Geographical Heterogeneities and Externalities in an Epidemiological-Macroeconomic Framework*

Davide La Torre† Danilo Liuzzi‡ Simone Marsiglio§

Forthcoming in Journal of Public Economic Theory

Abstract

We analyze the implications of geographical heterogeneities and externalities on health and macroeconomic outcomes by extending a basic epidemiological-macroeconomic model to a spatial dimension. Because of people's migration and commuting across different regions, a disease may spread also in areas far from those in which the outbreak originally occurs and thus the health status (i.e., disease prevalence) in specific regions may depend on the health status in other regions as well. We show that neglecting the existence of cross-regional effects may lead to misleading conclusions about the long run outcome not only in single regions but also in the entire economy, suggesting that single regions need to coordinate their efforts in order to achieve disease eradication. We analyze such a coordination by focusing on a control problem in which the social planner determines globally the level of intervention showing that in order to achieve eradication it is essential to accompany traditional disease control policies (i.e., prevention and treatment) with regulations limiting people's movements. Focusing on COVID-19 we present a calibration based on Italian data showing that, because of the infections generated by cross-regional commuting, even vaccination may not be enough to achieve disease eradication, and limitations on people's movements need to accompany vaccination in order to preclude COVID-19 from reaching an endemic state.

Keywords: Macroeconomic-Epidemiological Model, Externality, Heterogeneity, Public Policy, Social Distancing; Spatio-Temporal Dynamics
JEL Classification: C60, I10, I18

1 Introduction

Infectious diseases have historically played a major role in shaping the development pattern of different economies, and also nowadays in certain regions and in particular in developing countries they still do. Because of their implications on morbidity and mortality, communicable diseases affect the labor market, individual decisions and public health policies, yielding detrimental consequences on economic prosperity through a number of different channels (Acemoglu and Johnson, 2007; Adda, 2016; Cervellati et al., 2017; Klasing and Milionis, 2020). This explains why the United Nations have included among their sustainable development goals for 2030 a specific target related to ending epidemics of HIV, tuberculosis, malaria and neglected tropical diseases, along with fighting hepatitis, water-borne diseases and other communicable

*We are grateful to an associate editor and two anonymous referees for their constructive comments on earlier drafts of the paper. All remaining errors and omissions are our own sole responsibility. Simone Marsiglio's research has been supported by the Italian Ministry of University and Research as a part of the PRIN 2017 program (grant protocol number 2017FKHBA8), and by the University of Pisa under the 'PRA - Progetti di Ricerca di Ateneo" (Institutional Research Grants) - Project no. PRA_2020_79 "Sustainable development: economic, environmental and social issues".

†SKEMA Business School, Université Côte d'Azur, Sophia Antipolis, France. Contact: davide.latorre@skema.edu.

‡University of Milan, Department of Economics, Management and Quantitative Methods, Milan, Italy. Contact: danilo.liuzzi@unimi.it.

§University of Pisa, Department of Economics and Management, Pisa, Italy. Contact: simone.marsiglio@unipi.it.

diseases (UN, 2015). Indeed, the World Health Organization estimates that even today communicable diseases determine 30% of deaths and account for 51% of the related years of life lost worldwide. Despite the large variability in these statistics between high and low income countries (WHO, 2009), due to the high degree of integration achieved in modern economies, and in particular through international trade and migration channels, the disease prevalence in specific regions is likely to affect the development process in other regions as well, thus health outcomes in developed and developing countries are likely to be strictly related. For example, despite the outbreak of the SARS (Severe Acute Respiratory Syndrome) firstly occurred in China in 2002 the disease rapidly spread well beyond Chinese national borders and in few months SARS cases were found in more than 30 countries and regions all over the world (Guan et al., 2003); such a geographical diffusion was largely due to the fast movements of international tourists and business travelers favored by the modern transport technologies (Kimball, 2006). Even more striking is the recent coronavirus (COVID-19) outbreak which, after its first diffusion in China in late 2019, has rapidly spread all over the world in a matter of weeks to reach a pandemic status in March 2020, generating thus far (at the time of writing, in September 2021) more than 220,000,000 cases globally, reported in almost every country (Dong et al., 2020; WHO, 2020a); individual countries' response to such a pandemic has been very variable, ranging from a complete lockdown of either the entire country or some regions to mandatory or voluntary quarantines, with important implications on the effectiveness of the implemented control measures both at global and local levels (Cheng et al., 2020). Understanding thus the extent to which the health conditions in an individual economy depend not only on its specific characteristics and policies, but also on health conditions and policies implemented in other economies as well, is essential to effectively design policies allowing to eventually achieve the health-related sustainable development goal. This is exactly the goal of this paper which wishes thus to shed some light on how geographical externalities, due to traveling and commuting, and geographical heterogeneities, related to the effectiveness of health policies and the initial spread of the disease, interact in determining the health status in single regions and in the entire economy.

In order to discuss such geographical effects in the simplest possible way we focus on a basic epidemiological model, in which the population is divided in two groups: the healthy individuals who are susceptible to infection and the already infected individuals who can transmit the disease to the healthy ones; since infectives recover from the infection at a certain speed, there are continuous flows between the two population subgroups. Such a formulation lies at the basis of the simplest mathematical epidemiology model, namely the susceptible-infected-susceptible (SIS) model, finding its origin in the seminal work by Kermack and McKendrick (1927). Its possible outcomes have been extensively discussed in literature and are now well known: if a certain parameter (the basic reproduction number) is small the disease will be completely eradicated, otherwise it will persist even in the long run (Hethcote, 2000). SIS-type frameworks have recently been analyzed in a growing economic epidemiology literature, which mainly wishes to understand how public policy and individual actions may combine to determine long run health outcomes (Philipson, 2000; Klein et al., 2007). Several works try to understand the optimal determination of different health programs, broadly categorized in either treatment or preventive measures, by analyzing an optimal control problem in which policymakers aim to maximize the social welfare or minimize the social cost associated with public policy (Gersovitz and Hammer, 2004; Goldman and Lightwood, 2002; Anderson et al, 2010; La Torre et al., 2020). For example, by focusing on a traditional SIS model, Gersovitz and Hammer (2004) show that several factors, including the biology of the disease and the relative costs of intervention, determine the optimal implementation of treatment and prevention. Most of the papers focus on a microeconomic partial equilibrium perspective, and only few of them adopt a macroeconomic point of view to characterize the mutual relation between health and macroeconomic outcomes (Chakraborty et al., 2010; Goenka and Liu, 2012: Goenka et al., 2014; La Torre et al., 2020; Gori et al., 2021a). For example, by analyzing a macroeconomic-extended SIS setup to account for the health-income feedback effects, La Torre et al. (2020) show that prevention is most desirable whenever the infectivity rate is low while treatment becomes most desirable whenever the infectivity rate is high. Following the recent COVID-19 experience which has shown the possible dramatic

macroeconomic consequences of infectious diseases, we relate to this macroeconomic-epidemiological literature to introduce a SIS model into a stylized macroeconomic framework in which only healthy individuals work, households entirely consume their disposable income and income taxes are employed to finance health policy measures (i.e., prevention and treatment) which by reducing disease prevalence allow to improve economic production capabilities and so macroeconomic outcomes. Different from all the extant works which completely abstract from a geographical dimension to characterize the spatio-temporal disease dynamics and the policy measures implemented to contrast its diffusion, we do introduce a spatial framework to quantify the implications of geographical externalities and heterogeneities on optimal policymaking.

In order to move in this direction we embed the basic SIS model with a spatial characterization. In our spatial framework the economy develops along a line and different points represent different locations; different locations differ both in the initial spread of the disease and in the degree of effectiveness of the provided health services affecting thus the basic reproduction number, which ultimately determines the long run health outcome achieved in that specific location (geographic heterogeneity). However, each location is not completely independent from others since people move from one location to the next within the spatial economy due to migration and commuting motives (captured by the presence of diffusion and local terms, respectively), and this implies that health outcomes in single locations depend also on health outcomes in others (geographical externality). This spatial structure allows us to capture well the variability in health conditions which we can observe in the real world both within a single economy and between economies. Indeed, several studies document the existence of a wide regional heterogeneity in health care across regions, states and countries (see Skinner, 2012, and references therein). Such heterogeneity involves both differences in health outcomes (i.e., substantial variation in life expectancy) and in health services provision (i.e., large variation in the quantity and quality of services provided), which are largely due to factors significantly differing across regions and countries including income, financial incentives, capacity, constraints, access, price and risk (Skinner, 2012). Our spatial analysis allows us to show that neglecting the existence of geographical externalities may lead to misleading conclusions about the health outcome achieved not only in single regions but also in the entire economy. This suggests that in order to eventually achieve disease eradication, as aimed for by the UN' sustainable development goals, a certain degree of coordination across different regions may be needed. This result confirms what found by earlier studies in completely different contexts but similarly stressing the importance of cooperation across regions or economies to effectively achieve disease eradication (Barrett, 2003), and suggests another possible explanation of why eradication has never been achieved thus far with the only exception of smallpox (World Bank, 1993). Since coordination is essential to achieve desirable health outcomes, we analyze cross-regional coordination by analyzing a spatial control problem in which the global social planner, given the availability of resources determined at macroeconomic level, determines the level of intervention in each single location by accounting for geographical externalities and heterogeneities within the spatial economy. By focusing on a calibration based on a widely spread infectious disease (i.e., the seasonal flu), we show that at global level it may not always be possible to achieve complete eradication through traditional control measures (i.e., prevention and treatment) because of people's movements. However, the introduction of economic regulations limiting such movements, like those aimed at imposing travel restrictions (i.e., local or global travel bans), can help to ensure long run eradication, representing thus an important tool to complement health policy measures.

