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RESEARCH ARTICLE

A mean field game model for COVID-19 with human capital accumulation

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Abstract
In this manuscript, we study a model of human capital accumulation during the spread of disease following an agent-based approach, where agents behave maximising their intertemporal utility. We assume that the agent interaction is of mean field type, yielding a mean field game description of the problem. We discuss how the analysis of a model including both the mechanism of change of species from one epidemiological state to the other and an optimisation problem for each agent leads to an aggregate behaviour that is not easy to describe, and that sometimes exhibits structural issues. Therefore we eventually propose and study numerically a SEIRD model in which the rate of infection depends on the distribution of the population, given exogenously as the solution to the mean field game system arising as the macroscopic description of the discrete multi-agent economic model for the accumulation of human capital. Such a model arises in fact as a simplified but tractable version of the initial one.

Keywords Spatial SIR type models · Mean field games · Spatial interactions

JEL Classification E19 · I10 · D90 · O11 · C73

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1 Introduction

Typical models in epidemiology, as well as in economics, describe the time evolution of variables as the number (absolute or relative) of susceptible and infectious persons. In the last 20 years, there has been an increasing interest in spatial extensions of such models, in which the evolution of variables occurs not only in time but also across space. This is a major feature in the so-called economic geography literature, and in general in any model in which the dynamics is not supposed to be uniform across space. Particularly suitable for spatial extensions are interaction models, namely those in which the dynamics mechanism originates from some sort of interaction between agents and the intensity and effectiveness (or even the form) of such interaction depend on the relative or absolute position of the agents. The interest of these spatial models is twofold, as they can bring insight into the effects of the spatial distribution of agents on the evolution of variables or, the other way around, on the consequences of interaction on the movement of people across space.

The SIR epidemiological model is particularly meaningful under this viewpoint: in its classical formulation (Kermack and McKendrick 1927) the dynamics of the variables S and I are governed by the incidence of the disease, that is the product of the probability that contact with infectious individual results in contagion with the average number of susceptible individuals any infectious individual gets in contact with. Clearly, this average number is quite a rough estimate of the actual number of contacts people have, which may well be different in different places, due for example to differences in the spatial density of people.

In the epidemics literature there has been some work in the last years (Guo et al. 2020; Paeng and Lee 2017; Takács and Hadjimichael 2022) about extending SIS and SIR models with spatial components, before the pandemic began; this type of investigation have been pushed further more recently with the spread of COVID-19 (Colombo et al. 2020; Sy et al. 2021; Wong and Li 2020) since geographical differences in the pandemic evolution (both at large and small spatial scales) suggest that epidemic dynamical models should take into account—at least in some phases of the spread of the disease—the distribution of people and of significant economic variables across space. This can be done in several ways, depending on the mechanism and analysis one is interested in and on the pandemic phase one is observing.

The spatial density of people represents a significant factor in the dynamics of diseases; indeed many measures that regulators have taken to oppose the diffusion of the pandemics can be seen as density-dependent (e.g. the requirement to wear masks in crowded places; even lockdown can be seen as a way to drastically reduce large peaks in the spatial density of people).

The attempt to incorporate spatial features into SIR-type models poses two issues: on the epidemiological and economic side, to understand what type of space-dependency might be appropriate; on the mathematical side, passing from an ODE description to a PDE description, to have a tractable model, at least numerically. A strand of the literature considers the space movement of people as time flows by adding diffusion terms—typically Laplacian or nonlocal diffusion terms—in the equations of SIR-type modes, thus studying a system of PDEs. We refer e.g. to Faragó and Róbert

(2018), Li and Yang (2014), Reluga (2004), and further references in the introductions of Paeng and Lee (2017), Takács and Hadjimichael (2022).

Part of the literature focuses on the way the transition rate depends on the space-time distribution of people. For example in Paeng and Lee (2017) the density is constant in time but space dependent and arises in the standard true mass incidence function replacing the total number of individuals while in Hu et al. (2013) the density is constant and appears also in the transmission rate with a nonlinear dependence. In both the above-mentioned papers, the density is exogenous.

In the present paper, we are interested in exploiting the dependence on spatial distribution in the incidence of the disease. We choose SEIRD dynamics to model the epidemics, where the population is divided into the classes: *susceptible, exposed, infectious, removed, dead*. Especially after the COVID-19, diverse epidemiological models with additional compartments, such as vaccinated, hospitalised, isolated at home, or age-structured models have been proposed (see e.g. Tembine 2020 for a mean field game perspective on COVID-19 that considers many of these aspects and Fabbri et al. (2021) and its references for age-structured models). Our approach could be adapted to such epidemiological models with no major difficulties (but with more consistent modifications in the case of age-structured models with age as a variable). We decided to focus on the SEIRD model since at the same time it captures the main feature of COVID-19 and it stays simple enough to focus on the main interest of the present paper, that is, the dependence on the spatial distribution of people.

The main novelty consists in how we choose the spatial distribution of agents. The model we propose consider simultaneously the space–time evolution of the epidemics and of a key economic variable, human capital. The description of the evolution of human capital is of crucial importance in economics and has attracted more and more interest nowadays, see Boucekkine et al. (2008) for a review of human capital accumulation models on an epidemic setting, or Bleakley (2010) where some empirical evidence is proposed. In particular, in the model we consider for the space–time evolution of human capital, the dynamics of an individual's human capital are affected by the human capital of nearby individuals through a spatial interaction term, that is, we account for *spatial spillover* on accumulation of human capital. Individuals control their position in space to maximise their gain from such spillover, considering at the same time that moving towards areas with high human capital is a costly action. Assuming that no individual has an overwhelming influence on the system with respect to others, optimisation of each individual's position gives rise to a symmetric game among economic agents (the individuals).

When the number of agents is large the aggregate dynamics are typically described by a Mean Field Game (MFG hereinafter) system, arising heuristically as the limit as the number of agents tends to infinity. The typical MFG system consists of two partial differential equations: a Hamilton–Jacobi–Bellman (HJB hereinafter) equation describing the optimal control problem of the agents, and a Fokker-Planck (FP hereinafter) equation, describing the distribution of the population with respect to space and human capital.

An important motivation for the MFG approach we choose for our economy lies in the fact that a solution to the MFG system is expected to provide a good approximation of Nash equilibria for the discrete multi-agent model when the number of agents is large.

Moreover, it allows for substantial computational simplifications. In fact, it is well known that solving N coupled optimisation problems, or analogously an optimisation problem of dimension N becomes unviable from the numerical point of view as soon as N gets moderately large ($N = 10$ is already beyond reach in the general case). On the contrary the problem with the mean field limit reduces to a system of two coupled partial differential equations that can be handled in a simpler way.

For a complete presentation of such a model—without reference to epidemics—and in particular of the mathematical framework, we refer to the forthcoming paper (Ghilli et al. 2023), where the MFG model is studied analytically and numerically; the details of the model are discussed below in Sect. 2.2.

In the present paper, we link such MFG dynamics for the human capital with epidemic dynamics, contributing to the literature linking economic and epidemic variables. Gersovitz and Hammer (2004) proposed one of the first significant contributions to the *Epi-Econ* literature. In recent years the COVID-19 pandemic incentivized the flourishing of this field of research. While most studies deal with strategies to control epidemics from the point of view of a social planner (Acemoglu et al. 2021; Aspri et al. 2021; Calvia et al. 2023; Eichenbaum et al. 2021; La Torre et al. 2023; Dobson et al. 2023), only a minor part investigate the trade-off between epidemic and economic losses from the perspective of forward-looking interacting agents, see for example (Fabbri et al. 2023). Our contribution fall under this last category.

