_o; q), \hat{\theta}_c(t_o, k_o; q)).$$

This shows that F is continuous. We conclude by Theorem 4.4. □

4.3. **Recursive construction of equilibria.** In this section we present a recursive algorithm which allows to compute an equilibrium of our game. First we present the algorithm and then we prove, in Theorem 4.6, that, under suitable conditions, it provides an equilibrium.

In the algorithm we will build a couple $(\hat{v}, \hat{\Theta})$ which will be proved to be an equilibrium in the sense of Definition 4.2. We denote also by $\hat{\mu}$ the associated epidemic distribution. All the objects defined below depend on μ_0 but, to lighten the notation, we do not stress this dependence.

At time $t = 0$, we start with $\hat{\mu}(0) = \mu_0$ and with an arbitrary $\hat{v}(0, \cdot)$ such that $\hat{v}(0, D) = 0$. Then, at generic time $t \in \mathbb{N}$, having at hand the values of $\hat{v}(t, \cdot)$, $\hat{\mu}(t)$ and (for $t > 0$) $\hat{\Theta}(t-1, \cdot)$, we define $\hat{v}(t+1, \cdot)$, $\hat{\mu}(t+1)$ and $\hat{\Theta}(t, \cdot)$, as follows.

1. We set $\hat{v}(t+1, D) = 0$ and $\hat{\Theta}(t, D) = 0$.
2. For $k \in \{I, R\}$, we define, the couple

$$\Phi(k, \nu) = (\Phi_p(k, \nu), \Phi_c(k, \nu))$$

where[3]

$$\begin{cases} \Phi_p(k, \nu) := \left(\left(\dfrac{1}{\gamma_p(k, \nu)} - \dfrac{A_0(k)}{A_1(k)} \right) \vee 0 \right) \wedge 1, \\ \\ \Phi_c(k, \nu) := \left(\left(\dfrac{1}{\gamma_c(k, \nu)} - \dfrac{P_0}{P_1} \right) \vee 0 \right) \wedge 1, \end{cases} \tag{18}$$

and

$$\hat{\Theta}(t, k) := \Phi(k, \hat{\mu}(t)).$$

3. Given $\xi \in \mathbb{R}$, $\nu \in \mathcal{P}(\mathbb{K})$, we define

$$\hat{\vartheta}^S(\xi, \nu) = (\hat{\vartheta}^S_p(\xi, \nu), \hat{\vartheta}^S_c(\xi, \nu)) = \big((\tilde{\vartheta}^S_p(\xi, \nu) \wedge 1) \vee 0, \ (\tilde{\vartheta}^S_c(\xi, \nu) \wedge 1) \vee 0 \big),$$

where

$$\tilde{\vartheta}^S_p(\xi, \nu) = \frac{1}{\gamma_p(S, \nu) + (1-\rho)\beta_P \hat{a}(\nu)\xi} - \frac{A_0(S)}{A_1(S)},$$

$$\tilde{\vartheta}^S_c(\xi, \nu) = \frac{1}{\gamma_c(S, \nu) + (1-\rho)\beta_C \hat{b}(\nu)\,\xi} - \frac{P_0}{P_1},$$

where

$$\hat{a}(\nu) = \nu(I)\Phi_p(I, \nu), \quad \hat{b}(\nu) = \nu(I)\Phi(I, \nu), \tag{19}$$

and also define, for $k \in \mathbb{K}$, $\nu \in \mathcal{P}(\mathbb{K})$, $\vartheta^S \in [0, 1]$,

$$\hat{Z}(\vartheta^S, \nu) = \phi\Big(\nu(S)\vartheta^S, \ \nu(I)\Phi_p(I, \nu), \ \nu(R)\Phi_p(R, \nu) \Big).$$

4. Recalling the expression of U given in (12), we define, given $w_R, w_I \in \mathbb{R}$, $\xi \in \mathbb{R}_+$, $\nu \in \mathcal{P}(\mathbb{K})$,

$$\begin{cases} W(w_R, R, \nu, \xi) := \dfrac{1}{1-\rho} \Big(w_R - U(R, \hat{Z}(\hat{\vartheta}^S(\xi, \nu), \nu), \nu, \Phi(R, \nu) \Big), \\ W(w_I, w_R, I, \nu, \xi) := \dfrac{1}{1 - \pi_R - \pi_D} \left[\dfrac{w_I - U(I, \hat{Z}(\hat{\vartheta}^S(\xi, \nu), \nu), \nu, \Phi(I, \nu))}{1-\rho} - \pi_R\, W(w_R, R, \nu, \xi) \right]. \end{cases}$$

5. Given $w = (w_S, w_I, w_R) \in \mathbb{R}^3$, we consider the algebraic equation in the variable $\xi \in \mathbb{R}_+$

$$w_S = (1-\rho)W(w_I, I, \nu, \xi) + (1-\rho)\xi + f(S, \nu, \xi),$$

[3]Hereafter, given $a, b \in \mathbb{R}$, we denote $a \vee b = \max\{a, b\}$, $a \wedge b = \min\{a, b\}$.

where

$$f(S,\nu,\xi) = U(S,\hat{Z}(\hat{\vartheta}^S(\xi,\nu),\nu),\nu,\hat{\vartheta}^S(\xi,\nu)) - (1-\rho)\left(\beta_P\hat{a}(\nu)\hat{\vartheta}_p^S(\xi,\nu)+\beta_C\hat{b}(\nu)\hat{\vartheta}_c^S(\xi,\nu)\right)\xi. \tag{20}$$

For every $\nu\in\mathcal{P}(\mathbb{K})$, denote the set of solutions (possibly empty) to this equation by $\hat{\xi}(w,\nu)$. If $\hat{\xi}(w,\nu)$ is nonempty and a singleton, we can define

$$\tilde{\Phi}(t,S,\nu) := \hat{\vartheta}^S(\hat{\xi}(\hat{v}(t,\cdot),\nu),\nu).$$

6. Assume now that $\hat{\xi}(\hat{v}(t,\cdot),\hat{\mu}(t))$ is nonempty and a singleton, hence $\hat{\vartheta}^S(\hat{\xi}(\hat{v}(t,\cdot),\hat{\mu}(t)),\hat{\mu}(t))$ and $\tilde{\Phi}(t,S,\hat{\mu}(t))$ are well defined. We set

$$\begin{cases}\hat{v}(t+1,R) := W(\hat{v}(t,R),R,\hat{\mu}(t),\hat{\xi}(\hat{v}(t,\cdot),\hat{\mu}(t))),\\ \hat{v}(t+1,I) := W(\hat{v}(t,I),I,\hat{\mu}(t),\hat{\xi}(\hat{v}(t,\cdot),\hat{\mu}(t))),\\ \hat{v}(t+1,S) := \hat{\xi}(\hat{v}(t,\cdot),\hat{\mu}(t)) + \hat{v}(t+1,I),\end{cases}$$

and

$$\begin{cases}\hat{\Theta}(t,R) := \Phi(R,\hat{\mu}(t)),\\ \hat{\Theta}(t,I) := \Phi(I,\hat{\mu}(t)),\\ \hat{\Theta}(t,S) := \tilde{\Phi}(t,S,\hat{\mu}(t)) = \hat{\vartheta}^S(\hat{\xi}(t,\hat{\mu}(t)),\hat{\mu}(t)).\end{cases}$$