The COVID-19 outbreak has renewed the interest in understanding the consequences of communicable diseases on both macroeconomic and health outcomes. A growing number of works have recently analyzed how macroeconomic activity is related to epidemic diseases by investigating the role of the most commonly used form of control strategy, that is social distancing policies, to limit the spread of the epidemic (Brodeur et al., 2021; Boucekkine et al., 2021). Several papers discuss the effects of different social distancing measures in the short (Acemoglu et al., 2021; La Torre et al., 2021a) and long run (Alvarez et al., 2021; Gori et al., 2021b), in centralized (Gollier, 2020; Caulkins et al., 2021) and decentralized settings (Eichenbaum et al., 2021; Rothert, 2021), in the contexts of delays in disease transmissions and policy implementation

(Aspri et al., 2021 Hritonenko et al., 2021). However, none of them accounts for the spatial implications of the disease dynamics,[1] thus we contribute to this literature by discussing how the presence of geographical effects may modify our conclusions regarding the effectiveness of different health policy and the design of the most appropriate policy-mix to control the (local and global) spread of the disease. Different from other epidemic management programs in the past when social distancing measures have been only seldom used, in the fight against the novel COVID-19 numerous forms of social distancing policies have been employed in most countries, including quarantines, voluntary isolations, lockdowns, the requirements to wear face masks and to maintain a certain physical distance from others. By limiting individual behavior and interactions between individuals such policy measures have led to an important reduction in the utilization of the workforce and in labor productivity resulting in dramatic economic effects, such as a large number of job losses and a large drop in GDP. Therefore, social distancing represents a specific type of preventive tool affecting not only health outcomes but also economic activities through its effects on production capabilities. In order to assess the effectiveness of the real world policy response to the recent coronavirus pandemic, including social distancing, treatment and lately vaccination, we develop an extension of our baseline model to account for the peculiarities of COVID-19 and quantify the effects of social distancing on both disease dynamics and economic activity and its interaction with vaccination. Since reinfections from COVID-19 are rare (but possible) we need to allow for the possibility that a part of the individuals recovering from the disease gain temporary immunity. As immunity (which can be acquired also through vaccination) vanishes individuals return to be susceptible to the disease again. Such a setting requires to introduce a further subpopulation group, the recovereds, transforming our framework into a SIRS (susceptibles-infectives-recovereds-susceptibles) setup. In this context we present a calibration of our extended model to the Italian COVID-19 experience in order to analyze whether optimal vaccination and social distancing policies, combined with optimal treatment efforts, may allow for disease eradication. Our analysis surprisingly shows that, because of the infections generated by cross-regional commuting, vaccination per se is not enough to achieve disease eradication. Regulations limiting people's movements need to accompany vaccination in order to preclude COVID-19 from reaching an endemic state and eradicate the disease.

The paper proceeds as follows. Section 2 briefly summarizes the basic structure of the SIS epidemiological model, by focusing in particular on the role played by the basic reproduction number in determining the long run health outcome and on how traditional public health policies (through prevention and treatment measures) can affect such a basic reproduction number and thus be used to effectively achieve complete eradication. Section 3 presents our extension to a purely dynamic spatial framework in order to account for both geographical heterogeneities and geographical externalities; we show that the predictions based on the basic reproduction number (and thus on the role of prevention and treatment) are likely to no longer hold true in the presence of geographical externalities since the spatial spread of the disease tends to alter substantially the long run health outcome. This allows us to stress the policy implications of our model since designing public health policies aiming at eradicating a disease without taking into account the policies implemented in other regions as well is likely to lead to undesirable outcomes. Section 4 focuses on optimal policymaking by analyzing a spatially-structured optimal control macroeconomic-epidemiological problem in which prevention and treatment are determined globally in order to maximize social welfare. This setup allows us to stress that, even if internalizing geographical externalities and heterogeneities, traditional disease control policies may not be enough to achieve disease eradication, which instead may require the introduction of economic regulations limiting people's movements through travel restrictions. Section 5 extends our baseline model to a SIRS context in order to account for the peculiarities of COVID-19 and of the main policy measures implemented to fight it (i.e., social distancing and vaccinations), presenting a calibration to the Italian experience to understand the effectiveness of real world policies and their implications on both economic

[1] Despite the issue has not been discussed in economic epidemiology yet, few studies in mathematical epidemiology have recently introduced a spatial framework in traditional epidemiological models to characterize the spatial spread of COVID-19 (Wu et al., 2020; Tsori and Granek, 2021).

and health outcomes. Section 6 as usual presents concluding remarks and proposes directions of future research.

2 The Basic Model

The susceptible-infected-susceptible (SIS) epidemiological model represents one of the simplest frameworks to analyze disease dynamics. It is best suited to discuss the implications of infectious diseases which do not confer immunity, including sexually transmitted diseases or diseases caused by bacteria (Feng et al., 2005), seasonal influenza and other diseases characterized by seasonal patterns (Martcheva, 2009). The demographic structure in the SIS model is extremely simple and this allows to characterize the evolution and the eventual spread of a certain disease across the population in a very intuitive way.

The population, N, which is assumed to be constant, is composed by two groups: individuals who are infected, I_t, and individuals who are not infected but susceptible to infection, S_t. Infected individuals spontaneously recover from the disease at a speed $\delta > 0$, while susceptible individuals contract the disease at rate $\alpha > 0$ by interacting through random matching with infected ones (α measures both the number of interactions between infectives and suceptibles and the probability with which an interaction gives rise to a new infection), and the probability with which matching occurs depends on the actual spread of the disease across the population. In order to keep the analysis as simple as possible, for the time being, we assume that public health policy is exogenously given and affects the speed of transmission and recovery from the disease; specifically, the prevention (or prophylactic intervention) rate, denoted with $0 \leq p \leq 1$ reduces the probability of infection, while the treatment (or therapeutic intervention) rate, $0 \leq v \leq 1$, speeds the recovery up. At any moment in time disease-specific (α and δ) characteristics, along with public policies (p and v), determine the flow of individuals between the two population subgroups. Given the initial conditions, $S_0 \geq 0$, $I_0 \geq 0$, and $N = S_0 + I_0 \geq 0$, the evolution of the number of infectives and susceptibles is described by the following differential equations:

$$\dot{S}_t = \delta(1 + v^\beta)I_t - \alpha(1 - p^\omega)\frac{S_t I_t}{N} \tag{1}$$

$$\dot{I}_t = \alpha(1 - p^\omega)\frac{S_t I_t}{N} - \delta(1 + v^\beta)I_t, \tag{2}$$

where $0 < \omega < 1$ and $0 < \beta < 1$ measures the elasticity of prevention and treatment respectively, which affect the dynamics of infectives and susceptibles less than linearly because both forms of health policy exhibit diminishing marginal products (Gersovitz and Hammer, 2004). The system above can be straightforwardly recast in terms of the share of infectives, $i_t = \frac{I_t}{N}$, and the share of susceptibles, $s_t = \frac{S_t}{N}$, as follows:

$$\dot{s}_t = \delta(1 + v^\beta)i_t - \alpha(1 - p^\omega)i_t s_t \tag{3}$$

$$\dot{i}_t = \alpha(1 - p^\omega)i_t s_t - \delta(1 + v^\beta)i_t. \tag{4}$$

Since $1 = s_t + i_t$, the epidemic dynamics can be completely characterized by focusing simply on one of the two equations as follows:

$$\dot{i}_t = \alpha(1 - p^\omega)i_t(1 - i_t) - [\delta(1 + v^\beta) + b]i_t. \tag{5}$$

The above equation conveniently describes the evolution of the disease prevalence in the entire population and analyzing its equilibria is straightforward. As discussed in the epidemiology literature, the long run outcome crucially depends on the *"basic reproduction number"* (Hethcote, 2000), $\mathcal{R}_0$, given by:

$$\mathcal{R}_0 = \frac{\alpha(1 - p^\omega)}{\delta(1 + v^\beta)}. \tag{6}$$

This term measures the average number of secondary infections produced by a typical infectious individual introduced into a completely susceptible population, and clearly the disease prevalence will tend to increase

(decrease) over time as long as this is larger (smaller) than unity, suggesting that the epidemic outcome in the long run completely depends upon the size of the basic reproduction number. Indeed, according to whether $\mathcal{R}_0$ is larger or lower than one, a different health outcome, entirely quantified by the disease prevalence, will be achieved in the long run. Whenever $\mathcal{R}_0 \leq 1$ the disease prevalence will necessarily shrink over time and in the long run the economy will reach a disease-free equilibrium in which $\bar{i}_1 = 0$ (and thus $\bar{s}_1 = 1$). Whenever $\mathcal{R}_0 > 1$ the disease prevalence will tend to reach a strictly positive level and in the long run the economy will reach an endemic equilibrium in which $\bar{i}_2 = \frac{\alpha(1-p^\omega)-\delta(1+v^\beta)}{\alpha(1-p^\omega)}$ (and thus $\bar{s}_2 = \frac{\delta(1+v^\beta)}{\alpha(1-p^\omega)}$). Note that while the disease-free equilibrium exists for all parameter values, the endemic equilibrium is well defined and exists only if $\alpha(1-p^\omega) > \delta(1+v^\beta)$, that is if the effective transmission rate (inclusive of prevention), $\alpha(1-p^\omega)$, is larger than the effective recovery rate (inclusive of treatment), $\delta(1+v^\beta)$. By analyzing the (local) stability properties of the equilibria, it is possible to show that $\frac{\partial \dot{i}_t}{\partial i_t}|_{i_t=\bar{i}_1} = \alpha(1-p^\omega) - \delta(1+v^\beta)$ and $\frac{\partial \dot{i}_t}{\partial i_t}|_{i_t=\bar{i}_2} = -[\alpha(1-p^\omega) - \delta(1+v^\beta)]$, which implies that when only the disease-free equilibrium exists this will be asymptotically stable, while when also the endemic equilibrium exists this will be asymptotically stable and the disease-free equilibrium will become unstable.