A realistic model one could consider is one in which individuals tend to increase their level of human capital by being close to other individuals and at the same time maintain social distancing, in order to minimise the probability of getting infected. In such a model the space-time distribution of individuals would be endogenously determined by their optimal choices. The corresponding optimisation problem is however too difficult to solve, and it is not even clear how one should formulate the problem at the MFG level. The major technical difficulties in this direction arise in two ways. First, each class of the SEIRD model has different goals with respect to keeping social distancing; for example, a susceptible individual may want to keep some distance from a generic individual unless she has recovered. Second, from the single-individual point of view, one should account both for the optimisation of an agent's position and for her possible change of epidemic class at random times; this is conveniently modelled through piecewise-deterministic stochastic processes that jump from a class to the next at random times whose intensity depends on the interaction. To the best of our knowledge, the only available attempts to consider such features in the epidemic modelling literature are Doncel et al. (2020), Olmez et al. (2021), Petrakova and Krivorotko (2022), where, however, the optimisation problem is the same for all classes, thus avoiding the first technical difficulty mentioned above. We also mention (Bertucci 2020), where MFGs with changes of states are studied in a different context. We underline that these issues are related to the optimisation problem only: the appearance of random jumps is not a problem by itself if one wants to understand the aggregate dynamics in the setting of mean field limits. These have been broadly studied, especially in the case of biological applications, see e.g. (Catellier et al. 2021; Oelschläger 1989), where however no optimisation is involved. Nonetheless, a proper understanding of how to derive the aggregate MFG dynamic for jump processes in the controlled case we propose is still beyond reach for the current knowledge on MFGs, even though the theory of optimal control of jump process has been already developed by many extents, see e.g. (Calvia 2018, 2020) and references therein. We also remark that interacting systems with spatial interaction but without control are also mathematically well understood, see for example (Flandoli et al. 2019; Zanco 2020) and references therein. One way of simplifying the model described above is to neglect the transition between epidemiological states. In this way, one would obtain a MFG system for each compartment in our epidemiological model. MFGs with different classes (often called species in similar models)—but where no transitions between classes are allowed— have been proposed for example in Achdou et al. (2017), Bensoussan et al. (2018), Cirant (2015), Feleqi (2013). However, we believe that ruling out the transition between classes is a too restrictive assumption. Therefore we formulate a MFG model where we incorporate transitions but we assume that there is no distinction among classes in the optimisation goals. The macroscopic evolution in this case is described by one MFG system and one differential equation for each compartment. Moreover we suppose that the infection rate depends on the space-time distribution of the agents. In this model it is easily seen that the space-time distribution must satisfy a structural condition, namely to be independent of time. For this reason, we choose for our model the space marginal of the stationary solution to the FP equation in the MFG system. This actually means that to have a model that is sufficiently simple to be tractable, at least numerically, the distribution has to be exogenous. From the modelling point of view, considering a stationary distribution can be seen as corresponding to the assumption that the mechanism that links the evolution of human capital with the individuals' positions has run for a long time and has stabilised. Incorporating such stationary solution exogenously in our SEIRD model corresponds then to assume that the dynamics of the epidemic have a negligible effect on the distribution of people; this may be seen as the two distinct dynamics, the one described by the MFG system and the one described by the SEIRD model, happening on different time-scales, with a much shorter time-scale for the epidemic dynamic. Considering the space marginal of the stationary solution to the MFG system means that we cannot take into account the influence of the time-evolution in the accumulation of human capital on the epidemic dynamics. To our knowledge, there is little amount of study or evidence in the literature of the influence of such variables on the incidence term in epidemiological models, see Berkessel et al. (2021), Goenka and Liu (2020). On the contrary, some authors have been focusing on the effect of the evolution of epidemics on the level of human capital (Bleakley 2010; Deng et al. 2021); this phenomenon is inevitably ruled out by the simplifications we have to perform.

At the end of these simplifying steps, we obtain the following model:

$$\begin{cases} \partial_t S(t,x) = -\beta\big((S+E+I+R)(t,x)\big)\dfrac{S(t,x)}{(S+E+I+R)(t,x)}\displaystyle\int K_\chi(x,y)I(t,y)\mathrm{d}y\ , \\[2ex] \partial_t E(t,x) = \beta\big((S+E+I+R)(t,x)\big)\dfrac{S(t,x)}{(S+E+I+R)(t,x)}\displaystyle\int K_\chi(x,y)I(t,y)\mathrm{d}y - \theta E(t,x)\ , \\[2ex] \partial_t I(t,x) = \theta E(t,x) - \lambda I(t,x) - \delta I(t,x)\ , \\[2ex] \partial_t R(t,x) = \lambda I(t,x)\ , \\[2ex] \partial_t D(t,x) = \delta I(t,x)\ , \\[2ex] \rho V(x,h) = \frac{1}{2}\chi^2 h^2 \partial^2_{hh} V(x,h) + \frac{1}{2}\varepsilon^2 \partial^2_{xx} V(x,h) \\ \qquad\qquad\qquad + H_0(\partial_x V(x,h)) + H_1\big(x,h,\mu(x,h),\partial_h V(x,h)\big)\ , \\[2ex] \frac{1}{2}\sigma^2 \partial^2_{hh}(h^2\mu(x,h)) + \frac{1}{2}\varepsilon^2\partial^2_{xx}\mu(x,h) = \partial_x\left(\partial_p H_0(\partial_x V(x,h))\mu(x,h)\right) \\ \qquad\qquad\qquad\qquad\qquad + \partial_h\left(\partial_p H_1(x,h,\mu(x,h)), \partial_h V(x,h)\mu(x,h)\right)\ , \\[2ex] S(t,x) + E(t,x) + I(t,x) + R(t,x) + D(t,x) = \displaystyle\int \mu(x,h)\mathrm{d}h\ . \end{cases} \tag{1}$$

Here t denotes time, x denotes space and h denotes human capital. System (1) is made of an epidemic dynamics part (the first five equations), an economic optimisation part (the following two equations) and a compatibility condition (the last equation). Details are discussed in Sect. 2 and its subsections. We divide the population into five non-intersecting classes, namely

- the class of *susceptible* individuals, i.e. those that are currently not infected and can be infected;
- the class of *exposed* individuals, i.e. those that have got the infection but are not infectious yet, meaning they cannot propagate;
- the class of *infectious* individuals, i.e. those that can spread the disease;
- the class of *recovered* individuals, i.e. those who healed and are thus immune to the disease;
- the class of *dead* individuals, where death is due only to the disease and not to other causes.

The fraction of the total population that belongs to each class, as a function of time and space, is denoted by S, E, I, R, D, respectively. With respect to the classical SIR dynamic, there are two additional classes: E (*exposed* individuals) and D (*dead* individuals). The presence of class E implies that there is a latency period, typical of many infectious diseases, during which individuals have been infected but are not yet infectious. We focus on modelling just deaths due to the infection and we do not consider either births or death due to other causes than the infection. Moreover, we do not assume diffusion of individuals, as it seems unfit to this type of model at large spatial scales, see also the discussion in the introduction of Paeng and Lee (2017). On the other hand, we assume that the infection propagates in space, that is, a susceptible individual can be infected by an infectious individual provided they are close enough. This propagation effect is modelled through a convolution in the space variable (the integral term in the first two equations of (1)), where the support of the convolution kernel quantifies the threshold for possible contagion to occur. For analogous approaches, we refer to Colombo et al. (2020), Takács and Hadjimichael (2022) and references therein. We remark that differently from Colombo et al. (2020) we do not consider age dependence, focusing only on space dependence. As mentioned above, the way the transition rate depends on the distribution is inspired by Hu et al. (2013).

It is our belief that the models we discuss herein can be also effectively applied to the description of other diseases as well as other economic variables, modifying parameters and related properties of the models accordingly.

Finally we stress that we only provide a numerical solution to the stationary MFG system that arises in the model we consider; a rigorous proof of the existence of a stationary solution to the MFG system is not available and provides a mathematical challenge for the following reasons. The typical assumptions under which existence and uniqueness have been proved so far are separability of the Hamiltonian with respect to the distribution of the agents and the gradient of the value function and monotonicity of the coupling. In our case the Hamiltonian is not separable and consequently there is not a straightforward way of defining monotonocity with respect to the distribution. We refer to the end of paragraph 2.2.2 for a review of the available literature and a more detailed explanation of the underlying difficulties.

The remainder of the paper is organised as follows: in Sect. 2 we give some motivations to the model we consider and we present the joint human capital-epidemiological model used in the rest of the paper. In particular, in Sect. 2.1 the epidemiological component is presented, while in Sect. 2.2 we present the MFG component of the model related to human capital accumulation. Finally, in Sect. 3 numerical results are presented.

2 The hybrid MFG-SEIRD model

In this section we argue about the model we propose; in particular, we explain how the actual model we consider arises from a class of reasonable models due to mathematical and modellistic considerations.

We write here the equations we solve, and then we begin to discuss the meaning of each term as well as motivations and details.