7. Finally, we define

$$\hat{\mu}(t+1) = [Q^{\hat{\Theta}(t,\cdot)}(\hat{\mu}(t))]^T\hat{\mu}(t).$$

8. Then, we repeat, recursively on time, the construction above.

Theorem 4.6 *Let μ_0 and $\hat{v}(0,\cdot)$ with $\hat{v}(0,D)=0$ be assigned. Consider the recursive construction of the objects above and assume that $\hat{\xi}(\hat{v}(t,\cdot),\hat{\mu}(t))$ is well defined for each $t\in\mathbb{N}$ and that the resulting $\hat{v}$ is bounded. Then the couple $(\hat{v},\hat{\Theta})$ is an equilibrium starting at μ_0 in the sense of Definition 4.2, hence of Definition 4.1.*

Proof. First of all we observe that, under the above assumptions, the sequence $(\hat{v},\hat{\Theta})$ is well defined by induction. Now we show that (i) and (ii) of Definition 4.2 hold for such sequence.
We preliminarily notice that, given $(t_o,k_o)\in\mathbb{N}\times\{S,I,R\}$, the function $[0,1]^2\to\mathbb{R}$, $\vartheta=(\vartheta_p,\vartheta_c)\mapsto u(t_o,k_o,\vartheta)$ is strictly concave in $[0,1]^2$, since

$$D_\vartheta u(t_o,k_o,\vartheta) = \left(\frac{A_1(k_o)}{A_0(k_o)+A_1(k_o)\vartheta_p}-\gamma_p(k_o,\mu(t_o)),\ \frac{P_1}{P_0+P_1\vartheta_c}-\gamma_c(k_o,\mu(t_o))\right),$$

and

$$D^2_\vartheta u(t_o,k_o,\vartheta) = \begin{pmatrix} -\dfrac{A_1(k_o)^2}{(A_0(k_o)+A_1(k_o)\vartheta_p)^2} & 0\\ 0 & -\dfrac{P_1^2}{(P_0+P_1\vartheta_c)^2}\end{pmatrix}.$$

Now we fix $t_o\in\mathbb{N}$ and show that $\hat{v}(t_o,\cdot)$ solves the dynamic programming equation on the various occurrences of $k_o\in\mathbb{K}$, where $\hat{\Theta}(t_o,\cdot)$ are the maximizers of the right hand side of (17) .

($k_o=D$) In this case the dynamic programming equation reduces to

$$v(t_o,D) = u(t_o,D,(0,0)) + (1-\rho)v(t_o+1,D) = (1-\rho)v(t_o+1,D). \tag{21}$$

It is clear that the above constructed $\hat{v}$ is always zero on D and hence satisfies the above equation. The maximizer is the unique admissible control $\hat{\Theta}(t_o,D) := (0,0)$.

$(k_o = R)$ In this case the dynamic programming equation reduces to

$$v(t_o, R) = \sup_{\vartheta \in [0,1]^2} \big(u(t_o, R, \vartheta) + (1-\rho) v(t_o + 1, R)\big) \tag{22}$$

$$= (1-\rho) v(t_o + 1, R) + \sup_{\vartheta \in [0,1]^2} u(t_o, R, \vartheta). \tag{23}$$

The optimization above leads to the unique maximum point

$$(\hat{\vartheta}_p, \hat{\vartheta}_c) = \big((\tilde{\vartheta}_p \wedge 1) \vee 0,\ (\tilde{\vartheta}_c \wedge 1) \vee 0\big),$$

where

$$\begin{cases} \tilde{\vartheta}_p = \dfrac{A_1(R) - \gamma_p(I, \mu(t_o)) A_0(R)}{\gamma_p(R, \mu(t_o)) A_1(R)} = \dfrac{1}{\gamma_p(R, \mu(t_o))} - \dfrac{A_0(R)}{A_1(R)}, \\ \\ \tilde{\vartheta}_c = \dfrac{P_1 - \gamma_c(R, \mu(t_o)) P_0}{\gamma_c(R, \mu(t_o)) P_1} = \dfrac{1}{\gamma_c(R, \mu(t_o))} - \dfrac{P_0}{P_1}. \end{cases}$$

We therefore get

$$v(t_o + 1, R) = \frac{v(t_o, R) - u(t_o, R, \hat{\vartheta})}{1 - \rho}.$$

This is exactly the expression of the first equation in point 6 above. Hence $\hat{v}$ satisfies the dynamic programming equation (17) in this case. The maximizer is $\hat{\vartheta}$ above which coincides with the expression of $\hat{\Theta}$ given in the fourth equation in point 6 above.

$(k_o = I)$ In this case the dynamic programming equation reduces to

$$v(t_o, I) = \sup_{\vartheta \in [0,1]^2} \Big(u(t_o, I, \vartheta) + (1-\rho)\left((1 - \pi_R - \pi_D) v(t_o + 1, I) + \pi_R v(t+1, R)\right)\Big) \tag{24}$$

$$= (1-\rho)\left((1 - \pi_R - \pi_D) v(t_o + 1, I) + \pi_R v(t+1, R)\right) + \sup_{\vartheta \in [0,1]^2} u(t_o, I, \vartheta). \tag{25}$$

The optimization above leads to the unique maximum point

$$(\hat{\vartheta}_p, \hat{\vartheta}_c) = \big((\tilde{\vartheta}_p \wedge 1) \vee 0,\ (\tilde{\vartheta}_c \wedge 1) \vee 0\big),$$

where

$$\begin{cases} \tilde{\vartheta}_p = \dfrac{A_1(I) - \gamma_p(I, \mu(t_o)) A_0(I)}{\gamma_p(I, \mu(t_o)) A_1(I)} = \dfrac{1}{\gamma_p(I, \mu(t_o))} - \dfrac{A_0(I)}{A_1(I)}, \\ \\ \tilde{\vartheta}_c = \dfrac{P_1 - \gamma_c(I, \mu(t_o)) P_0}{\gamma_c(I, \mu(t_o)) P_1} = \dfrac{1}{\gamma_c(I, \mu(t_o))} - \dfrac{P_0}{P_1}. \end{cases}$$

We therefore get

$$v(t_o + 1, I) = \frac{1}{1 - \pi_R - \pi_D} \left[\frac{v(t_o, I) - u(t_o, I, \hat{\vartheta})}{1 - \rho} - \pi_R v(t+1, R) \right].$$

This is exactly the expression of the second equation in point 6 above. Hence $\hat{v}$ satisfies the dynamic programming equation (17) in this case. The maximizer is $\hat{\vartheta}$ above which coincides with the expression of $\hat{\Theta}$ given in the fifth equation in point 6 above.