The basic reproduction number plays thus a crucial role in understanding what will effectively happen to the disease in the long run, independently of the initial level of prevalence. Only whenever $\mathcal{R}_0$ is lower than one, that is whenever the effective recovery rate is higher than the effective transmission rate, complete eradication will be possible. Since traditional public health policy is able to affect the basic reproduction number (which falls with both prevention and treatment), it can be effectively used in order to achieve a disease-free equilibrium characterized by complete eradication, and both prevention and treatment can be equivalently used in order to achieve such an outcome. This suggests that, independently of disease-specific characteristics (captured by α and δ), the disease can be effectively eliminated from the population provided that the basic reproduction number, $\mathcal{R}_0$, is brought to (or maintained at) a value less than unity through public policy tools.

3 The Spatial Model

We now focus on an extension of the basic SIS model to allow for geographical heterogeneities and externalities by introducing a spatial dimension. A similar setup has been recently used to discuss the geographical implications of economic growth (Boucekkine et al., 2009; La Torre et al., 2015; Bucci et al., 2019) and environmental problems (Brock and Xepapadeas, 2010; Camacho and Pérez–Barahona, 2015; La Torre et al., 2019, 2021b). We assume a continuous spatial structure to represent that the spatial economy develops along a linear city (see Hotelling, 1929; La Torre et al., 2021b), where the population is mobile across different locations and thus the infectious diseases may diffuse across the spatial economy as well (Dietz and Sattenespiel, 1995; Wang, 2014).

We denote with $S_{x,t}$ and $I_{x,t}$, respectively the susceptibles and infectives in the position x at date t, in a compact interval $[x_a, x_b] \subset \mathbb{R}$, and we assume that the so-called Neumann boundary conditions hold true, that is there are no migration flows through the borders of $[x_a, x_b]$ namely the directional derivatives are null, $\frac{\partial S_{x,t}}{\partial x} = \frac{\partial I_{x,t}}{\partial x} = 0$, at $x = x_a$ and $x = x_b$. In this framework, any position x may be interpreted as a specific location while a set of adjacent locations as a region in the spatial economy (La Torre et al., 2019). Different from what discussed earlier for the a-spatial model, the epidemic dynamics cannot be fully characterized by focusing only on the evolution of the share of infectives, since the population is spatially distributed, $N = \int_{x_a}^{x_b} N_{x,t} dx$ with $N_{x,t} = S_{x,t} + I_{x,t}$, and thus it is not necessarily true that the shares of infectives and susceptibles sum to one in each location x (i.e., they do sum to one over the whole spatial domain). In particular, the share of infectives, $i_{x,t} = \frac{I_{x,t}}{N}$, and the share of suscetibles, $s_{x,t} = \frac{S_{x,t}}{N}$, in each location x jointly determine the share of the total population residing in that specific location, $n_{x,t}$ with $n_{x,t} = s_{x,t} + i_{x,t}$, while the sum of the shares of the total population residing in all locations sum to one,

$\int_{x_a}^{x_b} n_{x,t}dx = 1$. Therefore, we need to analyze the evolution of the share of infectives and suscetibles over time and across space, and the spatial model can be represented though a system of partial differential equations as follows:

$$\frac{\partial s_{x,t}}{\partial t} = d\frac{\partial^2 s_{x,t}}{\partial x^2} + \delta(1+v_x^{\beta})i_{x,t} - \alpha(1-p_x^{\omega})\int_{x_a}^{x_b} s_{x',t}i_{x',t}\varphi_{x',x}dx' \tag{7}$$

$$\frac{\partial i_{x,t}}{\partial t} = d\frac{\partial^2 i_{x,t}}{\partial x^2} + \alpha(1-p_x^{\omega})\int_{x_a}^{x_b} s_{x',t}i_{x',t}\varphi_{x',x}dx' - \delta(1+v_x^{\beta})i_{x,t}. \tag{8}$$

However, by recalling that $n_{x,t} = s_{x,t} + i_{x,t}$, it follows that $n_{x,t}$ solves the summation of equations (7) and (8), that is:

$$\frac{\partial n_{x,t}}{\partial t} = d\frac{\partial^2 n_{x,t}}{\partial x^2}, \tag{9}$$

with Neumann boundary conditions and initial conditions directly determined from those related to $s_{x,t}$ and $i_{x,t}$. This allows us to consider $n_{x,t}$ as a known exogenous variable, which thus can be substituted in (7) and (8) by writing $s_{x,t} = n_{x,t} - i_{x,t}$.

$$\frac{\partial n_{x,t}}{\partial t} = d\frac{\partial^2 n_{x,t}}{\partial x^2} \tag{10}$$

$$\frac{\partial i_{x,t}}{\partial t} = d\frac{\partial^2 i_{x,t}}{\partial x^2} + \alpha(1-p_x^{\omega})\int_{x_a}^{x_b} (n_{x',t} - i_{x',t})i_{x',t}\varphi_{x',x}dx' - \delta(1+v_x^{\beta})i_{x,t}. \tag{11}$$

The expression of $n_{x,t}$ is known in closed-form as it is the solution to the classical heat equation with Neumann condition and it reads as follows:

$$n_{x,t} = \sum_{n\geq 0} B_n e^{-\left(\frac{n\pi}{x_b - x_a}\right)^2 dt} \cos\left[\frac{n\pi(x-x_a)}{x_b - x_a}\right] \tag{12}$$

where $B_0 = \frac{1}{x_b - x_a}\int_{x_a}^{x_b} n_{x,0}dx$ and $B_n = \frac{2}{x_b - x_a}\int_{x_a}^{x_b} n_{x,0}\cos\left[\frac{n\pi(x-x_a)}{x_b - x_a}\right]dx$. In order to understand the disease dynamics, we can thus analyze the system of of partial differential equations (PDEs) given by (10) and (11), which generalizes to a spatial dimension the a-spatial model discussed in the previous section (see equation (5)) to account for population mobility across locations. In particular, equation (10) states that the population diffuses across space, from locations more populated to locations less populated, and since the population is composed by susceptibles and infectives also the two sub-population groups follow a similar diffusion pattern (see equations (7) and (8)).[2] As people move towards locations less densely populated, their individual probability of infection decreases but at the same time the degree of infection in locations receiving inflows of individuals from surrounding locations increases, meaning that individuals' attempts to reduce their disease exposure generates an externality which increases infection also in locations initially characterized by low population density and low disease prevalence. The use of similar reaction-diffusion systems to characterize the spatial spread of infectious diseases has been extensively used in mathematical epidemiology (Martcheva, 2015; Anita and Capasso, 2017).[3]

[2]Note that our epidemiological setting describes at the aggregate macro level the behavior of the population, and such an aggregative behavior derives from the behavior at the micro level of the different individuals in the population. Specifically, our setup implicitly assumes that individuals move randomly within the spatial domain following a Brownian motion, and this is reflected by the Laplacian terms in the PDEs while the diffusion parameters (assumed to coincide between the different population subgroups) measure the amplitude of the Brownian motions characterizing individuals' spatial movements (Ducasse, 2020).

[3]Apart from the use of reaction-diffusion partial differential equations, another approach frequently employed in mathematical epidemiology to capture the spatial dimension consists of adopting a network-based or a patch structure. When a patch framework is viewed on a sufficiently large scale it can be very-well approximated by a reaction-diffusion model which allows to characterize the spread of an infectious disease in a continuous spatial domain. Therefore, our setup can be interpreted as a limit version of a patch model in which individuals' mobility across a discrete number of locations determines the epidemic dynamics across space and over time (Dietz and Sattenespiel, 1995).