The SEIRD model we propose takes the form

$$\begin{cases} \partial_t S(t,x) = -\beta\big(\mu(x) - D(t,x)\big)\dfrac{S(t,x)}{\mu(x) - D(t,x)} \displaystyle\int K_\chi(x,y) I(t,y)\mathrm{d}y \ , \\ \partial_t E(t,x) = \beta\big(\mu(x) - D(t,x)\big)\dfrac{S(t,x)}{\mu(x) - D(t,x)} \displaystyle\int K_\chi(x,y) I(t,y)\mathrm{d}y - \theta E(t,x) \ , \\ \partial_t I(t,x) = \theta E(t,x) - \lambda I(t,x) - \delta I(t,x) \ , \\ \partial_t R(t,x) = \lambda I(t,x) \ , \\ \partial_t D(t,x) = \delta I(t,x) \ , \end{cases} \tag{2}$$

where $\mu(x) = (S + E + I + R + D)(t, x)$ is the x-marginal of the second component of the stationary solution (V, ν) to the MFG system

$$\begin{cases} \rho V(t,x,h) = \frac{1}{2}\sigma^2 h^2 \partial^2_{hh} V(t,x,h) + \frac{1}{2}\varepsilon^2 \partial^2_{xx} V(t,x,h) \\ \qquad\qquad + H_1\big(x, h, \nu(t,h,x), \partial_h V(t,x,h)\big) + H_0(\partial_x V(t,x,h)) \,, \\ \partial_t \nu(t,x,h) = \frac{1}{2}\varepsilon^2 \partial^2_{xx} \nu(t,x,h) - \partial_x \left(\partial_p H_0(\partial_x V(t,x,h))\nu(t,x,h)\right) \\ \qquad\qquad + \frac{1}{2}\sigma^2 \partial^2_{hh}(h^2 \nu(t,x,h)) - \partial_h \left(\partial_p H_1(x,h,\nu(t,x,h)), \partial_h V(t,x,h)\nu(t,x,h)\right) \,. \end{cases} \tag{3}$$

System (2) is described in Sect. 2.1 below. System (3) instead describes the economic dynamics; the details of the discrete multi-agent system it approximates, together with the precise definitions of the Hamiltonians H_0 and H_1, are given in Sect. 2.2. Here we discuss the link between the epidemics and the economic dynamics and the considerations that lead us to consider stationary solutions to the MFG system. We begin with a situation in which each agent behaves optimally in the sense of intertemporal utility maximisation, that is, each agent is assumed to be *forward looking*. Each agent's utility depends not only on the characteristic of the agent itself but also on the configuration of the system as a whole, i.e. it depends on the position, endowment of human capital and epidemic state of all other agents. On the economic side, the model we consider aims at describing the space–time evolution of the human capital of agents ($h(t)$ denotes the amount of human capital at time t in the following). Agents control their investment in education $f(t)$: this means that at each time t an agent chooses to invest $f(t)h(t)$ into the production of new human capital due to interaction and $(1 - f(t))h(t)$ in the maximisation of the current utility. The model includes a form of spatial interaction, in the sense that agents tend to maximise their level of human capital by being close in space to other agents with a higher level of h. This constitutes a form of *positive spatial spillover* with regard to human capital. Moreover, agents control their velocity $v(t)$ (direction and length of their space movement at time t) with the aim of maximising their gain from such spillover and meanwhile taking into account the cost of moving towards areas with high human capital. We consider Nash equilibria as aggregate optima for the system of agents when the number of agents is large. At the same time, depending on their current epidemiological state, agents try to maintain social distancing to minimise the probability of becoming infected, which would increase their probability of dying. These two mechanisms induce a *competing effect* between the tendency to agglomerate and the necessity of keeping spatial distance. Moreover, the epidemiological state of each agent can change, according to the state of neighbouring agents,e.g. susceptibles have a certain probability of getting infected if they are spatially close to other infected individuals. From the point of view of the control problem, this corresponds to considering different utilities depending on the epidemiological state of the agents.

The situation just described would be the most accurate formulation for the problem we are considering; it would be mathematically given in terms of a MFG system that includes the possible switch between epidemiological states. However, it turns out that studying, even numerically, a model that introduces such a switch inside the formalism of MFG systems where the control acts on a state variable different than the epidemiological state, poses a phenomenal mathematical challenge, which to the best of our knowledge has not been solved yet by the mathematical community. Even formulating any such MFG system seems currently out of reach.

Since the transition mechanism from one epidemiological state to the other is the primary driver of the epidemic dynamics we surely do not want to neglect it. Therefore we are compelled to simplify our original assumptions as much as to include the minimum reasonable requirements that make the model adequate for the case of study we have in mind. Those are (i) human capital accumulation in the form of endogenous saving and optimisation over space embedding spatial spillovers as a feature; (ii) transition between epidemiological states depending on the spatial configuration of agents. Thus we may consider a system of agents where each of them is maximising an objective function that does not depend on her epidemiological state, i.e. the objective function is common to all the states, but agents switch states depending on their neighbours.

This has two main consequences. On the one hand, assuming that the objective function does not depend on the epidemiological state means that, at the level of the continuum aggregate dynamics, we get a single MFG system, namely (3); it is composed of a Hamilton–Jacobi–Bellmann equation which intuitively describes the value function V of the optimisation problem given a distribution ν of people (in time, space and human capital), and of a Fokker-Planck equation which describes the distribution ν given an optimisation strategy V.
On the other hand, the link between (2) and (3) realises not only in the dependence of the infection dynamics on $\mu(t,x)$ (the space-marginal of ν) but also in the necessary equality

$$\mu(t,x) = S(t,x) + E(t,x) + I(t,x) + R(t,x) + D(t,x).$$

At the same time, the fact that agents belong to only one of the classes for every given time t implies that the distributions of the variables $S(t,x)$, $E(t,x)$, $I(t,x)$, $R(t,x)$, $D(t,x)$ should balance each other so that the total amount of mass in the system is preserved, i.e

$$0 = \partial_t S(t,x) + \partial_t E(t,x) + \partial_t I(t,x) + \partial_t R(t,x) + \partial_t D(t,x) = \partial_t \mu(t,x).$$

This readily brings us to a contradiction, since it implies that the distribution $\mu(t,x)$ of all the agents, independently of the class they belong to, should be stationary in time, as opposed to our initial assumptions. From the stationarity of the space marginal $\mu(t,x)$ one cannot readily derive that of $\nu(t,x,h)$. In fact, there is indeed the possibility to construct solutions which are not changing over time along x, but only along h, For example by a symmetry argument, one can show that a uniform in x initial condition will remain uniform over time (in absence of non-uniform exogenous factors), thus having dynamics only on the h component. However, we will not consider these pathological cases, since they are ignoring the presence of space altogether, therefore reducing the MFG to a much easier problem and the SEIRD dynamics to a classical ODE problem.

Motivated by the above considerations, we assume that the distribution of people in space is given via a density function which is exogenous and does not change with time but influences the incidence of the disease; we thus use the x-marginal of the stationary solution to the second equation in the MFG system (3) as the distribution of people. Our model is clearly not suitable for describing any phase of the epidemic evolution, and this is even more true for the COVID-19 pandemic where measures such as lockdowns or total absence thereof significantly impact the time-evolution of the epidemic situation. Indeed we take population density in space as exogenous and given a priori; this means that we do not consider the movement of people in time nor the feedback effect of the epidemic dynamics on the population density. Therefore our model describes an epidemic phase in which the population density can be considered stationary in space-time; this corresponds for example to an initial stage of the spread of the disease, when regulators have not yet taken measures to prevent the propagation of the infection thus the population distribution is not affected by the dynamics of the epidemics, as well as a further stage in which movement-restricting measures have been lifted. In both cases, the underlying idea that justifies considering some stationary distribution in space is that the mechanism that regulates people's movement in the absence of an epidemic emergency has a time scale that is significantly longer than the time scale of the pandemic period we observe.

As the spatial distribution is a fortiori exogenous, one could consider various distributions. There are of course many possibilities; the uniform distribution is surely the easiest choice but is not interesting in this regard as it would correspond to no differences across space, thus making the spatial extension of the model vanish in practice. Of course, one could in principle test any distribution, but we are interested in using a distribution that has a solid economic justification.

A more realistic approach would consider non-stationary solutions of such MFG, leading to μ depending also on time; this is however not consistent with what was argued above. Another possibility would be to directly couple the mean-field game dynamics with our SEIRD dynamics, following the literature that discusses changes in the human capital distribution due to the dynamics of the epidemic; as already hinted above, we underline that studying this scenario is currently out of reach, both analytically and numerically.

2.1 The SEIRD dynamics

In the following, we introduce the SEIRD dynamic for the evolution of the epidemic, where the rate of transmission depends on the distribution of the total population.

The space domain here is $\mathbb{S}^1$, the 1-dimensional torus. As it is often more convenient to consider the time evolution of positions as they belong to $\mathbb{R}$ (for example with stochastic calculus), we will solve the agent-based models below in $\mathbb{R}$ and then project the solutions on the torus: for every $x \in \mathbb{R}$ we consider the quantity $x \pmod 1 \in [0, 1)$ (that is the fractional part of x); positions in $[0, 1)$ are in one-to-one correspondence with positions in $\mathbb{S}^1$.