$(k_o = S)$ In this case the dynamic programming equation reduces to

$$v(t_o, S) = \sup_{\vartheta \in [0,1]^2} \Big(u(t_o, S, \vartheta) + (1-\rho)\left((1 - \tau^{\vartheta}_{\mathrm{ag}}(t_o)) v(t_o + 1, S) + \tau^{\vartheta}_{\mathrm{ag}}(t_o) v(t_o + 1, I)\right)\Big), \tag{26}$$

which can be rewritten as

$$v(t_o, S) = (1-\rho) v(t_o + 1, I) + (1-\rho)(v(t_o + 1, S) - v(t_o + 1, I)) \tag{27}$$

$$+ \sup_{\vartheta \in [0,1]^2} \Big(u(t_o, S, \vartheta) - (1-\rho) \tau^{\vartheta}_{\mathrm{ag}}(t_o)(v(t_o + 1, S) - v(t_o + 1, I))\Big), \tag{28}$$

Set $\xi := v(t_o+1,S) - v(t_o+1,I)$ and consider the optimization above in terms of the parameter $\xi \in \mathbb{R}_+$. The maximization leads to the unique maximum point

$$(\hat{\vartheta}_p(\xi), \hat{\vartheta}_c(\xi)) = \big((\tilde{\vartheta}_p(\xi) \wedge 1) \vee 0,\ (\tilde{\vartheta}_c(\xi) \wedge 1) \vee 0\big),$$

where

$$\tilde{\vartheta}_p(\xi) = \frac{1}{\gamma_p(S,\mu(t_o)) + (1-\rho)a(t_o)\xi} - \frac{A_0(S)}{A_1(S)}, \qquad \tilde{\vartheta}_c(\xi) = \frac{1}{\gamma_c(S,\mu(t_o)) + (1-\rho)b(t_o)\xi} - \frac{P_0}{P_1},$$

where

$$a(t_o) = \hat{a}(\mu(t_o)) = \mu(t_o,I)\hat{\Theta}_p(t_o,I) = \mu(t_o,I)\Phi_p(I,\mu(t_o)), \tag{29}$$

$$b(t_o) = \hat{b}(\mu(t_o)) = \mu(t_o,I)\hat{\Theta}_c(t_o,I) = \mu(t_o,I)\Phi_c(I,\mu(t_o)), \tag{30}$$

where $\hat{\Theta}(t_o,I)$ is defined in the previous item. Recalling the definition of f given in (20), we consider the algebraic equation in the variable $\xi \in \mathbb{R}$

$$v(t_o,S) = (1-\rho)v(t_o+1,I) + (1-\rho)\xi + f(S,\mu(t_o),\xi).$$

By assumption this equation has a unique solution $\xi(t_o) = \hat{\xi}(t_o,\mu(t_o))$ the unique solution to this equation. We get

$$v(t_o+1,S) = \hat{\xi}(t_o) + v(t_o+1,I).$$

This is exactly the expression of the third equation in point 6 above. Hence $\hat{v}$ satisfies the dynamic programming equation (17) in this case. The maximizer is $\hat{\vartheta}$ above which coincides with the expression of $\hat{\Theta}$ given in the sixth equation in point 6 above. □

5. Calibration of the model

In the calibration of the model we focus on the recent Italian experience for COVID-19. Italy was unfortunately the first Western country severely hit by COVID-19; the epidemic shock was sudden and unexpected as well as the deep impact on Italian mobility and production (see Figure 1 below). At the same time, Italy was also the first Western country to adopt strict restrictions in mobility in March 2020. Overall, this makes the Italian case particularly well-adapted to calibrate/estimate the relationship between mobility, production and dynamics of epidemic.[4]

The first step in the numerical calibration of the model is to specify the $Z(t)$ in (3). In order to take as small as possible the number of model's parameters, we consider the following one-parameter specification:

$$Z(t) \equiv 1 - \exp\left(-g\left[\Theta_p(t,S)\mu(t,S) + \Theta_p(t,I)\mu(t,I) + \Theta_p(t,R)\mu(t,R)\right]\right), \tag{31}$$

where g measures the sensitivity of individual income to aggregate mobility, i.e. the complementarities between individual and aggregate mobility in determining the level of individual income. In this respect we expect that g is greater than 0. Taking (31) into account, overall we have to set 19 parameters, which are listed in Table 1, together with their values and information on the methods used to their setting. Below we provide more details on the method used to set their values.

[4]Data and codes are available at `https://people.unipi.it/davide_fiaschi/ricerca/`.

Parameter	Meaning	Value	Method used to set the value
π_R	Daily probability of recovering when infected	0.07143	Taken from literature on COVID-19 (Voinsky *et al.*, 2020)
π_D	Daily probability of death	0.00052	Taken from literature on COVID-19 (Flaxman *et al.*, 2020)
β_P	The impact of mobility for production on infection	0.14606	Calculated on the base of an R_0 equal to 2.9 for Italy (`https://en.wikipedia.org/wiki/Basic_reproduction_number`) and on the fact that mobility of infected is on average 30% less as result of prevalence rate of symptoms of COVID-19 in infected people (Day, 2020)
β_C	The impact of mobility for consumption on infection	0.14606	Calculated on the base of an R_0 equal to 2.9 for Italy (`https://en.wikipedia.org/wiki/Basic_reproduction_number`) and on the fact that mobility of infected is on average 30% less as result of prevalence rate of symptoms of COVID-19 in infected people (Day, 2020)
ρ	Discount rate of utilities	0.000296	Taken from Laibson *et al.* (2018)
$\gamma_p(S)$, $\gamma_p(I)$, and $\gamma_p(R)$	Cost of mobility for production for different types of individuals in baseline scenario	0.29795, 0.42564, and 0.29795	Calibrated in order to have mobility and production equal to 1 in a free-epidemic economy for susceptibles and recovered and mobility equal to 0.7 for infected
$\gamma_c(S)$, $\gamma_c(I)$, and $\gamma_c(R)$	Cost of mobility for consumption for different types of individuals in baseline scenario	0.21375, 0.22840, and 0.21375	Calibrated in order to have mobility and production equal to 1 in a free-epidemic economy for susceptibles and recovered and mobility equal to 0.7 for infected
a_0^{SR} and a_0^I	Sensibility of individual income to aggregate mobility independent from individual mobility	0.70229 and 0.49160	For susceptible and recovered estimated from the relation between mobility and production in Italy in the period February 2020 - May 2021 (see Figure 1). For infected people calibrated at 70% of other individuals based on the prevalence of symptoms.
a_1^{SR} and a_1^I	Sensibility of individual income to individual mobility	0.29805 and 0.29805	Estimated from the relation between mobility and production in Italy in the period February 2020 - May 2021 setting mobility and production equal to 1 in a pre-epidemic economy (see Figure 1)
P_0 and P_1	Sensibility of individual consumption to individual mobility	0.47187 and 0.12828	Estimated from the relation between average propensity to consume and mobility for retail and recreation in Italy in the period February 2020 - May 2021
g	Sensibility of individual income to aggregate mobility	7.741615	Estimated from the relation from mobility and production in Italy in the period February 2020 - May 2021 (see Figure 1)
M	Utility to be dead	-1.30	Calibrated to avoid negative lifetime utility for each survival individual
$\mu(0,S)$, $\mu(0,I)$, and $\mu(0,R)$	Initial state of epidemic	$1 - 1/60.000.000$, $1/60.000.000$, and 0	Calibrate on an economy of 60 millions of individuals as Italy in 2020

TABLE 1. List of model's parameters, their values and notes on how they are calculated/calibrated/estimated.

5.1. **Calibration of the epidemiological parameters.** The calibration of the epidemiological parameters focuses on daily dynamics as standard in epidemiology (Ferguson *et al.*, 2020). Several studies provide basic information on COVID-19 main epidemiological characteristics. In particular, Voinsky *et al.* (2020) report that the average number of days for recovering from COVID-19 is 14, which implies $\pi_R = 0.07142$. Flaxman *et al.* (2020), instead, document an overall probability to die once infected of 0.94% in Italy and an average number of days from infection to death of 18, which implies $\pi_R = 0.00052$.