Therefore, the spatial structure of our framework allows us to characterize geographical externalities and heterogeneities. Geographical externalities are captured by two terms, the diffusion, $d\frac{\partial^2}{\partial x^2}$, and the integral term, $\int s_{x',t} i_{x',t} \varphi_{x',x} dx'$, which describe how infectives and susceptibles tend to move across the spatial economy and which thus imply that the infection even if originated in a specific location can spread also to other locations as well. Specifically, the former term describes movements due to migrations and relocations which take place over time and lead individuals to permanently move from one location to an adjacent one; the coefficient $d = d_I = d_S \geq 0$ represents the diffusion parameter, assumed to be the same for both infectives and susceptibles, which measures the speed at which such cross-regional effects take place. The latter term describes instead movements due to commuting and business trips which take place on a daily basis and lead individuals to move for a short period of time to locations even far away from their original venues; independently of their origin x', because of such types of movements on a daily basis individuals get in contact with a number of individuals originally located even far away, thus all the contacts between infectives and susceptibles contribute to determine the spread of the disease in the location x; the kernel $\varphi_{x',x}$ measures the extent to which these contacts between individuals originated from different locations matter for the disease prevalence in one specific location, quantifying the magnitude of such effects. While the diffusion term describes a "dynamic externality" whose effects are generated over time, the integral term describes a "static externality" with effects occurring instantaneously (La Torre et al., 2015); the diffusion and integral effects jointly characterize the geographical externalities which determine the evolution of the disease prevalence across the entire spatial economy. Geographical heterogeneities are captured by the two factors: the different level of disease prevalence across space, described by the non-homogenous initial spatial distribution of susceptibles $s_{x,0}$ and infectives $i_{x,0}$, and the eventual variability in epidemic and policy characteristics across space, described by the spatial-dependance of the preventive, p_x, and treatment measures, v_x. Differences in the initial conditions reflect the disease history in specific locations which may lead some regions to experience higher disease prevalence than others; for example, an outbreak is typically well localized initially and only over time the disease may spread in other locations. Differences in epidemic and policy characteristics reflect the situation that in each single venue the rates of prophylactic or therapeutic interventions may be different from those in others, because of their specific availability of resources (i.e., hospital beds, hospital personnel) or the local effectiveness of the implemented measures (i.e., individual attitude towards the adoption of preventive behavior).

Formally analyzing partial differential equations and their short run and long run outcomes is not straightforward from a mathematical point of view and goes well beyond the scope of this paper (see Polyanin, 2002, for a rigorous presentation and discussion). Therefore, we proceed in our analysis by illustrating the implications of geographical heterogeneities and externalities graphically. We perform some numerical simulations based on a calibration of our model to the specific case of the seasonal flu; it is however possible to show that our qualitative results are robust even when calibrating the model to consider the peculiarities of other diseases (such as the common cold). The seasonal flu is a common and widely spread disease resulting every year in about 3 to 5 million cases of severe illness and about 250,000 to 500,000 deaths at world level (WHO, 2018). Epidemiological studies provide accurate estimates of the main biological characteristics of different diseases, which allow to calibrate the relevant parameters. Specifically, the recovery rate can be computed as the inverse of the average number of days to recover which, combined with the basic reproduction number, from (6) allows us to compute the infectivity rate (in the absence of health policy measures) as $\alpha = \delta \mathcal{R}_0$. Estimates for the seasonal flu suggest that $\mathcal{R}_0 = 1.5$, and people generally recover in about seven days, which implies $\delta = 0.14$ and $\alpha = 0.21$ (La Torre et al., 2021a). Without loss of generality, we set the initial condition for the share of infectives to 0.05 and the diffusion parameter to 0.01, namely $i_{x,0} = 0.05$ and $d = 0.01$, but it is possible to show that results do not qualitatively change with different values of the parameters (La Torre et al., 2021a). To the best of our knowledge, estimates of the elasticities of preventive and treatment measures are not available to calibrate the relevant parameters, thus in the following we shall arbitrarily set $\beta = \omega = 0.5$ (but also in this case changing these parameter values

will not alter our qualitative conclusions) while we shall change the values of p and v to show how different policy intensities may affect our analysis. In particular, in order to make the figures as clear as possible in the lateral regions ($x \in [-1, -1/3)$ and $x \in (1/3, 1]$) we shall set $p = v = 0.6$, while in the central region ($x \in [-1/3, 1/3]$) either $p = v = 0.04$ (in case I) or $p = v = 0.001$ (in case II).

In order to isolate the effects due to heterogeneity in the health status and heterogeneity in the provision of health services, for the time being we assume that the spatial economy is characterized by the same initial level of disease prevalence (initial same health status) but regions within the spatial economy differ in terms of the effectiveness of the health services (different provision of heath services) which may reflect differences in income, capacity, or access (Skinner, 2012). Specifically, in order to make a comparison with the a-spatial model earlier described, in this section we focus only on the implications of spatial variability in the health services effectiveness by assuming that there are no differences in the initial health status, which will be introduced in the next section when we analyze optimal policymaking in our spatial context. In order to make our arguments as intuitive as possible, we also assume that biological factors characterizing the spread of the disease are the same in the entire spatial economy, meaning that geographical factors do not influence the speed of recovery and the probability of transmission[4]. Such heterogeneity in the effectiveness of public policy implies that the basic reproduction number $\mathcal{R}_0$ (see equation (6)) may be spatially heterogeneous as well. By relying on our analysis from the previous section we can predict the health outcome in different regions: regions characterized by high effectiveness of health services will achieve a disease-free equilibrium (i.e., $\mathcal{R}_0$ may be lower than one), while those characterized by low effectiveness will achieve an endemic equilibrium (i.e., $\mathcal{R}_0$ may be higher than one). We wish thus to understand whether such conclusions hold true even in the presence of geographical externalities and, if they do not, what role geographical externalities may play in shaping the evolution of the disease prevalence in the spatial economy. In order to look at this, we consider a framework in which health policy measures (prevention and treatment rates) are low in the central region (thus $\mathcal{R}_0$ is high) and high in the lateral ones ($\mathcal{R}_0$ is low), but there is some difference in the policy levels in the central region; specifically, in case I policy measures are relative higher than in case II.

In order to distinguish between the implications of the dynamic and static externalities, we first abstract from the static externality by setting the Dirac's delta function as the kernel, and we reintroduce such a static effect later by changing our specification of the kernel. By choosing the Dirac's delta function as the kernel, that is $\varphi_{x',x} = \begin{cases} \infty & x = x' \\ 0 & x \neq x' \end{cases}$, the above system of PDEs reads as follows:

$$\frac{\partial n_{x,t}}{\partial t} = d\frac{\partial^2 n_{x,t}}{\partial x^2} \tag{13}$$

$$\frac{\partial i_{x,t}}{\partial t} = d\frac{\partial^2 i_{x,t}}{\partial x^2} + \alpha(1 - p_x^{\omega})(n_{x,t} - i_{x,t})i_{x,t} - \delta(1 + v_x^{\beta})i_{x,t}, \tag{14}$$

clearly suggesting that the disease is transmitted only by interactions between susceptibles and infectives located in the same venue x (i.e., in the absence of commuting and business trips, the spread of the disease takes place slowly due to the infections generated by migration and relocations). This allows us to focus only the effects of diffusion (dynamic externality) on the disease prevalence in different regions and in the entire economy. The outcome of our analysis based on (13) and (14) is illustrated in Figure 1 (case I) and Figure 2 (case II).

The result of our simulations from case I is shown in Figure 1. In the left panel we represent the spatio-temporal epidemic dynamics in the absence of diffusion which perfectly confirms our predictions from the previous a-spatial section: the central region converges towards an endemic equilibrium while the lateral regions to a disease-free equilibrium. In the right panel we represent the dynamics in the presence of diffusion which clearly shows that the long run health outcome is substantially different from what we would expect

[4]Heterogeneity in disease characteristics can be equivalently interpreted as heterogeneity in the effectiveness of the health services provided, thus we believe that such an additional source of heterogeneity can be ignored with no loss of generality.

relying on a completely a-spatial approach as in the previous section: the spatial economy converges overall towards a disease-free equilibrium, since even the central region which is meant to achieve an endemic equilibrium with no dynamic externalities achieves a disease-free equilibrium thanks to such cross-regional effects. This suggests that while geographic heterogeneity in the long run health outcome of different regions exists with no externalities, in the presence of externalities such heterogeneity completely disappears and disease eradication can be achieved in each single location.

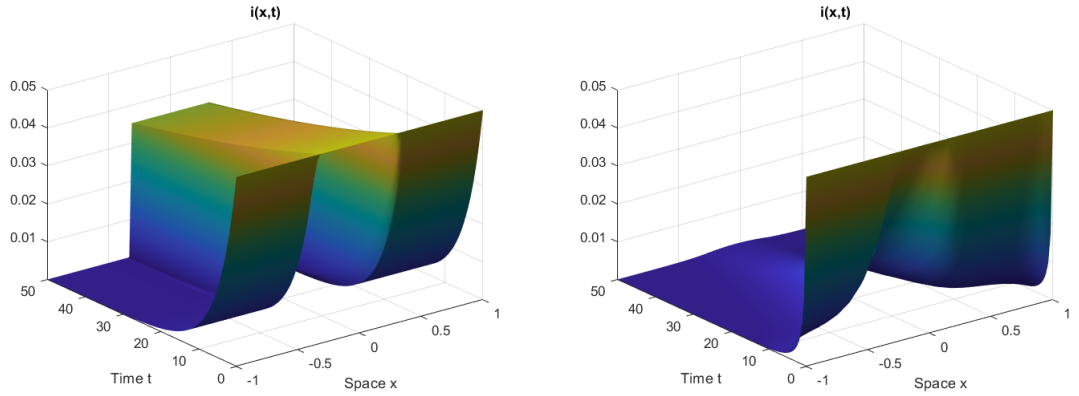

Figure 1: Evolution of the share of infectives in the spatial economy, with no diffusion (left) and with diffusion (right), in case I (prevention relatively high in the central region). No static externality.