To measure distances consistently on the torus we define, for every $x, y \in [0, 1)$, the distance

$$d_{\mathbb{S}^1}(x, y) := \min\{(x - y) \pmod 1, (y - x) \pmod 1\} \ ;$$

this clearly corresponds to the arc-length distance on $\mathbb{S}^1$ assuming that $\mathbb{S}^1$ has total length 1.

The population has constant size and is spatially distributed on $\mathbb{S}^1$ according to a density $\mu(x)$.

In our model we allow for a latency period between the time at which an individual gets infected and the time at which it can infect others; we do not consider births and deaths for ordinary causes, focusing only on the deaths caused by the infection. This implies that in the epidemic phase described by our model, the number of deaths not ascribable to the infection is negligible with respect to the number of deaths in the population due to the infection. We also assume that all newborn individuals can contract the disease and that all recovered individuals are immune.

The main novelty we propose is the space-dependent way in which we model the incidence of the disease. In the classical SIR-type models the form of the incidence is heuristically motivated as follows: if the population has size N, in a unit of time a single infectious individual is on average in contact with $C = cN$ individuals, for some proportionality constant $c > 0$; among those individuals, S/N are susceptible, thus leading to cS contacts with susceptible individuals in one unit of time. Since transmission of the disease is not certain, only pcS contacts produce new infectious, with $p \in (0, 1)$. This must be multiplied by the number of infectious, leading to the incidence being $pcSI$, usually written as βSI. Since we take into account the distribution of individuals in space, this has to be modified accordingly: at the simplest level this implies substituting N with $\mu(x)$, that is the number of people in the position x of the infectious individual; this would lead again to the incidence having the form βSI. However there is evidence in the literature (see for example Hu et al. 2013; Liu and Stechlinski 2012; Hethcote 2000) that it is often not accurate to assume the average number of contacts C of an infectious individual be linear in N; if we assume that it is not, then the above argument yields for the incidence term the form

$$\beta(\mu(x))\frac{S(t, x)}{\mu(x)}I(t, x) \ . \tag{4}$$

Since the spatial structure of our model is continuous, with an incidence of the form (4) one would have a contagion effect only with individuals that are *exactly* at the same location at the same time. This is clearly not realistic, as the spread of the infection from an infectious individual occurs in some small area around her, with people being closer having a higher probability of contracting the infection from the contagious individual, and people sufficiently far away having zero probability thereof. To model this feature we take the convolution in space of the variable I with a smooth compactly supported symmetric kernel K; therefore the incidence at time t and location x depends not only on the number of infectious individuals at the same time and location, but also on the number of infectious individuals *around* location x, with a maximal range of infection that is given by one-half time the width of the support of K, and with infection strength that is maximal at location x and decreases as one moves farther away from x. So, we propose the following form of incidence:

$$\beta(\mu(x))\frac{S(t,x)}{\mu(x)}\int_{\mathbb{S}^1} K_\chi(x,y)I(t,y)\mathrm{d}y \tag{5}$$

where $\beta\colon \mathbb{R}\to\mathbb{R}$ is an increasing function such that $\beta(0)=0$ and $K_\chi(x,y)=\frac{1}{\chi}\widetilde{K}\left(\frac{1}{\chi}d_{\mathbb{S}^1}(x,y)\right)$ for $\chi>0$ and $\widetilde{K}\colon[0,1/2]\to\mathbb{R}$ a smooth function such that $K(x)=0$ for every $x\in[1/2-\epsilon,1/2]$, for some $\epsilon\in\left(0,\frac{1}{2}\right)$. In this way, $\widetilde{K}$ can be identified with a function on $\mathbb{S}^1$ whose support is a strict compact subset of $\mathbb{S}^1$. For analogous approaches, we refer to Colombo et al. (2020), Takács and Hadjimichael (2022).

Denoting by λ the recovery rate of infectious individuals, by $\frac{1}{\theta}$ the latency period of the infection (i.e. the amount of time that passes between contracting the disease and becoming infectious, thus contributing to the spread of the disease), and by δ the death rate, our space-time dynamical model thus can be written as (2), that we rewrite for convenience:

$$\begin{cases}
\partial_t S(t,x) & = -\beta\big(\mu(x)-D(t,x)\big)\dfrac{S(t,x)}{\mu(x)-D(t,x)}\displaystyle\int_\Omega K_\chi(x,y)I(t,y)\mathrm{d}y\,,\\
\partial_t E(t,x) & = \beta\big(\mu(x)-D(t,x)\big)\dfrac{S(t,x)}{\mu(x)-D(t,x)}\displaystyle\int_\Omega K_\chi(x,y)I(t,y)\mathrm{d}y-\theta E(t,x)\,,\\
\partial_t I(t,x) & = \theta E(t,x)-\lambda I(t,x)-\delta I(t,x)\,,\\
\partial_t R(t,x) & = \lambda I(t,x)\,,\\
\partial_t D(t,x) & = \delta I(t,x)\,,
\end{cases} \tag{2}$$

together with suitable initial conditions. Comparing to (5), in the incidence function we subtracted from $\mu(x)$ the proportion $D(t,x)$ of dead individuals since we expect deaths to have no influence in the incidence term.

For every $x\in\mathbb{S}^1$ we have

$$S(t,x)+I(t,x)+E(t,x)+R(t,x)+D(t,x)=\mu(x)\,, \tag{6}$$

while (2) implies

$$\partial_t\left(S(t,x)+E(t,x)+I(t,x)+R(t,x)+D(t,x)\right)=0\,,$$

confirming that the total population remains constant. In this setting, contrary to some of the cited papers in the introduction, there is no movement of people due to the epidemic mechanism.

In our numerical analysis, we will consider two main cases: β in (5) constant in $\mu(x)$, analysed in Paeng and Lee (2017) or β having a nonlinear dependence in $\mu(x)$ as proposed in Hu et al. (2013). The case β constant in $\mu(x)$ coincides with the true mass action or proportioned mixing incidence where $\mu(x)$ replaces the total number of individual N. This case can be heuristically derived from a constant contact rate not depending on the size of the population. Hence the per-link contact rate $\frac{C}{\mu(x)}$ decreases with larger density. However, due to (6) an increase in $\mu(x)$ does not necessarily implies an increase in the number of susceptible individuals. In the present paper we consider the case where β explicitly depends on $\mu(x)$ as in (5) and in particular shows a nonlinear dependence as argued in Hu et al. (2013), since empirical data of human and wildlife diseases indicate that a nonlinear function may work better at modelling transmission rates when looking at the full spectrum of densities. However, the case where β is constant is also allowed in our framework, since as argued in Liu and Stechlinski (2012), Hethcote (2000) it seems more consistent with the known result that daily contact patterns are independent of community size.

For simplicity, we normalise here the total population to 1. This implies that μ is a probability density, so that

$$\int_{\mathbb{S}^1} \mu(x)\mathrm{d}x = 1 \ ,$$

if, in a population of size N distributed in space according to $\mu(x)$, one wants to write the dynamics for the actual numbers $S^{(N)} = NS$, $E^{(N)} = NE$, $I^{(N)} = NI$, $R^{(N)} = NR$, $D^{(N)} = ND$ of individuals in each class one finds, for example in the first equation of (2),

$$\begin{aligned}\partial_t S^{(N)}(t,x) &= -\beta^{(N)}\big(\mu^{(N)}(x) - D^{(N)}(t,x)\big)\frac{S^{(N)}(t,x)}{\mu}^{(N)}(x)\\ &\quad -D^{(N)}(t,x)\int_{\mathbb{S}^1} K_\chi(x,y)I^{(N)}(t,y)\mathrm{d}y,\end{aligned}$$

where $\mu^{(N)}(x) = N\mu(x)$ and $\beta^{(N)}(z) = \beta\left(\frac{z}{N}\right)$. The other equations in (2) can be derived similarly.

The change of variables gets slightly more complicated if we allow for demography, i.e. rates of births and deaths not due to the disease. We choose not to consider this extension here to simplify our exposition and presentation of numerical results.