Finally, for setting β_P and β_C we assume that they are equal, so that observed infection rate is the product between β_P (β_C) and the average mobility of infected individuals once mobility of susceptible is normalized to one in an economy without infected (see System of (2)). Day (2020) report that the prevalence rate of symptoms of COVID-19 in infected people is about 30%, i.e. 70% of infected people are asymptomatic. Assuming that the latter maintain the same mobility, we set average mobility of infected individual 30% less than the one of susceptible. Since the observed infection rate can be expressed as $(\pi_D + \pi_R) R_0$, then $\beta_P = \beta_C = 0.7 (\pi_D + \pi_R) R_0 = 0.14606$ given a basic reproduction rate R_0 of COVID-19 equal to 2.9 for Italy.[5]

5.2. **Calibration of economic part.** The calibration of parameters governing the relationship between income and mobility are based on the Italian experience in the period February 15, 2020 - May 31, 2021 reported in Figure 1.

Italian economic activity as estimated by OECD Weekly Tracker of GDP growth[6] appears very correlated with mobility for workplaces as reported by Google Mobility Trend. [7] The strong drop in mobility in the period between February 23, 2020 and March 8, 2020 (almost -10%) well before the first introduction of mobility restrictions at national level in the week of March 8, 2020, supports our idea of an endogenous response of individual to epidemic evolution, which burst in Italy at the of February 2020. The severe restrictions on mobility imposed in two steps in March 2020 leaded to a drop in mobility and economic activity of about 70% and 25% with respect to reference period respectively. The relax in restriction in May 2020 led to a bounce back in both variables, but recover was not complete. In the autumn of 2020, as result of the second pandemic wave, Italy again experienced new mobility restrictions, with associated reduction in economic activity.

Normalizing economic activity and mobility to 1 in an economy with only susceptible and taking (3) and (31) to formulate a (nonlinear) relationship between mobility and economic activity, a nonlinear estimation procedure produces an estimate of g, a_0^{SR} and a_1^{SR} of 0.70229, 0.29805 and 7.74162 respectively. a_0^I and a_1^I are set to 0.49160 and 0.29805 to respect the assumption that mobility of infected individual is 70% of susceptible.

As regard P_0 and P_1, they are set observing that, according to (3) and (9), average propensity to consume can be expressed as a function of consumption mobility, P_0, and P_1. Taking as proxy for consumption mobility the mobility for retail and recreation from Google Mobility

[5] `https://en.wikipedia.org/wiki/Basic_reproduction_number`.

[6] `https://www.oecd.org/economy/weekly-tracker-of-gdp-growth/`.

[7] `https://www.google.com/covid19/mobility/`).

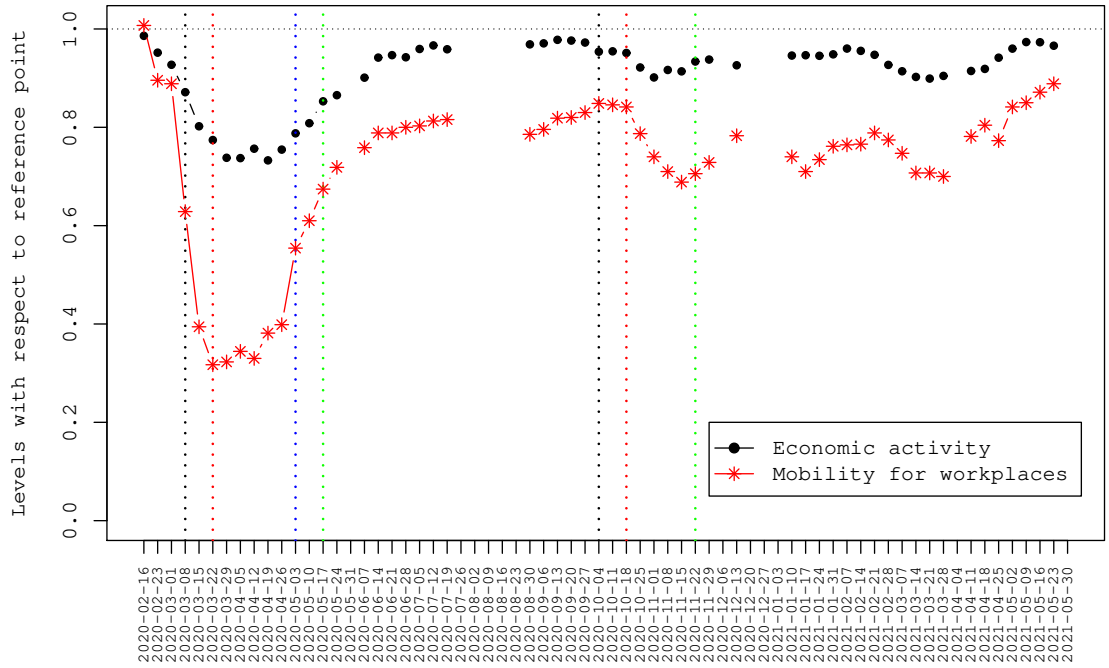


FIGURE 1. The relationship between weakly mobility for workplace and weakly economic activity in the period February 15, 2020 - May 31, 2021 (Italian holiday weeks are not reported). Dashed lines indicate weeks of new imposed mobility restrictions at national level (March 9, 202, March 22, 2020, October 8, 2020 and October 24, 2020) and of a relax in mobility restrictions (May 4, 2020, May 18, 2020, and November 24, 2020). *Source*: Google Mobility Trend (`https://www.google.com/covid19/mobility/`) and OECD Weekly Tracker of GDP growth (`https://www.oecd.org/economy/weekly-tracker-of-gdp-growth/`)

Trend[8] and the quarterly average propensity to consume from Italian national account, we estimate $P_0 = 0.47187$ and $P_1 = 0.12828$. Finally, the utility of state dead M is set equal to -1.3 to avoid that, independent of state of epidemic and economic activity, lifetime utility of survival individuals can be negative.

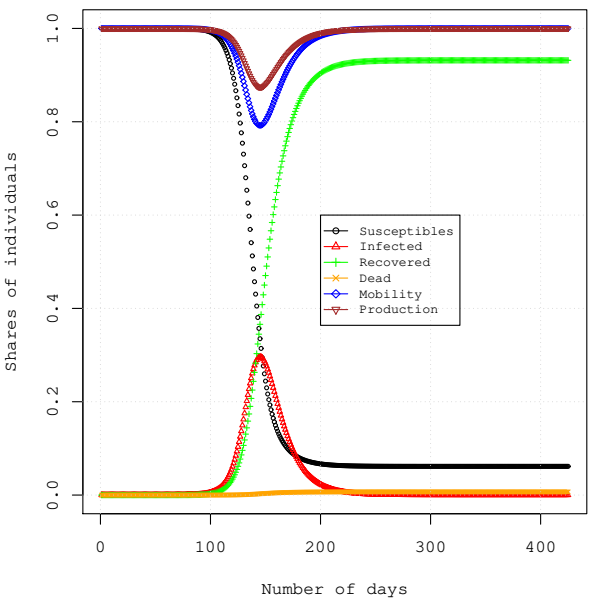
TABLE 2. "Dumb" SIRD versus economic SIRD (ESIRD) model with endogenous mobility. Numerical experiments based on the parameters reported in Table 1.