The result from case II is instead shown in Figure 2. On the left we represent the spatio-temporal epidemic dynamics in the absence of diffusion which is qualitatively identical to what discussed for the case I, apart from the fact that the endemic equilibrium which the central region converges to is characterized by a higher level of prevalence. On the right we represent the dynamics in the presence of diffusion which shows that also in this case the long run health outcome is substantially different from what we would expect: the spatial economy converges overall towards an endemic equilibrium, since even the lateral regions which are meant to achieve a disease-free equilibrium with no dynamic externalities achieve an endemic equilibrium thanks to such cross-regional spillovers. In this case geographical heterogeneity in the long run health outcome of different regions exists both in the absence and in the presence of externalities; however, geographical externalities tend to smooth this heterogeneity out.

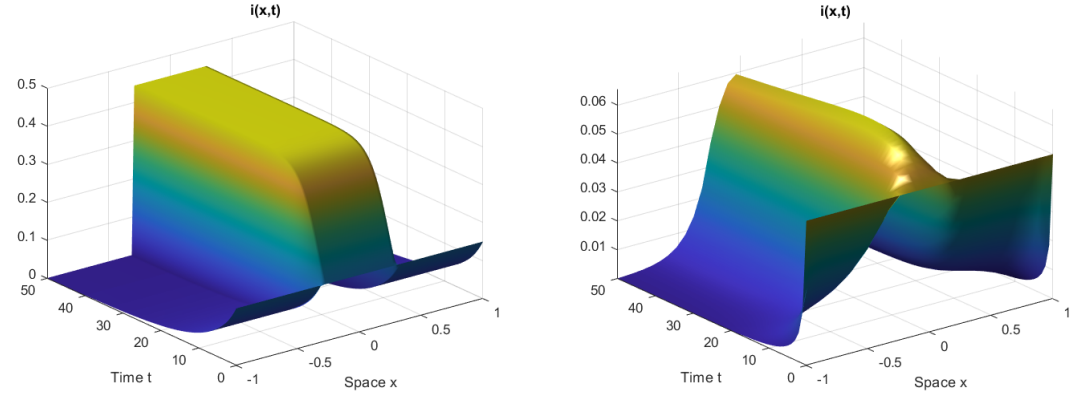

Figure 2: Evolution of the share of infectives in the spatial economy, with no diffusion (left) and with diffusion (right), in case II (prevention relatively low in the central region). No static externality.

By comparing Figure 1 and 2 we can derive some interesting conclusions. Because of geographical

externalities single regions are mutually related and thus the long run health outcome in a specific region does not depend only on its own public health choices but also on those in other regions as well. This implies that geographical heterogeneities in long run health outcomes of different regions may tend to be smoothed out. Moreover, it is possible that regions meant to achieve a good (bad) health outcome will experience a deterioration (improvement) in their health status in the long run because of geographical spillovers. Which of these two alternative situations is most likely to occur crucially depends on the overall level of prevalence in the spatial economy; indeed, while spatial diffusion will tend to improve the outcome in the central region (characterized by an endemic equilibrium in the absence of diffusion), what happens to the lateral regions (characterized by a disease-free equilibrium) is uncertain, and in particular if the share of infectives in the entire spatial economy is too high the outcome in lateral regions will tend to deteriorate. Neglecting to account for geographical externalities is likely thus to distort our predictions about long run health outcomes, potentially leading to sub-optimal policies; for example, by considering such externalities in case II the lateral regions could realize that it might be enough to increase the effectiveness of their health services (by increasing either prevention or treatment measures) in order to achieve complete eradication as in case I. These results clearly suggest that in order to effectively achieve disease eradication in a specific region or in the entire spatial economy single regions need to take into account the policies implemented in other regions as well and eventually coordinate their efforts to reach a common goal.

After discussing the effects of the dynamic externality, we now focus on the implications of the static externality by setting the kernel as follows: $\varphi_{x',x} = \frac{1}{\sqrt{2\pi}} e^{-\frac{(x-x')^2}{2}}$, suggesting that the static effects are stronger between adjacent locations and become weaker as the distance between locations increases (i.e., commuting and business trips typically take place not too far away from the origin). Such a specification of the kernel reintroduces the integral term as in (7) and (8), which now read as follows:

$$\frac{\partial n_{x,t}}{\partial t} = d\frac{\partial^2 n_{x,t}}{\partial x^2}, \tag{15}$$

$$\frac{\partial i_{x,t}}{\partial t} = d\frac{\partial^2 i_{x,t}}{\partial x^2} + \alpha(1-p_x^{\omega}) \int_{x_a}^{x_b} \frac{1}{\sqrt{2\pi}} (n_{x',t} - i_{x',t}) i_{x',t} e^{-\frac{(x-x')^2}{2}} dx' - \delta(1+v_x^{\beta}) i_{x,t}. \tag{16}$$

The implications of the static externality are illustrated in Figure 3, where in the left panel we represent the spatio-temporal epidemic dynamics in case I, while in the right panel the dynamics in case II. It is straightforward to realize that the disease prevalence with static effects is higher in each location than the corresponding level in the absence of static effects, both for case I (compare the left panel in Figure 3 with the right panel in Figure 1) and case II (compare the right panel in Figure 3 with the right panel in Figure 2). The result is extremely intuitive: since the static externality introduces an additional means of disease transmission (i.e., commuting and business trips), the health outcome tends to be always poorer than what we would experience in the absence of static externalities. In particular we can note that both in case I and case II each region in the spatial economy converges to an endemic equilibrium and geographical heterogeneity in their long run health outcome arises.

The analysis of the above results allows us to derive another interesting conclusion. Geographic externalities do not only imply that different regions are connected and thus they need to coordinate their efforts to achieve eradication, but they also suggest that quantifying the speed at which a disease propagates is not simple at all. Indeed, the potential existence of static externalities implies that assessing the level of prevention and treatment needed in the overall economy to achieve complete eradication may be extremely difficult. In order to see why this might be the case, focus on case I: if it is true that in the absence of static externalities the spatial economy can achieve complete eradication, when such externalities are taken into account this is no longer true and the entire economy converges to an endemic equilibrium. This suggests that policymakers need to carefully consider all the potential means by which a certain disease may be transmitted and quantify their effects in order to determine the required level of prevention and treatment to effectively achieve disease eradication. It may even be possible that simply relying on traditional health

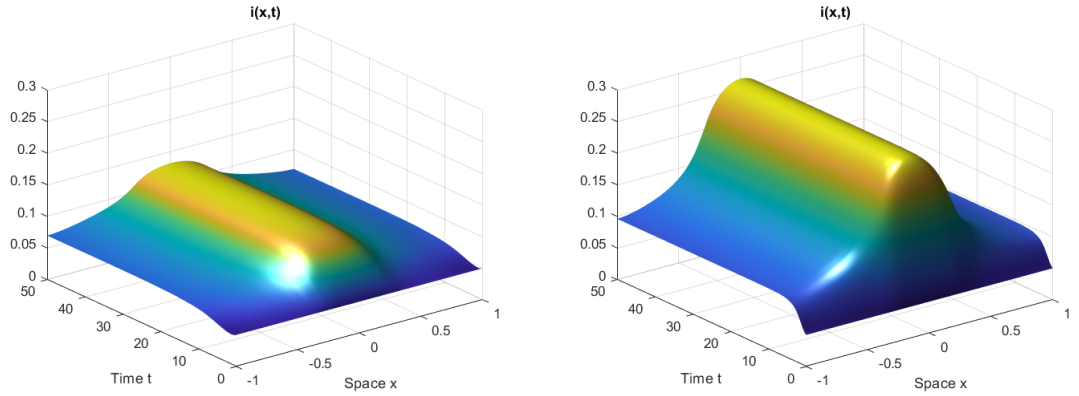

Figure 3: Evolution of the share of infectives in the spatial economy with diffusion and integral term, in case I (left) and in case II (right).

policies in the form of preventive and treatment measures is not enough to achieve disease eradication. If this is the case economic policies in the form of the introduction of regulations imposing travel restrictions and thus limiting the effects of geographical externalities (i.e., travel bans within the spatial economy precluding cross-regional movements), may be the only possibility to effectively achieve eradication. We shall analyze this argument with more depth in the next section when we discuss the determination of optimal epidemic control policies in a macroeconomic-epidemiological setting.