2.2 The Mean Field Game model for the economy

The MFG whose stationary solution provides the exogenous spatial distribution for our epidemiological model is

$$\begin{cases} \rho V(t,x,h) = \frac{1}{2}\sigma^2 h^2 \partial^2_{hh} V(t,x,h) + \frac{1}{2}\varepsilon^2 \partial^2_{xx} V(t,x,h) \\ \qquad\qquad + H_1\big(x,h,\nu(t,h,x),\partial_h V(t,x,h)\big) + H_0(\partial_x V(t,x,h)) \ , \\ \partial_t \nu(t,x,h) = \frac{1}{2}\varepsilon^2 \partial^2_{xx}\nu(t,x,h) - \partial_x\left(\partial_p H_0(\partial_x V(t,x,h))\nu(t,x,h)\right) \\ \qquad\qquad + \frac{1}{2}\sigma^2\partial^2_{hh}(h^2\nu(t,x,h)) - \partial_h\left(\partial_p H_1(x,h,\nu(t,x,h)), \partial_h V(t,x,h)\nu(t,x,h)\right) \ . \end{cases} \tag{3}$$

where t denotes time, x position in $\mathbb{S}^1$ and h human capital. Even though several approaches are possible when measuring human capital in practice, in this paper we only focus on the theoretical side of the problem. To explain the meaning of all the terms appearing in (3) we need to discuss how one obtains such a system of PDEs. Indeed it arises as a continuum macroscopic description of a discrete (microscopic) multi-agent system whose features we outlined in the introduction: in such a system agents interact through their human capital and decide how to move in space and how to invest their current human capital in order to maximise a given utility function. The influence of nearby individuals on the dynamics of human capital constitutes an instance of spatial spillover on the accumulation and consumption of human capital. The system of PDEs encodes the balance between the effects on optimisation of a distribution $\nu(x, h)$ (described by the first equation in (3) which is a Hamilton–Jacobi–Bellman equation) and the effects on ν of adopting a strategy V (described by the second equation, a Fokker-Planck equation that is non-local due to taking interactions into account).

The MFG system is symmetric, in the sense that it arises from a model in which both the interaction function and the utility function are the same for each agent, and agents are thus exchangeable (see Sect. 2.2.1 below). Interaction is *weak*, meaning that no agent has a strong influence on the other agents' choices but the overall distribution has an impact on the optimisation problem of each agent. This effect is encoded by a spatial interaction term consisting of a weighted average of the human capital of nearby agents.

Heuristically, the MFG system is linked to the multi-agent system not only as a discrete-to-continuum approximation of the dynamics, but also in that solutions to the mean-field game are approximate Nash equilibria for the model with a finite (but large) number of agents; this feature has been rigorously proved only in simple cases (much simpler than the one we deal with herein) but is now widely accepted in economics and mathematical modelling the idea that MFGs are the correct tool to study situations as the one we are describing here, see for example Achdou et al. (2014).

Well-posedness of the mean-field game and the numerics developed to find a stationary solution are discussed in Ghilli et al. (2023); here we briefly describe the optimisation problem for the system of finitely many interacting agents, we link it formally to the mean-field game and comment on the numerical methods used to find μ.

2.2.1 The discrete multi-agent model

In what follows we denote by $\mathcal{P}_2$ the space of Borel probability measures on $\mathbb{S}^1 \times \mathbb{R}_+$ with finite second moment. Throughout this paper one can always assume that probability measures are absolutely continuous with respect to the Lebesgue measure on $[0, 1] \times \mathbb{R}_+$, implying that they have a density.

We consider a finite number $N \in \mathbb{N}$ of agents, indexed by $j = 1, \ldots, N$, whose positions and human capitals at time $t \geq 0$ are denoted respectively by $x_j(t) \pmod 1 \in \mathbb{S}^1$ and $h_j(t) \in \mathbb{R}_+$, $j = 1, \ldots, N$.

Each agent has control over her own position, choosing the control process $v_j(t)$ in the equation

$$\begin{cases} \mathrm{d}x_j(t) = v_j(t)\mathrm{d}t + \epsilon \mathrm{d}Z_j(t) & \text{for } t \geq 0, \\ x_j(0) = x_{j,0}, \end{cases} \tag{7}$$

where

(i) $\epsilon > 0$ is the noise intensity,
(ii) Z_j is a standard 1-dimensional Brownian motion and
(iii) $x_{j,0}$ are random variables taking values in $\mathbb{S}^1$, independent and identically distributed with some distribution μ_0^1, that we assume to be absolutely continuous and to have a finite second moment.

The spatial dynamics of the agents affect their human capital, which evolves according to the equation

$$\begin{cases} \mathrm{d}h_j(t) = f_j(t)h_j(t)^\alpha \bar{h}_{-j}(t)^\chi - \zeta h_j(t)\mathrm{d}t + \sigma h_j(t)\mathrm{d}W_j(t) & \text{for } t \geq 0, \\ h_j(0) = h_{j,0}, \end{cases} \tag{8}$$

where

(i) $\alpha, \chi \in (0,1)$ are constants such that $\alpha + \chi < 1$. This assumption means that we treat the case of decreasing returns to scale;
(ii) $f_j(t) \in [0,1]$ is the "investment in education" and can be chosen by the agent; the motivation for introducing f is that part of the agent's human capital, namely $f_j(t)h_j(t)^\alpha$, is used to increase the human capital itself (here $h_j(t)^\alpha$ is the "personal income"), while the remaining part $(1 - f(t))h_j(t)$ is used to produce final goods (see (10) below).
(iii) $\zeta > 0$ is the depreciation rate of human capital;
(iv) $\sigma > 0$ is the intensity of the noise, which is here chosen as a multiplicative noise to ensure the positivity of the solutions;
(v) W_j is a standard 1-dimensional Brownian motion;
(vi) $h_{j,0}$ are random variables taking values in $\mathbb{R}_+$, independent and identically distributed with some distribution μ_0^2, that we assume to be absolutely continuous and to have finite second moment;
(vii) $\bar{h}_{-j}(t)$ is an average depending on $x_j(t)$:

$$\bar{h}_{-j}(t) := \frac{\sum_{k \neq j} \eta\left(d_{\mathbb{S}^1}\left(x_k(t) - x_j(t)\right)\right) h_k(t)}{\sum_{k \neq j} \eta\left(d_{\mathbb{S}^1}\left(x_k(t) - x_j(t)\right)\right)}, \tag{9}$$

where the function η encodes the interaction between agents and is given by

$$\eta \colon [0, 1/2] \to \mathbb{R}_+$$
$$\eta(x) = 1 \text{ if } x \leq \epsilon_1, \quad \eta(x) = 0 \text{ if } \frac{1}{2} - \epsilon_2 \leq x \leq \frac{1}{2},$$

where $0 < \epsilon_1 < \frac{1}{2}$, $\frac{1}{2} - \epsilon_2 > \epsilon_1$, and $\eta(x)$ is continuous and affine between ϵ_1 and $\frac{1}{2} - \epsilon_2$; in this way η is Lipschitz continuous on its domain $[0, 1/2]$.

The function η should be thought of as a smooth version of the indicator function of a right-neighbourhood of 0; indeed $\eta\left(d_{\mathbb{S}^1}(x-y)\right)$ equals 1 whenever x and y are closer than ϵ_1, and equals 0 when x and y are far from each other. Therefore the interaction term $\bar{h}_j$ should be understood as follows: at each time t, agent j's human capital benefits mostly from that of all other agents that at time t are located within ϵ_1 from her. The value of human capital coming from this interaction term is divided by the number of agents that are located within ϵ_1 from her; this mean-field-type scaling is necessary to ensure that the interaction term does not blow up when the number of agents increases.

All initial conditions $x_{j,0}$ and $h_{j,0}$ and the Brownian motions Z_j and W_j are defined on some common probability space $(\Omega, \mathcal{F}, \mathbb{P})$. All the Brownian motions are supposed to be independent.

Both the equations for x_j and h_j depend on controls (respectively $v_j(\cdot)$ and $f_j(\cdot)$); for each j, the aim of agent j is to choose such controls as to maximise the gain functional

$$\begin{aligned} &J_j\left(x_{0,j}, h_{0,j}; v_j(\cdot), f_j(\cdot)\right) := \\ &\mathbb{E}\left[\int_0^{+\infty} e^{-\rho t}\left(u\left([(1-f(t))h_j(t)^\alpha]^{1-\gamma}\bar{h}_{-j}(t)^\gamma A\left(x_j(t)\right)\right)\right.\right. \\ &\left.\left.-a(v_j(t))\right) dt\right], \end{aligned} \tag{10}$$

where $\mathbb{E}$ denotes expectation with respect to $\mathbb{P}$. The parameters and functions appearing in the definition of J_j are:

(i) $\rho > 0$ is the discount factor;
(ii) u is the utility function, which takes the constant relative risk aversion form

$$u(z) = \frac{z^{1-p}}{1-p} \tag{11}$$

for some $p \in (0, 1)$.
(iii) $-a(v(t))$ represents the cost for the energy employed in the displacement;
(iv) $[(1-f(t))h_j(t)^\alpha]^{1-\gamma}$ represents the final goods produced thanks to the human capital;
(v) $\bar{h}_j(t)^\gamma$ is the spatial spillover on consumption;
(vi) $A(x)$ represents the local amenities, that is, location-dependent features that may make agents want to move also independently of interactions in human capital; here we choose $A(x) = \frac{1}{2}\sin\left(2\pi\left(x - \frac{1}{4}\right)\right) + 1$.