Model	Peak prevalence	Cumulative deaths	Minimum of production	Minimum of mobility	Economic loss	Mobility loss	$\mu_S(425)$	$\mu_I(425)$	$\mu_R(425)$	$\mu_D(425)$ (death rate)
Dumb SIRD	17,784,284	408,678	0.87	0.79	−0.011	−0.019	0.062	0.000	0.932	0.007
ESIRD	5,858,062	297,577	0.883	0.693	−0.032	−0.082	0.314	0.003	0.678	0.005

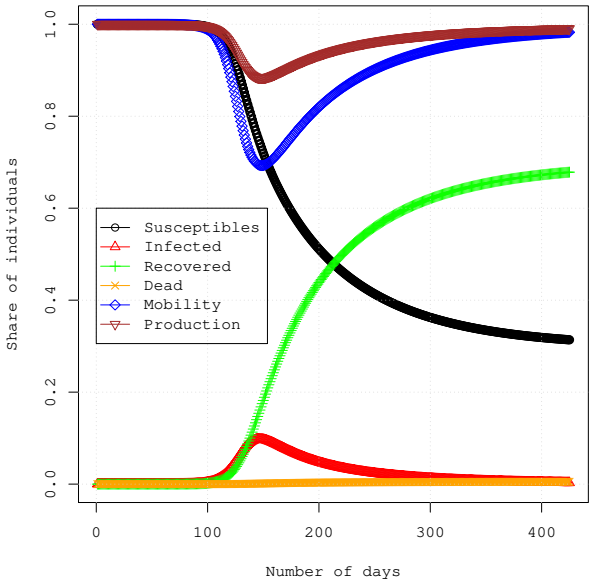
5.3. **"Dumb" SIRD versus economic SIRD (ESIRD) model.** Table 2 and Figure 2 highlight the importance of considering endogenous mobility choice in the analysis. In particular,

[8] `https://www.google.com/covid19/mobility/`.

the comparison between the "dumb" SIRD, where mobility of susceptible, infected and recovered is maintained constant for the whole period of simulation and equal to their initial baseline values, and the ESIRD model, where individual mobility is decided in an optimizing framework without any imposed restriction, points out the 30% more cumulative deaths of dumb SIR as opposed to a lower drop in mobility and production (both as peak and as cumulative impact). After 425 days from its outbreak epidemic is substantially ended in both models, i.e. μ_I is almost zero, but the optimized mobility of individual in ESIRD has led to a non negligible mass of susceptible equal 31.4% in day 425 and substantially lower death rate (0.5% versus 0.7%).



(a) Dynamics of epidemic, economic activity and mobility with "dumb" individuals



(b) Dynamics of epidemic,economic activity and mobility with individuals optimizing their mobility choices.

FIGURE 2. Comparison between "dumb" SIRD model versus SIRD model with endogenous mobility. Numerical experiments based on the parameters reported in Table 1.

6. QUESTIONING THE ESIRD

In this section we discuss how our framework could be used to evaluate alternative policies of mobility restriction. The high peak prevalence reported for ESIRD in Table 2 explains why several countries imposed strong mobility restrictions in 2020. A peak of infected of 5,858,062 individuals would correspond to a need of about 398,749 beds in hospitals, taking 6.8% the proportion of infected individuals hospitalised (Verity *et al.*, 2020). For example, Italy in February 2020 had about 190,000 available beds in hospital, making "laissez faire" approach to COVID-19 not practicable (not considering the advantage to take time in waiting for a vaccine).

In the following we therefore study some mitigation strategies as defined in (Ferguson *et al.*, 2020, p.3), i.e. "to use NPIs (non-pharmaceutical intervention) not to interrupt transmission

completely, but to reduce the health impact of an epidemic" in the hope (as it is effectively happened) of a rapid development of a vaccine. In particular, we will focus on policies that, by increasing mobility costs (γs), reduce individual mobility and therefore the infection rate and the peak prevalence. In this regard, Nouvellet *et al.* (2020) provide strong evidence that reducing mobility is the key factor for bringing down COVID-19 transmission, while Vollmer *et al.* (2020) present scenario analysis based on different mobility in Italy.

At the same time, reduction mobility hurts production, putting policy maker in front a trade-off between economic losses and fatalities due to COVID-19, i.e. it is possible to point out a *pandemic possibilities frontier* as in Kaplan *et al.* (2020) and Acemoglu *et al.* (2020). However, we add two dimensions in the discussion, the first related to the share of remaining susceptible at the end of the period of analysis, which could make easier a fresh outbreak of epidemic in the future, and the second related to the social feasibility of some policies based on a long reduction of individual mobility.

Table 3 reports the effect of different policies increasing (in the same percentage) the cost of mobility for production and consumption with respect to the baseline model when the share of infected individuals exceeds 3% and to maintain this increase until the share of infected individual gets down to 0.5% or to 0.1% in the more severe scenario (mrs).

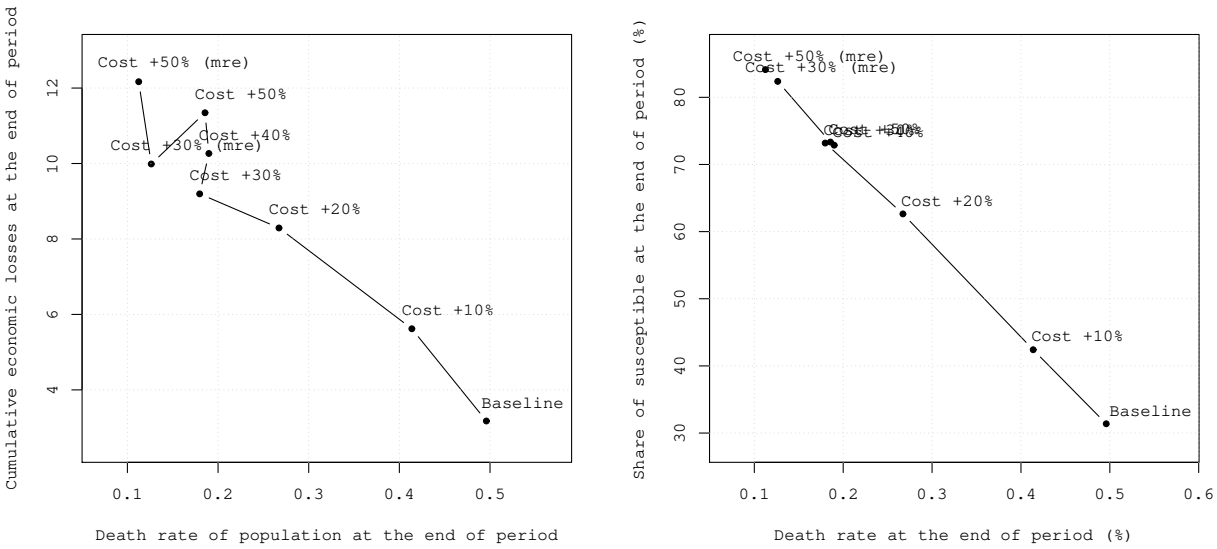
TABLE 3. Alternative scenarios of restriction of mobility (severity of lockdown) and exit from these restrictions (mrs adopts a more strict threshold for relaxing the restrictions). Numerical experiments based on the parameters reported in Table 1.

Scenario	Peak prevalence	Cumulative deaths	Minimum of production	Minimum of mobility	Economic loss	Mobility loss	$\mu_S(425)$	$\mu_I(425)$	$\mu_R(425)$	$\mu_D(425)$ (death rate)
Baseline ESIRD	5,858,062	297,577	0.883	0.693	−0.032	−0.082	0.314	0.003	0.678	0.005
Cost +10%	3,594,938	248,258	0.877	0.651	−0.056	−0.165	0.424	0.006	0.566	0.004
Cost +20%	1,633,960	160,311	0.867	0.603	−0.083	−0.254	0.626	0.005	0.365	0.003
Cost +30%	1,275,206	107,837	0.837	0.518	−0.092	−0.280	0.732	0.020	0.246	0.002
Cost +40%	1,258,593	113,914	0.800	0.439	−0.103	−0.299	0.729	0.010	0.260	0.002
Cost +50%	1,249,959	111,359	0.753	0.357	−0.113	−0.310	0.733	0.011	0.254	0.002
Cost +30% (mrs)	1,241,037	75,794	0.835	0.515	−0.100	−0.307	0.824	0.002	0.173	0.001
Cost +50% (mrs)	1,256,080	67,485	0.747	0.348	−0.122	−0.335	0.841	0.004	0.154	0.001

Peak prevalence decreases up to a rise of 30% in mobility cost and then it is almost rigid to further increment (see Table 3). Peak prevalence of 1,275,206 individuals would amount to a need of 86,801 beds in hospitals. Not reported numerical investigations show that to decrease this peak prevalence would require to start mobility restrictions with a lower share of infected individuals than 3%.