4 Optimal Spatial Policymaking

Thus far we have discussed health policies in a completely exogenous fashion, while we now endogeneize them by optimally determining the level of health expenditure to be allocated to either prevention or treatment at macroeconomic level. As epidemic management programs tend naturally to be short-lived, we focus on a finite time horizon framework in which the social planner tries to maximize social welfare by choosing the optimal level of preventive, $p_{x,t}$ and therapeutic, $v_{x,t}$, treatment. Similar to La Torre et al. (2020, 2021a), health policy measures are completely publicly funded, and the government levies lump sum taxes on individuals' income in order to finance prevention and treatment by maintaining a balanced budget at any moment in time. Over a short time frame saving and capital accumulation play only a marginal role and so we assume that individuals entirely consume their disposable income, which equals the difference between income and the taxes employed to finance public health policies: $c_{x,t} = y_{x,t} - p_{x,t} - v_{x,t}$, where $c_{x,t}$ denotes consumption and $y_{x,t}$ income. Output is produced through a linear technology employing only healthy individuals: $y_{x,t} = s_{x,t} = n_{x,t} - i_{x,t}$. Social welfare is the weighted sum of two terms: the discounted sum of utilities ($\rho > 0$ is the discount factor) and end-of-planning horizon utility, with $\phi \geq 0$ representing the relative weight of the latter term in terms of the former. As in Boucekkine et al. (2009), the instantaneous utility function is linear and depends on consumption, which at the end-of-planning horizon perfectly coincides with income as health policy measures and so taxes are null at the end of the epidemic management program. Given the initial conditions $s_{x,0} > 0$, $i_{x,0} > 0$, and $n_{x,0} = i_{x,0} + s_{x,0}$ the global social

planner's optimization problem reads as follows:

$$\begin{aligned}
\max_{p_{x,t},v_{x,t}} \quad & W = \int_0^T \int_{x_a}^{x_b} \left(n_{x,t} - i_{x,t} - p_{x,t} - v_{x,t}\right) e^{-\rho t} dx dt + \phi \int_{x_a}^{x_b} (n_{x,T} - i_{x,T}) e^{-\rho T} dx \\
s.t. \quad & \frac{\partial n_{x,t}}{\partial t} = d \frac{\partial^2 n_{x,t}}{\partial x^2} \\
& \frac{\partial i_{x,t}}{\partial t} = d \frac{\partial^2 i_{x,t}}{\partial x^2} + \alpha (1 - p_{x,t}^{\omega}) \int_{x_a}^{x_b} (n_{x',t} - i_{x',t}) i_{x',t} \varphi_{x',x} dx' - \delta (1 + v_{x,t}^{\beta}) i_{x,t} \\
& n_{x,0}, i_{x,0} > 0 \text{ given}
\end{aligned} \tag{17}$$

and, similar to what seen in section 2, the expression of $n_{x,t}$ is known and provided by (12). In our above formulation, since the economy is spatially structured social welfare is defined as the sum of utilities across all locations within the entire spatial economy, represented by the spatial integral in the objective function. By removing the spatial dimension and the macroeconomic considerations in the above problem, our model boils down to a simplified version of Gersovitz and Hammer's (2004). Other formulations to analyze optimal control policies in SIS-type epidemiological models have been proposed (Anderson et al, 2010; Goldman and Lightwood, 2002; Rowthorn and Toxvard, 2012; La Torre et al., 2020, 2021a), but to the best of our knowledge none has ever considered how the introduction of a spatial dimension and eventual geographical heterogeneities affect optimal policymaking. Our spatially-structured optimal control formulation allows us to understand how the presence of such geographical externalities affect policymaking in single regions within the entire spatial economy, analyzing eventual free-riding effects.

The generalized current value Hamiltonian function, $\mathcal{H}(i_{x,t}, n_{x,t}, p_{x,t}, v_{x,t})$, where $n_{x,t}$ acts as a mere auxiliary variable, reads as:

$$\mathcal{H} = n_{x,t} - i_{x,t} - p_{x,t} - v_{x,t} + \lambda_{x,t} \left[d \frac{\partial^2 i_{x,t}}{\partial x^2} + \alpha (1 - p_{x,t}^{\omega}) \int_{x_a}^{x_b} (n_{x',t} - i_{x',t}) i_{x',t} \varphi_{x',x} dx' - \delta (1 + v_{x,t}^{\beta}) i_{x,t} \right],$$

with $\lambda_{x,t}$ being the costate variable. The optimality condition is given by the following expression:

$$\frac{\partial \lambda_{x,t}}{\partial t} = \rho \lambda_{x,t} - d \frac{\partial^2 \lambda_{x,t}}{\partial x^2} + 1 - \alpha (1 - p_{x,t}^{\omega}) (n_{x,t} - 2 i_{x,t}) \int_{x_a}^{x_b} \lambda_{x',t} \varphi_{x',x} dx' + \lambda_{x,t} \delta (1 + v_{x,t}^{\beta}), \tag{18}$$

while for the control variables, the maximum principle reads as:

$$\max_{p_{x,t},v_{x,t}} \mathcal{H}(i_{x,t}, n_{x,t}, p_{x,t}, v_{x,t}), \tag{19}$$

which implies:

$$p_{x,t} = \left[-\alpha \omega \lambda_{x,t} \int_{x_a}^{x_b} (n_{x',t} - i_{x',t}) i_{x',t} \varphi_{x',x} dx' \right]^{\frac{1}{1-\omega}} \tag{20}$$

$$v_{x,t} = \left[\frac{1}{-\delta \beta \lambda_{x,t} i_{x,t}} \right]^{\frac{1}{\beta - 1}} \tag{21}$$

We need thus to solve the following system of two backward-forward partial differential equations where the control variables $p_{x,t}$ and $v_{x,t}$ are selected via the maximum principle, while the initial conditions on the state variable $i_{x,t}$, the final conditions on the costate variable $\lambda_{x,t}$ and the Neumann conditions on the first

derivatives are given below:

$$
\begin{aligned}
\frac{\partial i_{x,t}}{\partial t} &= d\frac{\partial^2 i_{x,t}}{\partial x^2} + \alpha(1-p^{\omega}_{x,t})\int_{x_a}^{x_b}(n_{x',t}-i_{x',t})i_{x',t}\varphi_{x',x}dx' - \delta(1+v^{\beta}_{x,t})i_{x,t} \\
\frac{\partial \lambda_{x,t}}{\partial t} &= \rho\lambda_{x,t} - d\frac{\partial^2 \lambda_{x,t}}{\partial x^2} + 1 - \alpha(1-p^{\omega}_{x,t})(n_{x,t}-2i_{x,t})\int_{x_a}^{x_b}\lambda_{x',t}\varphi_{x',x}dx' + \lambda_{x,t}\delta(1+v^{\beta}_{x,t}) \\
p_{x,t} &= \left[-\alpha\omega\lambda_{x,t}\int_{x_a}^{x_b}(n_{x',t}-i_{x',t})i_{x',t}\varphi_{x',x}dx'\right]^{\frac{1}{1-\omega}} \\
v_{x,t} &= \left[\frac{1}{-\delta\beta\lambda_{x,t}i_{x,t}}\right]^{\frac{1}{\beta-1}} \\
i_{0,x} &= i_0(x) \\
\lambda_{x,T} &= -\phi \\
\left.\frac{\partial i_{x,t}}{\partial x}\right|_{x=x_a} &= \left.\frac{\partial i_{x,t}}{\partial x}\right|_{x=x_a} = 0 \\
\left.\frac{\partial \lambda_{x,t}}{\partial x}\right|_{x=x_a} &= \left.\frac{\partial \lambda_{x,t}}{\partial x}\right|_{x=x_a} = 0
\end{aligned}
$$

As in the previous section, we proceed with numerical simulations to analyze the implications of our spatial framework, and we keep relying on the parameter values earlier employed in our seasonal flu calibration. Moreover, following the macroeconomic literature the rate of time preference is assumed to about 4% on a yearly basis, which on a daily basis implies $\rho = 0.04/365$, and the weight attached to diseases prevalence at the end of the planning horizon has been normalized to 1, that is $\phi = 1$ (La Torre et al., 2021a; 2021b). Different from the previous section in which we have focused on the geographical heterogeneity associated with variability in the effectiveness of health services, we now focus on the optimal provision of health services along with the effects of geographical heterogeneity related to differences in the health status. In order to do so we assume that the initial conditions of the share of infectives and population are respectively given by $i_{x,0} = \frac{3}{4}i_0 + \frac{1}{4}i_0 e^{-20x^2}$ where $i_0 = 0.05$ and $n_{x,0} = \frac{1}{2}$, with $x \in [-1, 1]$, representing a situation in which the disease prevalence is initially higher in the central than in the lateral regions.

The results of our numerical analysis (bases on an approach similar to La Torre et al.'s, 2015) are presented in Figure 4, where we show the spatio-temporal dynamics of prevention (left panel), treatment (middle panel) and the share of infectives (right panel), in the absence (top panels) and in the presence (bottom panels) of the static externality. From a qualitative point of view the dynamics is similar in the two scenarios, but from a quantitative point of view the results are substantially different: the evolution of prevention and treatment are non-monotonic, increasing initially and decreasing later (with prevention and treatment increasing again at the end of the planning horizon in the presence of the static externality), and while the intensity of the policy tools is large enough to allow for a monotonic decrease in the share of infectives in the absence of the static externality, this is not the case in the presence of the integral term where the share of infectives monotonically increases over time. The behavior of the optimal policies and disease prevalence at the end of the epidemic management program are strikingly different: in the absence of the static externality, that is in the presence of travel restriction regulations, it is optimal to completely eradicate the disease globally (both in the central than in the lateral regions) by optimally determining traditional health policies (i.e., prevention and treatment). By contrast, in the presence of the static externality, that is in situations in which travel restriction regulations are not applicable, it is optimal not to achieve complete eradication maintaining a positive share of infectives along with a positive preventive effort in the central region. This means that the optimal long run outcome significantly changes according to the policies the policymakers have at their disposal, and thus from a social planner's perspective it is convenient to achieve an endemic equilibrium in some region if only traditional health policies are available, while it is convenient to achieve a disease-free equilibrium everywhere in the spatial economy if also economic policies affecting