Each agent's choice of controls determines the general dynamics of all agents, because of the interaction term $\bar{h}_{-j}$.

2.2.2 The Mean-Field Game system

The multi-agent system described above is symmetric, in the sense that agents are indistinguishable in their contribution to each other's human capital and they are all maximising the same game functional; thus it is reasonable to look for Nash equilibria of the system. Nash equilibria are however impossible to compute exactly for a system with a large number of agents. Therefore a standard way to investigate equilibria is to take the limit as the number of agents goes to infinity and to study the limiting system. A solution to such limiting system will then approximate a Nash equilibrium for the original multi-agent system when the number N of agents is sufficiently large. The limiting system is a mean-field game, that is, it is made of an HJB equation coupled with a FP equation; in our model, the presence of the interaction term $\bar{h}_{-j}$ both in the dynamics and in the gain functional makes the MFG fully coupled.

To write down the MFG it is convenient to introduce the function $F : \mathbb{S}^1 \times \mathcal{P}_2 \to \mathbb{R}_+$ defined as

$$F(x,\omega) = \frac{\int_{\mathbb{S}^1 \times \mathbb{R}_+} \eta\left(d_{\mathbb{S}^1}(x,y)\right) k\omega\,(\mathrm{d}y, \mathrm{d}k)}{\int_{\mathbb{S}^1 \times \mathbb{R}_+} \eta\left(d_{\mathbb{S}^1}(x,y)\right) \omega\,(\mathrm{d}y, \mathrm{d}k)}; \tag{12}$$

in this way the interaction term $\bar{h}_{-j}(t)$ takes the form

$$\bar{h}_{-j}(t) = F\left(x_j(t), S(t)\right)$$

where $S(t)$ is the *empirical measure* of the system, defined as the sum of Dirac deltas

$$S(t) = \frac{1}{N} \sum_{j=1}^{N} \delta_{(x_j(t), h_j(t))} \,.$$

We now define the Hamiltonians of the two coupled control problems in the multi-agent system, one for the control v and the other for the control s. H_0 is defined as

$$\begin{aligned} H_0 &: \mathbb{R} \to \mathbb{R} \\ H_0(p) &= \sup_{v \in K} \{-a(v) + vp\}; \end{aligned}$$

H_1 instead is defined as

$$\begin{aligned} H_1 &: \mathbb{R}_+ \times \mathbb{S}^1 \times \mathbb{R}_+ \times \mathcal{P}_2 \times \mathbb{R} \to \mathbb{R} \\ &H_1(t,x,h,\omega,q) = \\ &\sup_{f \in [0,1]} \left\{ (fh^\alpha F(x,\omega)^\chi - \zeta h)q + u\left(A(x)[(1-f)h^\alpha]^{1-\gamma} F(x,\omega)^\gamma\right) \right\}, \end{aligned}$$

We thus eventually get to the anticipated MFG system associated to the optimal control problem (10),

$$\begin{cases} \rho V = H_1\big(x, h, \nu, \partial_h V\big) + \frac{1}{2}\sigma^2 h^2 \partial^2_{hh} V + \frac{1}{2}\varepsilon^2 \partial^2_{xx} V + H_0(\partial_x V) \,, \\ \partial_t \nu = \frac{1}{2}\sigma^2 \partial^2_{hh}(h^2 \nu) + \frac{1}{2}\varepsilon^2 \partial^2_{xx} \nu - \partial_x \left(\partial_p H_0(\partial_x V)\nu\right) - \partial_h \left(\partial_p H_1(x, h, \nu, \partial_h V)\nu\right) \,, \end{cases} \tag{13}$$

whose solution is a couple (V, ν) of functions of the three variables $t > 0$, $x \in \mathbb{S}^1$ and $h > 0$ such that for every $t > 0$ $\nu(t, \cdot, \cdot)$ is a probability density on $\mathbb{S}^1 \times \mathbb{R}_+$. Given ν, the function V solves the first PDE above in the classical sense, while given V the function ν solves the second PDE above in the sense of distributions. The system is equipped with an initial condition for ν, namely $\mu_0(x, h)$, such that its marginal with respect to x is μ_0^1 and its marginal with respect to h is μ_0^2. Moreover we need a set of boundary conditions for the h-boundary $\{h = 0\}$; we impose Dirichlet boundary conditions, namely

$$V(t, x, 0) = 0, \quad \nu(t, x, 0) = \bar{\mu}(t, x) \text{ for every } (t, x) \in \mathbb{R}_+ \times \mathbb{S}^1$$

where for every $t > 0$ $\bar{\mu}(t, \cdot)$ is a bounded probability density on $\mathbb{S}^1$ with finite second moment; this forces the compatibility condition

$$\mu_0(x, 0) = \bar{\mu}(0, x).$$

For every fixed function $\mathbb{R}_+ \ni t \mapsto \mu(t) \in \mathcal{P}_2$ the first equation in (13) is the HJB equation for the optimisation problem of a representative agent that interacts with a continuum of agents whose locations and human capitals are distributed according to μ; the value function for such optimisation problem is then a solution of such HJB equation. The second equation in (13) instead describes the time evolution of the space and human capital distribution of a continuum of agents, given a solution V of the first equation.

We stress that the Hamiltonian H_1 is not separable with respect to the distribution and the gradient of the value function.

As explained in the introduction of the paper and in the first paragraph of Sect. 2, in linking the dynamics of any space-time distribution of people to the dynamics of the epidemic we are forced to consider time-invariant distributions; we, therefore, use as our a priori spatial distribution the x-marginal of the μ-component of a stationary solution to (13). Such marginal is a probability density on $\mathbb{S}^1$.

We underline that here we find the stationary solution numerically. To rigorously prove existence and uniqueness of a stationary solution to the MFG system introduced above is still a mathematical challenge, due to the following reasons. First of all the typical assumptions under which existence and uniqueness have been proved so far are that the Hamiltonian in the HJB equation is separable with respect to the distribution of the agents and the gradient of the value function and there is a sort of monotonicity of the coupling. As already stressed above in our case we do not have separability of the Hamiltonian. Then since the standard condition of monotonicity requires separability, we cannot apply the standard theory for existence and uniqueness.

In the stationary case the only available works up to our knowledge for existence results for non separable Hamiltonians are Gomes and Mitake (2015), Gomes et al. (2014), Ferreira and Gomes (2018): in Gomes et al. (2014) very particular Hamiltonians are considered which do not cover our case and in Ferreira and Gomes (2018) existence is studied induced by monotonicity. In the time dependent case, the only available works up to our knowledge for existence results for non separable Hamiltonians are Cirant et al. (2020), Ambrose (2022), Gomes and Voskanyan (2015), Achdou and Porretta (2018). We stress that in Cirant et al. (2020), Ambrose (2022), Gomes and Voskanyan (2015) existence is proved under smallness conditions on the time horizon. Up to our knowledge, the only available work in the time dependent case without any restriction on the time horizon is Achdou and Porretta (2018). A particular case worth mentioning of non separable Hamiltonians which has been studied recently is that of congestion, see Gomes and Mitake (2015) (stationary case) and Gomes and Voskanyan (2015), Achdou and Porretta (2018) (time dependent case). However in Gomes and Mitake (2015) existence is proved under the main assumption that the Hamiltonian is quadratic, whereas in Gomes and Voskanyan (2015) the authors still do make structural assumptions such as monotonicity on part of the Hamiltonian and in Achdou and Porretta (2018) the authors assume a growth condition on the cost function which penalizes the motion where the distribution density is higher. The assumption of the smallness of the time horizon in the time dependent case in the above cited papers gives even more insight into the difficulties that one can get in studying the stationary case, which is inherently linked to the long-time stability of solutions of the time-dependent associated case.