However, increasing mobility costs have also a growing negative impact both on economic activity and a death rate. This trade-off is represented in Figure 3a, which corresponds to the pandemic possibilities frontier discussed in Kaplan *et al.* (2020) and Acemoglu *et al.* (2020) but calculated in a very different theoretical framework. We can appreciate from Figure 3a how a

scenario with 30% of additional cost and an exit threshold of 0.1% from mobility restriction Pareto dominate the scenario with 30% of additional cost and an exit threshold of 0.5%.



(a) Trade-off between cumulative economic losses and cumulative death rates (number of fatalities on total population) after 425 days from the outbreak of epidemic in different scenarios

(b) Trade-off between cumulative death rates (number of fatalities on total population) and the share of susceptible after 425 days from the outbreak of epidemic in different scenarios

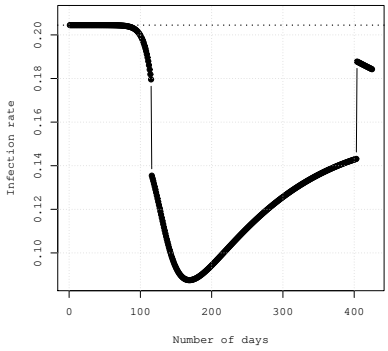
FIGURE 3. Trade-offs in alternative scenarios of mobility restrictions and exit from these restrictions. Numerical experiments based on the parameters reported in Table 1.

However, the former scenario presents two additional non favourable characteristics with respect to the latter. First, as reported in Figure 3b, the share of susceptible after 425 days from the outbreak of epidemics is substantially higher (82.4% versus 73.3%); moreover, as highlighted by Figures 4e and 5e, it requires a prolonged period of mobility restrictions (almost one year!). In this respect, scenarios with 30% of additional cost and an exit threshold of 0.5% or with 50% of additional cost and an exit threshold of 0.1% endogenously present a succession of periods with and without mobility restrictions making this scenario more socially feasible.

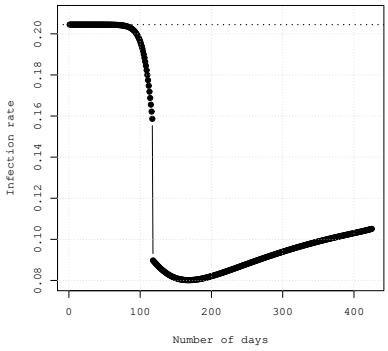
We conclude observing that, even though individuals are perfectly informed of restriction policy and on the behaviour of pandemic, several scenarios include waves of infections, as result of the endogenous switching between a regime with mobility restrictions and one without any restriction (see, e.g., Figures 5c-5f).

7. Concluding remarks

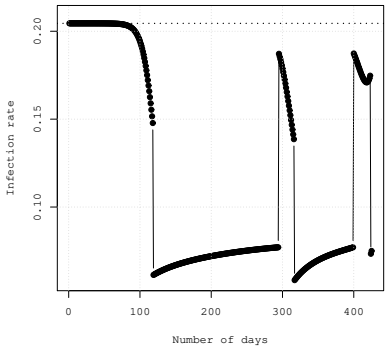
We provide a dynamic macroeconomic general equilibrium model with pandemic, denoted ESIRD, where perfect-foresight forward looking agents' (short-term) mobility positively affects their income (and consumption), but also contributes to the spread of pandemic in an extended SIRD model. Dynamics of economy and pandemic is jointly driven by strategic complementaries in production and negative externalities on infection rates of individual mobilities. We therefore address one of the main economic-driven leverages of compartmental epidemiological models,



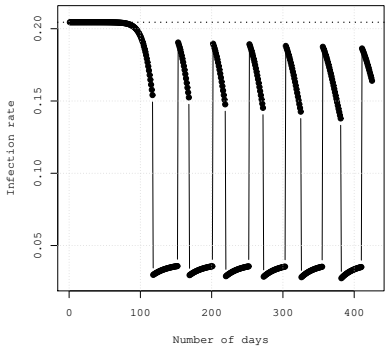
(a) Infection rate when lockdown implies an increase of 10% of cost of mobility



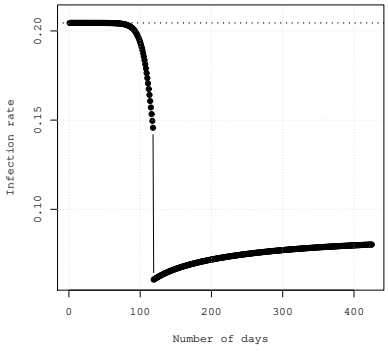
(b) Infection rate when lockdown implies an increase of 20% of cost of mobility



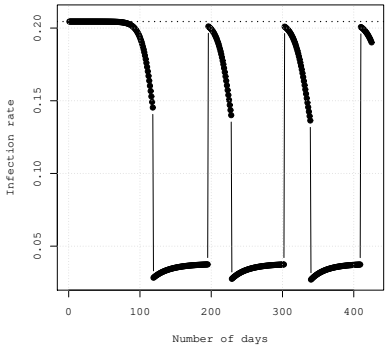
(c) Infection rate when lockdown implies an increase of 30% of cost of mobility



(d) Infection rate when lockdown implies an increase of 50% of cost of mobility



(e) Infection rate when lockdown implies an increase of 30% of cost of mobility and more restrictive conditions for the exit of lockdown

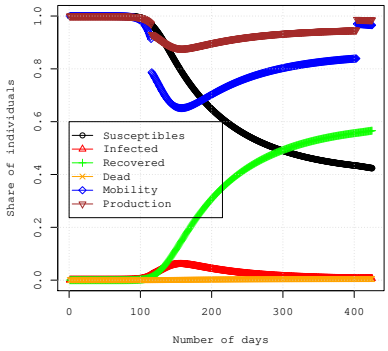


(f) Infection rate when lockdown implies an increase of 50% of cost of mobility and more restrictive conditions for the exit of lockdown

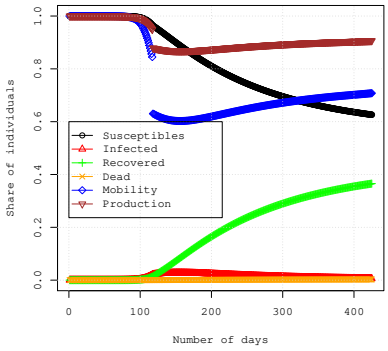
FIGURE 4. Dynamics of infection rate in different scenarios of mobility restriction (severity of lockdown) and exit from these restrictions (threshold for relaxing the restrictions). Numerical experiments based on the parameters reported in Table 1.

i.e. the endogenization of reproduction rate of epidemic (Avery *et al.*, 2020). After having proved the existence of a Nash equilibrium and studied the recursive construction of equilibrium(a), we conduct some numerical investigations on the forward-backward system resulting from individual optimizing behaviour, calibrating model's parameters on Italian experience on COVID-19 in 2020-2021.