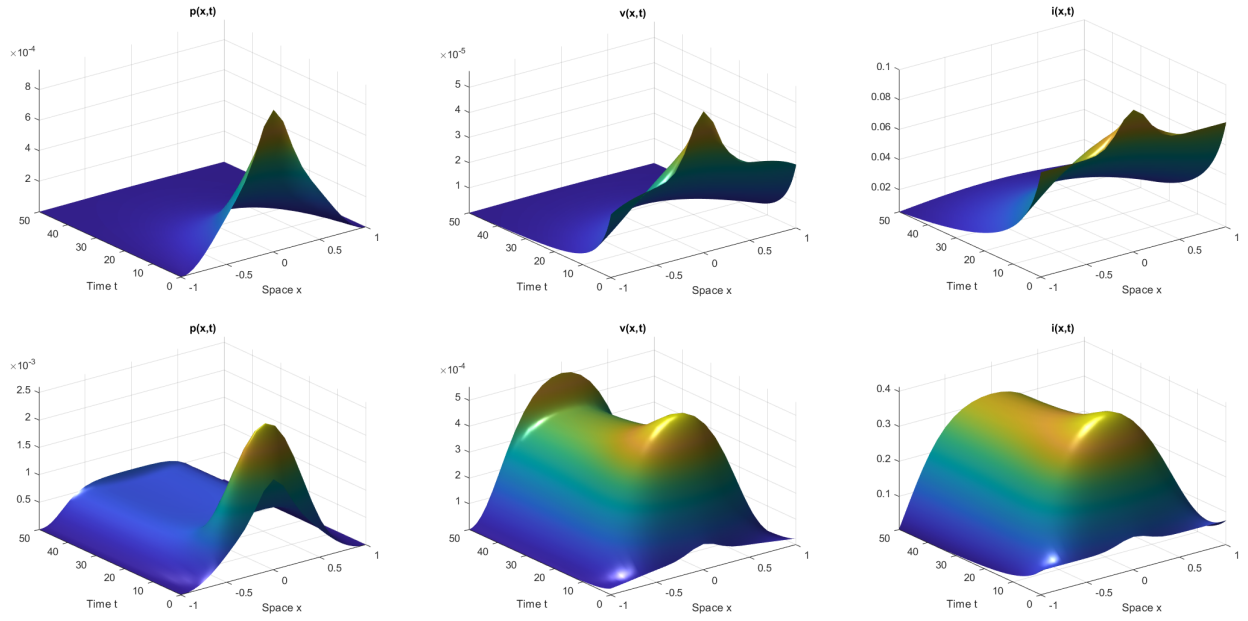

Figure 4: Evolution of prevention (left), treatment (center) and the share of infectives (right), in the absence (top) and in the presence (bottom) of the static externality.

individuals' travel possibilities can be employed. This type of result supports what discussed in the previous section about the importance of economic regulations limiting people's movements in order to achieve disease eradication at global level.

It may be useful to compare our results with La Torre et al.'s (2021a) who, by analyzing an epidemic management program over a finite time horizon in a setting without a spatial dimensions and so without geographical implications, show that it may be optimal to allow a disease to reach an endemic state. Our geography-extended analysis instead suggests that: (i) if there is no static externality then the process of disease diffusion may reverse their conclusions as in this case it would be optimal to achieve disease eradication everywhere in the spatial economy; (ii) if the static externality exists then the extra incidence caused by individual movements within the spatial economy may prevent a disease-free outcome to be attained, confirming their conclusions regarding the optimality of an endemic state. Therefore, exactly as we have already discussed in a purely dynamic context without optimal policymaking, we can conclude that neglecting the existence of cross-regional effects may lead to misleading conclusions about local and global epidemiological outcomes.

5 A COVID-19 Extended Model

The recent coronavirus epidemic has renewed the interest in understanding the implications of disease control measures on both macroeconomic and health outcomes. The most commonly health policy tools worldwide employed in order to reduce the spread of COVID-19 have taken the form of social distancing measures (i.e., maintaining physical distancing between people, avoiding gatherings, wearing face masks, imposing local or global lockdowns), which have, on the one hand, the effect to reduce the new infectious contacts (lowering disease incidence) and, on the other hand, to reduce productivity (lowering economic activity). For example, the requirement to maintain physical distance between others or to avoid gatherings may make the performing of normal daily tasks on the workplace particularly complicated negatively affecting economic production (La Torre et al., 2021a). Therefore, social distancing policy represents a specific form of preventive measure which, different from traditional ones such as prophylactic interventions, deteriorates

further the trade off between macroeconomic and health outcomes. Moreover, the ongoing promotion of massive vaccination campaigns everywhere in the world has raised growing concerns regarding the need to continue relying on social distancing to reduce disease incidence and prevalence. Therefore, in order to assess the effectiveness of the policy measures currently implemented in the fight of COVID-19, we need to extend our baseline model to account for social distancing and vaccination, along with the peculiarities of COVID-19. Indeed, our SIS framework suggests that upon recovery individuals do not acquire immunity and return to be susceptible, meaning that they can get infected again. Whether infection from COVID-19 grants any form of immunity is currently debated: despite as to date there is no evidence that *"people who have recovered from COVID-19 and have antibodies are protected from a second infection"* (WHO, 2020b), recent epidemiological studies show that reinfections are rare and estimate the protection against repeat infection to be about 80% and to last for five to six months (Hansen, 2021; Ledford, 2021). Therefore, in order to better describe the epidemiology of COVID-19 it seems convenient to rely on a SIRS framework in which a part of the individuals who have recovered from the disease acquires temporary immunity while the remaining part does not and thus it returns immediately to be susceptible. Since immunity is only temporary, it vanishes over time and after a certain period of time also vaccinated individuals returns to be susceptible again.

5.1 The Epidemiological Setting

Before analyzing the spatial implications of optimal policymaking in the context of COVID-19, it may be useful to introduce the epidemiological setup in a completely a-spatial framework to allow for a comparison with section 2. The population is composed by susceptibles, infectives and recovereds, R_t. Upon recovery, a share $0 < \mu < 1$ of the infectives acquires immunity and the remaining share $1 - \mu$ returns to be susceptible. Immunity is only temporary such that after some time also the recovereds get back being susceptibles, and $\epsilon > 0$ measures the speed of immunity loss. Apart from treatment and prenventive measures, where prevention takes the form of social distancing, also a vaccine is available which allows to grant temporary immunity, exactly as with natural recovery. Given $S_0 \geq 0$, $I_0 \geq 0$ and $R_0 \geq 0$, with $N = S_0 + I_0 + R_0$, the evolution of the number of susceptibles, infectives and recovereds is described by the following differential equations:

$$\dot{S}_t = \delta(1+v^\beta)(1-\mu)I_t - \alpha(1-p^\omega)\frac{S_t I_t}{N} + \epsilon R_t - u^\theta S_t \tag{22}$$

$$\dot{I}_t = \alpha(1-p^\omega)\frac{S_t I_t}{N} - \delta(1+v^\beta)I_t, \tag{23}$$

$$\dot{R}_t = \delta(1+v^\beta)\mu I_t - \epsilon R_t + u^\theta S_t, \tag{24}$$

where $0 < u < 1$ is the vaccination rate and $0 < \theta < 1$ measures the elasticity of vaccination, which is assumed to affect the dynamics of recovereds and susceptibles less than linearly because vaccination exhibits diminishing marginal product, exactly as prevention (social distancing) and treatment. Vaccination allows to make susceptible individuals immune, reducing the possible interactions between infectives and susceptibles, lowering thus disease incidence. Note that if $\mu = 0$ the model simplifies into a SIS setting extended for vaccination granting temporary immunity. If also $\epsilon = u = 0$ then it boils down to our baseline SIS.

By defining also the share of recovereds as $r_t = \frac{R_t}{N}$ and since $1 = s_t + i_t + r_t$, the epidemic dynamics can be characterized by analyzing the following planar system of differential equations:

$$\dot{s}_t = \delta(1+v^\beta)(1-\mu)i_t - \alpha(1-p^\omega)s_t i_t + \epsilon(1-s_t-i_t) - u^\theta s_t \tag{25}$$

$$\dot{i}_t = \alpha(1-p^\omega)s_t i_t - \delta(1+v^\beta)i_t \tag{26}$$

In this case the model's outcome depends on the magnitude of the following basic reproduction number:

$$\mathcal{R}_0 = \frac{\epsilon\alpha(1-p^\omega)}{\delta(1+v^\beta)(\epsilon+u^\theta)} \tag{27}$$

With respect to the basic reproduction number that we have introduced in our baseline SIS model (see equation (6)), also the parameters ϵ and u affect the size of $\mathcal{R}_0$. Intuitively, a higher vaccination rate reduces $\mathcal{R}_0$ while a higher immunization loss rate increases it. Exactly as in our baseline SIS model, whenever $\mathcal{R}_0 \leq 1$ the economy will converge to a disease-free equilibrium, which now is characterized by $\bar{i}_1 = 0$ and $\bar{s}_1 = \frac{\epsilon}{\epsilon+u^\theta}$ (and thus $\bar{r}_1 = 1 - \bar{s}_1 - \bar{i}_1$), while whenever $\mathcal{R}_0 > 1$, it will converge to an endemic equilibrium characterized by $\bar{i}_2 = \frac{\epsilon\alpha(1-p^\omega)-\delta(1+v^\beta)(\epsilon+u^\theta)}{\alpha(1-p^\omega)[\delta(1+v^\beta)\mu+\epsilon]}$ and $\bar{s}_2 = \frac{\delta(1+v^\beta)}{\alpha(1-p^\omega)}$ (and thus $\bar{r}_2 = 1-\bar{s}_2-\bar{i}_2$).[5] Apart from the different equilibrium value of the shares of susceptibles and infectives and the presence of a positive share of recoverereds, along with the role of vaccinations in reducing the basic reproduction number, from a qualitative point of view the results are identical to those earlier discussed in our baseline SIS model.