2.2.3 Numerical solution to the steady state problem

In this section, we study the steady state of the system of equations (13) from a numerical viewpoint. Therefore we consider the following problem

$$\begin{cases} \rho V = H_1(x,h,\mu,D_h V) + \frac{1}{2}\sigma^2 h^2 D^2_{hh} V + \frac{1}{2}\varepsilon^2 D^2_{xx} V + H_0(D_x V) & \text{in } \mathbb{S}^1 \times \mathbb{R}_{++} \\ \frac{1}{2}\sigma^2 \partial^2_{hh}(h^2\mu) + \frac{1}{2}\varepsilon^2 \partial^2_{xx}\mu = \partial_x\left(D_p H_0(D_x V)\mu\right) + \partial_h\left(D_p H_1(x,h,\mu,D_h V)\mu\right) & \text{in } \mathbb{S}^1 \times \mathbb{R}_{++} \end{cases} \tag{14}$$

with Dirichlet boundary conditions as in the non stationary case

$$V(x,0) = 0, \quad \mu(x,0) = \bar{\mu}(x) \quad \text{in } \mathbb{S}^1.$$

The problem of finding numerical solutions to MFGs is in general very challenging. For a full discussion of the methodology applied to find numerical solutions to (14) we refer to Ghilli et al. (2023). In this manuscript, we limit ourselves to a brief outline of the main elements. The numerical discretization is performed by means of classical finite difference method. However, for MFGs problem one has to pay special attention to the way the discretization of the Hamiltonian is performed. In particular in order to ensure consistency of the discretization scheme we adopted the *Kushner-Dupuis* approach, see Achdou and Capuzzo-Dolcetta (2010) and also (Achdou et al., 2020, Chapter 2.) for a more general summary on the topic. The choice of an appropriate

Table 1 Parameters of system of equations (14)

Parameter	Value	Parameter	Value
L (length of the torus)	1	α	0.5
ε_1	0.3	χ	0.1
ε_2	0.1	γ	0.15
H_{max}	15	σ	0.7
$A(x)$	$\frac{1}{2}\sin(2\pi(x-\frac{1}{4}))+1$	ζ	0.15
ϵ	0.5	ρ	0.1

truncation of the domain $\mathbb{R}_+$ for the h variable is another difficult topic that one has to take care of. Numerical simulations in fact are performed on the truncated domain $\mathbb{S}^1 \times [0, H_{max}]$ where the upper bound value H_{max} has to be imposed artificially. In particular, we had to adopt an iterative strategy in the choice of H_{max} by computing a sequence of solutions with increasing H_{max} up until the bulk of the distribution of μ was fully enclosed in the truncated domain. See Table 1for a full list of the parameters used, including the threshold H_{max}.

One of the key parameters characterizing the stationary solution of the MFG is the function $A(x)$, $x \in \mathbb{S}^1$, describing the distribution over space of local amenities. In fact, in the stationary case, the only source of spatial heterogeneity is encoded in the exogenous function $A(x)$. This is not true in general when studying the time evolution of the system out of equilibrium, in which the initial distribution plays an important role in determining the spatial distribution of agents at any given period of time t. However, when studying the system at equilibrium the influence of the initial configuration vanishes as t grows to $+\infty$ and the only remaining factor affecting the shape of the solutions has to be accounted to the function A. In particular, one can see that in absence of heterogeneity, i.e. if $A(x)$ is uniform, solutions to system of equations (14) are translation invariant on the space variable. That is if one assumes that $(V(x,h), \mu(x,h))$ is a solution to the stationary problem, the same holds for $\big(V(x+x',h), \mu(x+x',h)\big)$ for any $x' \in \mathbb{S}^1$. On the contrary, when $A(x)$ is not uniform, the spatial distribution of agents is subject to a positive influx from local exogenous amenities. In particular one can imagine that being the objective functional (10) monotone increasing with respect to the function A, each agent will relocate trying to obtain the best possible level (net of reallocation costs) of local amenities, that is where $A(x)$ is higher. Figure 1shows the equilibrium solution to (14) when the function A has been taken to have a single maximum peak at $x = 0.5$. We can see in the central panel how the bulk of the population concentrates more in those areas corresponding to higher spatial amenities ($x \approx 0.5$), as well as around an intermediate value ($h \approx 5$) along the h variable. The optimal investment rate exhibits a decreasing behaviour with respect to the h variable, and even more reaches the value of zero in the area of the domain corresponding to highly educated individuals living within the central large city. We see in particular how agents distribute following the same shape as argued in the previous lines. See moreover Table 1 for a full list of parameters, including the function A.

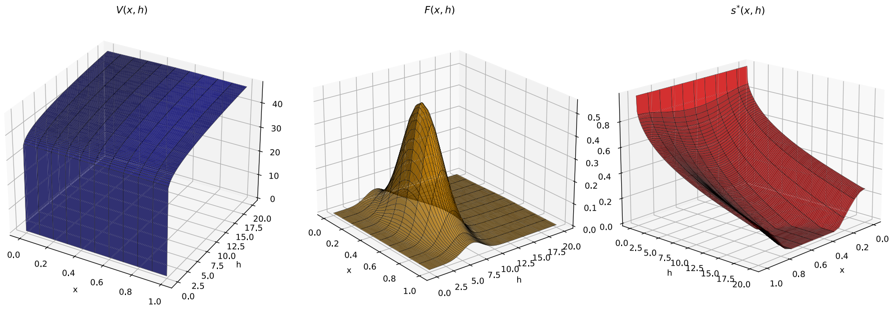

Fig. 1 Numerical equilibrium solution to system of equation (14). The left panel shows the plot of the value function $V(x,h)$ (blue), the one in the middle is that of the Fokker–Plank (right) and the rightmost one shows the optimal investment (red) (colour figure online)

The case shown in Fig. 1 is precisely the case we had in mind, and that we will also employ in the next section while discussing the spatial epidemic model (2). In particular, we imagine that having $A(x)$ with one single peak represents the presence of a single and large city whose centre is located in correspondence of the maximum of A. This idea is reinforced by the shape of $\mu(x,h)$ as shown in Fig. 1 where the bulk of the population concentrates in the middle.

3 Numerical results on the spatial SEIRD model with heterogeneous distribution

In this section, we present some qualitative results related to the SEIRD model (2) when the spatial distribution of agents is taken as the space marginal of the stationary distribution (14). We will see, with the aid of numerical simulations, how the population density plays a crucial role in the evolving of the epidemic through time.

The choice of parameters plays a key role in the evolution of epidemiological models. From the theoretical point of view, different choices may lead to different qualitative behaviour in the equilibrium, exhibiting a phenomenon of phase transition for the steady state. At the same time, different sets of parameters describe also specific characteristics of different diseases, as well as of that of the same disease in different countries. Several works, many of which appeared in recent years, focus on the problem of statistically estimating those parameters from available data.

In the present paper we don't aim to represent a real-world scenario, since we are still limited by the current level of mathematics and numerical capabilities, but to bring a methodological contribution. However, in order to be as close as possible to reality, we decided to consider the case of COVID-19 for the US, hence considering a set of epidemiological parameters as close as possible to the scenario we have in mind. As mentioned above, a plethora of papers are present on the topic of parameter estimation, but we take the work (Korolev 2021) as a reference, therefore taking $(\beta, a, \gamma, \delta) = (0.9, 0.25, 0.075, 0.025)$ (from the case $\sigma = 1/4$, $\gamma = 1/10$ in Korolev 2021). See moreover Table 2for a full list of parameters.

Table 2 Parameters of system of equations (2)

Parameter	Value	Parameter	Value
θ	0.25	χ	0.04
λ	0.075	I_0	0.01
δ	0.025	$r_{I,0}$	0.1
β (when constant)	0.9	$\beta(\mu)$	$0.9\frac{\sqrt{\mu(1+\mu)}}{\sqrt{2}}$

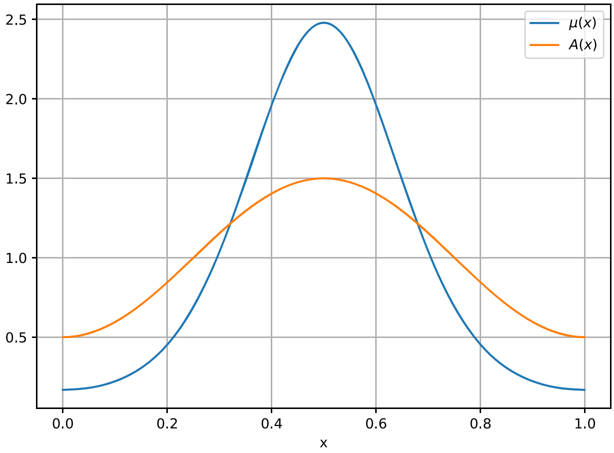

Fig. 2 The function $A(x)$ (orange) vs. the spatial distribution of agents $\mu(x)$ (blue) obtained from the Mean Field Game (colour figure online)