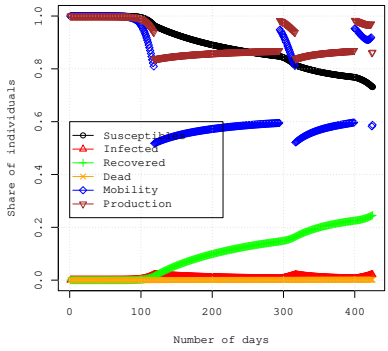
In our ESIRD model the forward-looking behavior of agents tends to smooth the peak prevalence of pandemic with respect to the simplest SIRD model with "dumb" agents, but in our numerical explorations peak prevalence appears to be still too high to be sustainable for the Italian health system (e.g. in relation to the number of available beds in hospital). Once establish that self-regulation of individual mobility decisions is not sufficient to manage the pandemic, we evaluate different regimes of mobility restrictions, which can be easily accommodate within our theoretical framework.



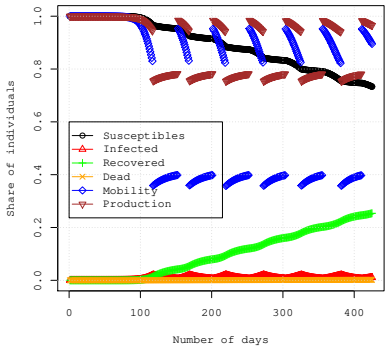
(a) Dynamics when lockdown implies an increase of 10% of cost of mobility



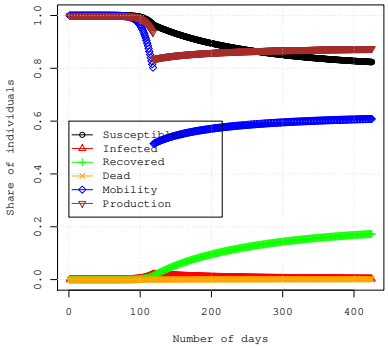
(b) Dynamics when lockdown implies an increase of 20% of cost of mobility



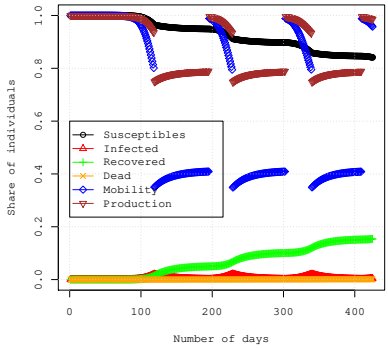
(c) Dynamics when lockdown implies an increase of 30% of cost of mobility



(d) Dynamics when lockdown implies an increase of 50% of cost of mobility



(e) Dynamics when lockdown implies an increase of 30% of cost of mobility and more restrictive conditions for the exit of lockdown



(f) Dynamics when lockdown implies an increase of 50% of cost of mobility and more restrictive conditions for the exit of lockdow

FIGURE 5. Dynamics of epidemics and of main economic variables in alternative scenarios of mobility restrictions and exit from these restrictions. Numerical experiments based on the parameters reported in Table 1.

In particular, we argue that regimes compatible with the saturation of healthcare system must be evaluated in terms of trade-off between economic losses and fatalities as proposes, e.g., by Kaplan *et al.* (2020); Acemoglu *et al.* (2020), but also for their social feasibility of maintaining prolonged periods of mobility restrictions and for leaving higher shares of susceptible at the end of the period, which makes fresh outbreak of epidemic more likely. In this respect, we point out that successive small waves of epidemic can be the result of an efficient regime of mobility restrictions.

Our analysis raises a series of issues for future research.

We ignore heterogeneity of population in terms of "risk groups" (typically, in case of Cavid-19, age cohorts, see Salje *et al.*, 2020 and Acemoglu *et al.*, 2020), and therefore we cannot evaluate any policy conditioned to individual characteristics, as, for instance, done by Brotherhood *et al.* (2020) or Gollier (2020). We also focus on a world before the vaccine, that is standard in this kind of models (Boppart *et al.*, 2020) and consistent with the period used to calibrate the

model. However, in a world with vaccine, or with an expected date of its availability, different questions arises for the timing, targets and costs of vaccination (Hung & Poland, 2021) as well as on the timing of mobility restrictions. Finally, we did not include other non-pharmaceutical interventions, and in particular we do not model testing policies, as, for instance, in Eichenbaum *et al.* (2020b).

Some extensions of empirical analysis appear very promising. Firstly, the possibility to study scenarios where mobility restrictions are (mostly) focused on mobility for production or on mobility for consumption. For example, in Europe the second waves of restrictive measures in the period Oct 2020 - May 2021, largely revolved around mobility for consumption.[9] A second extension concerns the more precise estimation of the relationship between individual mobility, aggregate mobility and production in presence of strategic complementarities, which poses non trivial issue of identification (Manski, 2000).

From the theoretical point of view, a general question on the proposed theoretical framework is if the Markov equilibrium can be found in a generalized feedback form, where the closed-loop strategies Θ depends not only on time and state of each agent, but also on the current distribution of the other agents. A possible answer is to look at the Master Equation associated to our model, which, in turn, could be a first step to obtain stronger properties of the equilibria, like subgame perfection, and, possibly, uniqueness (see, e.g., Section 1.4 in Cardaliaguet & Porretta (2020)).

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[9]See for instance, for France, JORF 0080, 3 April 2021, Text 28, `https://www.legifrance.gouv.fr/jorf/id/JORFTEXT000043327303`.

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Appendix A. Procedure of simulation

NOTE: In this appendix the notation is lightened from that used in the body of the article to avoid making the formulas too heavy thinking and difficult to read.

(A) **Set initial value of utilities and their feasible range.** We argue that as t goes to infinitum the number of infected agents converges to zero, i.e. $\lim_{t\to\infty}\mu(t,I)=0$ and $\lim_{t\to\infty}\mu(t,R)\gg 0$ and the lifetime utilities is maximum. Then:

$$U(S)^{\max}=\lim_{t\to\infty}U(t,S)=\lim_{t\to\infty}\frac{\kappa(t,\mu(t,I),SR)+\ln(1+\bar{Z}(t))}{\rho}= \tag{32}$$
$$=\frac{\kappa(\infty,0,SR)+\ln\left(1+1/\gamma_p(\infty,0,SR)-a_0^{SR}/a_1^{SR}\right)}{\rho};$$

$$U(R)^{\max}=\lim_{t\to\infty}U(t,R)=\lim_{t\to\infty}\frac{\kappa(t,\mu(t,I),SR)+\ln(1+\bar{Z}(t))}{\rho}= \tag{33}$$
$$=\frac{\kappa(\infty,0,SR)+\ln\left(1+1/\gamma_p(\infty,0,SR)-a_0^{SR}/a_1^{SR}\right)}{\rho};$$

$$U(I)^{\max}=\lim_{t\to\infty}U(t,I)=\frac{\rho\kappa(\infty,0,I)+(1-\rho)\pi_R\kappa(\infty,0,SR)}{\rho\left[1-(1-\rho)\left(1-\pi_R-\pi_D\right)\right]}+$$
$$+\frac{\left[1-(1-\rho)\left(1-\pi_R\right)\right]\ln\left(1+1/\gamma_p(\infty,0,SR)-a_0^{SR}/a_1^{SR}\right)}{\rho\left[1-(1-\rho)\left(1-\pi_R-\pi_D\right)\right]}. \tag{34}$$

where:

$$\kappa(t,\mu_I,SR):=\ln\left(\frac{a_1^{SR}}{\gamma_p(t,\mu_I,SR)}\right)+\gamma_p(t,\mu_I,SR)\frac{a_0^{SR}}{a_1^{SR}}+\ln\left(\frac{P_1}{\gamma_c(t,\mu_I,SR)}\right)+\gamma_c(t,\mu_I,SR)\frac{P_0}{P_1}-2; \tag{35}$$

and

$$\kappa(t,\mu_I,I):=\ln\left(\frac{a_1^{I}}{\gamma_p(t,\mu_I,I)}\right)+\gamma_p(t,\mu_I,I)\frac{a_0^{I}}{a_1^{I}}+\ln\left(\frac{P_1}{\gamma_c(t,\mu_I,I)}\right)+\gamma_c(t,\mu_I,I)\frac{P_0}{P_1}-2. \tag{36}$$