As previously mentioned in Sect. 2.2 the shape of A dictates the population distribution along the space variable. In Fig. 2we clearly see this fact, by noticing the shape of the function $\mu(x)$. We see how the spatial distribution of agents resembles the shape of $A(x)$. This accounts for the fact that the objective function (10) is monotone increasing in the function A with respect to the position of agents, and reflects the tendency of agents to concentrate more where local amenities are more present. Since the bulk of the mass of the population concentrates around the point 0.5, in order to better appreciate the effects of the population distribution, we take an initial configuration for the SEIRD model (2) where the outbreak of the epidemic origins from the outskirts. Hence, we set the initial condition to have a small cluster of infects centred around the point 0.3 in the class I. Namely we set

$$\begin{aligned} &S(0,x) = \mu(x) - I_0 \mathbb{1}_{B^\varepsilon(0.3, r_{0,I})}, \quad E(0,x) = 0, \\ &I(0,x) = I_0 \mathbb{1}_{B^\varepsilon(0.3, r_{0,I})}, \quad R(0,x) = 0, \quad D(0,x) = 0, \quad \forall x \in S^1 \end{aligned}$$

where $\mathbb{1}_{B^\varepsilon(0,r)}$ is a suitable mollification of the indicator function in order to have a smooth initial condition, and the parameters I_0 and $r_{I,0}$ are set to the values 0.01 and

0.1 respectively. In particular, define

$$\varphi(x) = \begin{cases} \exp\left(-\frac{1}{1-|x|^2}\right)/C_\varphi & \text{if } |x| < 1 \\ 0 & \text{if } |x| \geq 1 \end{cases}, \quad \varphi_\varepsilon(x) = \frac{1}{\epsilon}\varphi\left(\frac{x}{\epsilon}\right),$$

where C_φ is normalisation constant introduced so that φ integrates to one, and define

$$\mathbb{1}_{B^\epsilon(c,r)}(x) = (\mathbb{1}_{B(c,r)} * \varphi_\epsilon)(x) \quad \forall c, r.$$

Notice that the parameter $\epsilon > 0$ is just used as a regularization parameter for the indicator function. Also, it can be taken arbitrarily small since the function $\mathbb{1}_{B^\varepsilon(0,r)}(x)$ is of class C^∞ as soon as the parameter ϵ is positive, without any further assumption on its magnitude. Of course, many different initial conditions are possible. The one that we selected is chosen so that the initial cluster, centre in $x = 0.3$, is not located in the location of maximum density, $x = 0.5$. Moreover, being the model defined on the 1-dimensional torus, taking the initial cluster of infected to the farthest point from the peak of the agent distribution would make the Euclidean distance between the two points identical moving in either of the two possible directions (left or right). Therefore, we selected an initial condition which tries to avoid any kind of misleading results due to spatial symmetry.

Concerning the interaction kernel K_χ in equation (2) we recall that it describes the fact that people may get in contact not only with those individuals located in their same position but also from the neighbouring areas. In particular, we consider the function $\widetilde{K}$ as a suitable mollification of the *hat function* $H(x)$, defined as

$$H(x) = \begin{cases} 1 - |x| & \text{if } |x| < 1, \\ 0 & \text{otherwise.} \end{cases}$$

Then we rescale the kernel $\widetilde{K}$ as

$$\frac{1}{\chi}\widetilde{K}\left(\frac{x}{\chi}\right)$$

thus keeping the total area of the kernel untouched while changing the radius at which infections can be transmitted. This way of rescaling is fundamental in order to disentangle the effects of the parameter χ, which measures the radius of interaction, from the function β (parameter in case it is taken constant), which measures the transmission rate. In all the cases that we consider we set $\chi = 0.04$. Since our model is defined on the periodic one-dimensional space S^1 this value has no particular meaning in absolute terms. However, one important point to remark is that the selected value is chosen to be one order of magnitude smaller than the radius of interaction R of the Mean Field Game system (see Table 1). This reflects the fact that information, being, in general, an immaterial good, can travel much further (per unit of time) than viruses or people. Hence the exchange of information that agents receive, in the form

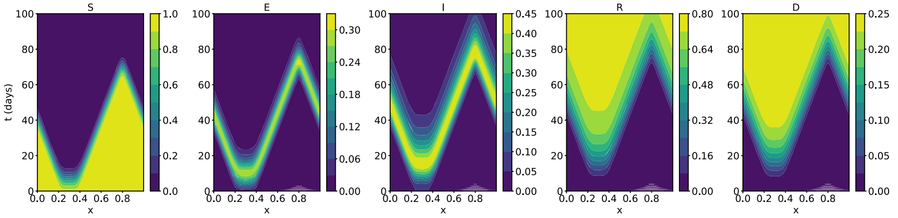

Fig. 3 Space–time evolution of the SEIRD model (2) with uniform population distribution $\mu(x) \equiv 1$. The horizontal axis represents the space variable, while time is shown vertically

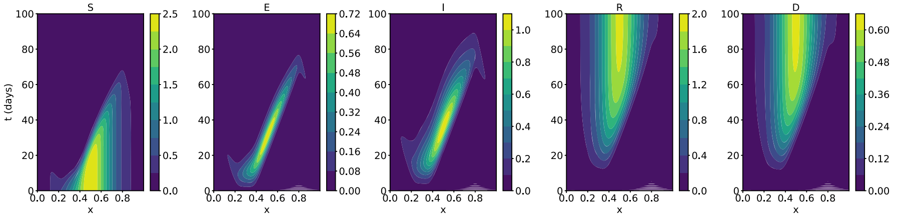

Fig. 4 Space–time evolution of the SEIRD model (2) with spatial distribution $\mu(x)$ obtained from the Mean Field Game system (14). The horizontal axis represents the space variable, while time is shown vertically

of spatial spillovers from other individuals, can be seen at a much higher distance than the radius of infection, therefore supporting a higher value of R with respect to χ.

We simulate the SEIRD model (2) by means of classical finite difference method. We run the model for a time period of 100 days, and analyse the result in a space-time plot, see Figs. 3 and 4. We first start by looking at the effect that population density has on the evolution of the pandemic. Figure 3 shows the case where the spatial distribution of population is neglected, and a completely flat distribution is taken, while the subsequent figure, Fig. 4, describes the same scenario but with population density as obtained from the Mean Field Game. In both cases β is constant. We see that the infection originates around $x = 0.3$ and briefly spreads to neighbouring locations. The farthest locations from where the infection outbreaks are the last ones to receive the contagious wave of infected, around $x \approx 0.8$. The final configuration approaches the uniformity in space in terms of deaths. The value of β is kept homogeneous as in Table 2. In Fig. 4 the infectious disease again originates around $x = 0.3$. We can appreciate from the graph how the diffusion of the virus favours the locations where the total population is higher. The final configuration shows the same behaviour. The value of β is kept constant as in Table 2 to the value 0.9. Finally, we also consider the case where the infection rate β is itself dependent on the density μ, see Fig. 5. This choice is not new and has been explored in Hu et al. (2013) taking into account the average contact rate each individual has. The rationale behind it is the following: the number of contacts of an average person has a direct impact on the characteristic infectious rate of a given disease (like COVID-19 as in our case). Therefore, it is reasonable to assume that, since the number of contacts per individual increases on average, as a function of the population density, so does the infection rate β. Therefore,

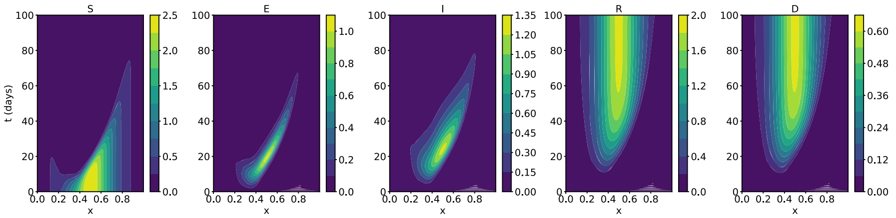

Fig. 5 Space–time evolution of the SEIRD model (2) with spatial distribution $\mu(x)$ obtained from the Mean Field Game system (14) and β depending on $\mu(x)$. The horizontal axis represents the space variable, while time is shown vertically

we introduce the function $\beta(\mu)$. Following (Hu et al. 2013, p. 129) we take

$$\beta(\mu) = 0.9\frac{\sqrt{\mu(1+\mu)}}{\sqrt{2}}.$$

The choice of the constants appearing in the function $\beta(\mu)$ has the following reasoning: the factor $\sqrt{2}$ is introduced so that, in the case of a uniform distribution for μ (in our case $\mu(x) \equiv 1$), then the right part of the formula reduces to the factor 1. By this choice, the factor 0.9 in front makes it so that when $\mu(x) \equiv 1$ the value of $\beta(\mu)$ coincides with the one used in the case where the distribution of agent is assumed uniform across the space, that is $\beta = 0.9$, see Fig. 3 for numerical simulations in that case. In Fig. 5 with respect to the scenario with constant β here we see the differences in the evolution of infected. In particular, the spreading of the disease is much more localised around the centre of the graph, where not only the population density is higher, but also the infection rate increases. The opposing effect happens closer to $x = 0$ (analogously $x = 1$ due to periodicity). See in particular panels corresponding to classes E and I.

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Conflict of interest The authors have no competing interests to declare that are relevant to the content of this article.

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