(B) **Feasible range.** Feasible range is defined as follows:

$$T:=\left\{(x,y,z)\in(0,U(R)^{\max})\times(0,U(I)^{\max})\times(0,U(R)^{\max}):y\le x\le z\right\}. \tag{37}$$

(C) **Set initial conditions of population at period 0 with ϵ very small**

$$\mu(0,S)=1-\epsilon; \tag{38}$$
$$\mu(0,I)=\epsilon; \tag{39}$$
$$\mu(0,R)=0; \tag{40}$$
$$\mu(0,D)=0. \tag{41}$$

(D) **Set the initial value of utilities in the three states in the feasible range T by choosing $\delta^I,\delta^S,\delta^R\ge 0$.**

$$U(0,R) = U(R)^{\max}(1-\delta^R); \tag{42}$$

$$U(0,S) = U(0,R)(1-\delta^S); \tag{43}$$

$$U(0,I) = U(0,S)(1-\delta^I) \tag{44}$$

(E) **Calculate** $a(0)$ **and** $b(0)$:

$$a(0) = \beta_P \times \mu(0,I) \times \overline{\theta}_p(0,\mu(0,I),I) \tag{45}$$

$$b(0) = \beta_C \times \mu(0,I) \times \overline{\theta}_c(0,\mu(0,I),I), \tag{46}$$

where

$$\overline{\theta}_p(0,\mu(0,I),I) = 1/\gamma_p(0,\mu(0,I),I) - \frac{a_0^I}{a_1^I} \text{ and} \tag{47}$$

$$\overline{\theta}_c(0,\mu(0,I),I) = 1/\gamma_c(0,\mu(0,I),I) - \frac{P_0}{P_1}. \tag{48}$$

(F) **Find** $\Delta U(1,S,I) := U(1,S) - U(1,I)$ **by solving the following implicit equation**

(49)

$$\begin{aligned} 0 = &-(1-\rho)\left(1-\pi_R-\pi_D\right)\Delta U(1,S,I) + \left(1-\pi_R-\pi_D\right)U(0,S) - U(0,I) + \pi_R U(0,R) + \\ &- \pi_R\kappa(1,\mu(1,I),R) + \kappa(1,\mu(1,I),I) - \left(1-\pi_R-\pi_D\right)\chi\left(\Delta U(1,S,I)\right) + \pi_D \ln\left(1+Z\left(\Delta U(1,S,I)\right)\right), \end{aligned}$$

where

$$\begin{aligned} \chi\left(\Delta U(1,S,I)\right) := &\\ &\ln\left(\frac{a_1^{SR}}{\gamma_p(1,\mu(1,I),S) + (1-\rho)a(0)\Delta U(1,S,I)}\right) + \\ &+ \frac{a_0^{SR}}{A_1^{SR}}\left\{\gamma_p(1,\mu(1,I),S) + (1-\rho)a(0)\Delta U(1,S,I)\right\} + \\ &+ \ln\left(\frac{P_1}{\gamma_c(1,\mu(1,I),S) + (1-\rho)b(0)\Delta U(1,S,I)}\right) + \\ &+ \frac{P_0}{P_1}\left\{\gamma_c(1,\mu(1,I),S) + (1-\rho)b(0)\Delta U(1,S,I)\right\} - 2 \end{aligned} \tag{50}$$

and

$$\begin{aligned} \bar{Z}(0) = Z\left(\Delta U(1,S,I)\right) = \mu(0,S) \times &\left[\frac{1}{\gamma_p(1,\mu(1,I),S) + (1-\rho)a(0)\Delta U(1,S,I)} - \frac{a_0^{SR}}{a_1^{SR}}\right] + \\ &+ \mu(0,I) \times \overline{\theta}_p(0,\mu(0,I),I) + \mu(0,R) \times \overline{\theta}_p(0,\mu(0,I),R). \end{aligned} \tag{51, 52}$$

where

$$\overline{\theta}_p(0,\mu(0,I),R) = 1/\gamma_p(0,\mu(0,I),R) - \frac{a_0^{SR}}{a_1^{SR}} \text{ and} \tag{53}$$

$$\overline{\theta}_c(0,\mu(0,I),R) = 1/\gamma_c(0,\mu(0,I),R) - \frac{P_0}{P_1}. \tag{54}$$

(G) **Calculate the level of movement of susceptible**

$$\overline{\theta}_p(0,\mu(0,I),S) = \frac{1}{\gamma_p(0,\mu(0,I),S) + (1-\rho)a(0)\Delta U(1,S,I)} - \frac{a_0^{SR}}{a_1^{SR}}; \tag{55}$$

$$\overline{\theta}_c(0,\mu(0,I),S) = \frac{1}{\gamma_c(0,\mu(0,I),S) + (1-\rho)b(0)\Delta U(1,S,I)} - \frac{P_0}{P_1}. \tag{56}$$

(H) **Calculate the level of utilities at period 1**

(57)

$$U(1,R) = \frac{U(0,R) - \ln\left(1+\bar{Z}\left(0\right)\right) - \kappa(0,\mu(0,I),R)}{1-\rho};$$

(58)

$$U(1,I) = \frac{U(0,I) - \pi_R U(0,R) + \pi_R\kappa(0,\mu(0,I),R) - \kappa(0,\mu(0,I),I) - (1-\pi_R)\ln\left(1+\bar{Z}\left(0\right)\right)}{(1-\rho)\left(1-\pi_R-\pi_D\right)};$$

(59)

$$U(1,S) = U(1,S,I) + U(1,I);$$

(I) **Upgrade the composition of population**

$$\mu(1,S) = \mu(0,S)\left[1 - a(0)\overline{\theta}_p(0,\mu(0,I),S) - b(0)\overline{\theta}_c(0,\mu(0,I),S)\right], \tag{60}$$

$$\mu(1,I) = \mu(0,S)\left[a(0)\overline{\theta}_p(0,\mu(0,I),S) + b(0)\overline{\theta}_c(0,\mu(0,I),S)\right] + \mu(0,I)(1-\pi_R-\pi_D), \tag{61}$$

$$\mu(1,R) = \mu(0,I)\pi_R + \mu(0,R), \tag{62}$$

$$\mu(1,D) = \mu(0,D) + \mu(0,I)\pi_D. \tag{63}$$

(J) **Check if Condition (37) is satisfied. If not start with a new set of δs at point D. If Condition (37) is satisfied and the number of periods is lower of a given threshold repeat points E-I by taking the new level of μs at point I.**

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