5.2 Optimal Spatial Policymaking

We now extend our purely a-spatial model to a spatial context in which the health policy, in terms of prevention (social distancing), treatment and vaccination, is optimally determined. By relying on the same spatial framework and following the same approach as in section 3, and exploiting the fact that $n_{x,t} = s_{x,t} + i_{x,t} + r_{x,t}$ where $r_{x,t}$ denotes the share of recovereds in location x, the spatio-temporal epidemic dynamics can be described by the following system of PDEs:

$$\frac{\partial n_{x,t}}{\partial t} = d\frac{\partial^2 n_{x,t}}{\partial x^2} \tag{28}$$

$$\frac{\partial i_{x,t}}{\partial t} = d\frac{\partial^2 i_{x,t}}{\partial x^2} + \alpha(1-p^\omega_{x,t})\int_{x_a}^{x_b} s_{x',t} i_{x',t} \varphi_{x',x} dx' - \delta(1+v^\beta_{x,t}) i_{x,t} \tag{29}$$

$$\frac{\partial s_{x,t}}{\partial t} = d\frac{\partial^2 s_{x,t}}{\partial x^2} + \delta(1+v^\beta_{x,t})(1-\mu) i_{x,t} - \alpha(1-p^\omega_{x,t})\int_{x_a}^{x_b} s_{x',t} i_{x',t} \varphi_{x',x} dx' + \epsilon(n_{x,t} - s_{x,t} - i_{x,t}) - u^\theta_{x,t} s_{x,t} \tag{30}$$

The economic setting is identical to what we have earlier discussed in section 4, but the peculiarity of social distancing as a form of prevention requires us to partly modify our baseline model in order to account for the fact the social distancing does not only affect the disease dynamics by diverting resources from consumption activities, but also reduces output by lowering the productivity of (healthy) labor. Specifically, similar to La Torre et al. (2021a), under social distancing net (of policy effects) output is given by $q_{x,t} = (1-\xi p_{x,t}) y_{x,t} = (1-\xi p_{x,t})(n_{x,t} - i_{x,t})$, where $\xi \geq 0$ measures the output lost due to the reliance upon preventive (i.e., social distancing) measures. If $\xi = 0$ the health policy tool does not affect output and thus we are back to our baseline setting characterizing the effects of prophylactic interventions. If $\xi > 0$ the effects on output are negative describing the implications of social distancing, which overall reduces the availability of resources to finance the epidemic management program. Consumption equals the difference between income (net of the effects of social distancing) and the taxes employed to finance public health policies in the form of social distancing, treatment and vaccination: $c_{x,t} = (1-\xi p_{x,t}) y_{x,t} - p_{x,t} - v_{x,t} - u_{x,t}$. Different from other forms of health policies, the effect of social distancing on consumption, is twofold: the need to finance preventive measures diverts resources away from consumption possibilities, and the detrimental effects induced on production reduces output and disposable income. It follows that our global

[5] It is straightforward to show that, if $\mathcal{R}_0 < 1$ only the equilibrium (s_1, i_1) exists and the Jacobian matrix associated with (25) - (26) evaluated at the disease-free equilibrium presents two negative eigenvalues such that (s_1, i_1) is a stable node; if $\mathcal{R}_0 > 1$ also equilibrium (s_2, i_2) exists and the analysis of the eigenvalues of the Jacobian matrix suggests that in this case (s_1, i_1) is unstable while (s_2, i_2) is a stable node.

social planner's optimization problem becomes:

$$
\begin{aligned}
\max_{p_{x,t},v_{x,t},u_{x,t}} \quad & W = \int_0^T \int_{x_a}^{x_b} \left[(1-\xi p_{x,t})(n_{x,t}-i_{x,t}) - p_{x,t} - v_{x,t} - u_{x,t}\right] e^{-\rho t} dx dt + \phi \int_{x_a}^{x_b} (n_{x,T}-i_{x,T}) e^{-\rho T} dx \\
s.t. \quad & \frac{\partial n_{x,t}}{\partial t} = d\frac{\partial^2 n_{x,t}}{\partial x^2} \\
& \frac{\partial i_{x,t}}{\partial t} = d\frac{\partial^2 i_{x,t}}{\partial x^2} + \alpha(1-p^{\omega}_{x,t}) \int_{x_a}^{x_b} s_{x',t} i_{x',t} \varphi_{x',x} dx' - \delta(1+v^{\beta}_{x,t}) i_{x,t} \qquad (31) \\
& \frac{\partial s_{x,t}}{\partial t} = d\frac{\partial^2 s_{x,t}}{\partial x^2} + \delta(1+v^{\beta}_{x,t})(1-\mu) i_{x,t} - \alpha(1-p^{\omega}_{x,t}) \int_{x_a}^{x_b} s_{x',t} i_{x',t} \varphi_{x',x} dx' + \\
& \qquad\qquad + \epsilon(n_{x,t} - s_{x,t} - i_{x,t}) - u^{\theta}_{x,t} s_{x,t} \\
& n_{x,0}, i_{x,0} > 0, s_{x,0} > 0 \text{ given}
\end{aligned}
$$

By following the same approach as in the previous section and denoting the costate variables with $\lambda_{x,t}$ and $\eta_{x,t}$, after some algebra the optimality conditions can be written as follows:

$$
\begin{aligned}
\frac{\partial n_{x,t}}{\partial t} &= d\frac{\partial^2 n_{x,t}}{\partial x^2} \\
\frac{\partial i_{x,t}}{\partial t} &= d\frac{\partial^2 i_{x,t}}{\partial x^2} + \alpha(1-p^{\omega}_{x,t}) \int_{x_a}^{x_b} s_{x',t} i_{x',t} \varphi_{x',x} dx' - \delta(1+v^{\beta}_{x,t}) i_{x,t} \\
\frac{\partial s_{x,t}}{\partial t} &= d\frac{\partial^2 s_{x,t}}{\partial x^2} + \delta(1+v^{\beta}_{x,t})(1-\mu) i_{x,t} - \alpha(1-p^{\omega}_{x,t}) \int_{x_a}^{x_b} s_{x',t} i_{x',t} \varphi_{x',x} dx' - u^{\theta}_{x,t} s_{x,t} + \epsilon(n_{x,t} - s_{x,t} - i_{x,t}) \\
\frac{\partial \lambda_{x,t}}{\partial t} &= \rho\lambda_{x,t} - d\frac{\partial^2 \lambda_{x,t}}{\partial x^2} + 1 - \xi p_{x,t} - \alpha(1-p^{\omega}_{x,t}) s_{x,t} \int_{x_a}^{x_b} \lambda_{x',t} \varphi_{x',x} dx' + \lambda_{x,t}\delta(1+v^{\beta}_{x,t}) \\
& \quad -\delta(1+v^{\beta}_{x,t})(1-\mu)\eta_{x,t} + \alpha(1-p^{\omega}_{x,t}) s_{x,t} \int_{x_a}^{x_b} \eta_{x',t} \varphi_{x',x} dx' + \epsilon\eta_{x,t} \\
\frac{\partial \eta_{x,t}}{\partial t} &= \rho\eta_{x,t} - d\frac{\partial^2 \eta_{x,t}}{\partial x^2} - \alpha(1-p^{\omega}_{x,t}) i_{x,t} \int_{x_a}^{x_b} \lambda_{x',t} \varphi_{x',x} dx' + \alpha(1-p^{\omega}_{x,t}) i_{x,t} \int_{x_a}^{x_b} \eta_{x',t} \varphi_{x',x} dx' + \\
& \quad +\eta_{x,t} u^{\theta}_{x,t} + \epsilon\eta_{x,t} \\
p_{x,t} &= \left[\frac{\xi(n_{x,t} - i_{x,t}) + 1}{(\eta_{x,t} - \lambda_{x,t})\alpha\omega \int_{x_a}^{x_b} s_{x',t} i_{x',t} \varphi_{x',x} dx'}\right]^{\frac{1}{\omega-1}} \\
v_{x,t} &= \left[\frac{1}{-\lambda_{x,t}\delta\beta i_{x,t} + (1-\mu)\eta_{x,t}\delta\beta i_{x,t}}\right]^{\frac{1}{\beta-1}} \\
u_{x,t} &= \left[-\frac{1}{\eta_{x,t}\theta s_{x,t}}\right]^{\frac{1}{\theta-1}} \\
\lambda_{x,T} &= -\phi \\
\eta_{x,T} &= 0
\end{aligned}
$$

As in the previous section, we proceed with numerical simulations to analyze the implications of our spatial framework on the optimal determination of the health policy measures to face the COVID-19 epidemic. We calibrate our model on the Italian experience during the first epidemic wave during which the basic reproduction number (in the absence of vaccination) has been estimated to be equal to 2.29 ($\mathcal{R}_0 = 2.79$), while the time of recovery has been estimated to be about three weeks (Remuzzi and Remuzzi, 2020), and from this information we can compute the recovery rate and the infectivity rate as $\delta = 0.0476$ and $\alpha = 0.1328$, respectively (La Torre et al., 2021a). Recent studies suggest that upon recovery about 80% of the infectives acquire transitory immunity along with the fact that the duration of immunity granted by recovery

or vaccination is about six months, which imply that $\mu = 0.8$ and $\epsilon = 0.0056$ (Hansen, 2021; Ledford, 2021). Some studies measure the impact of social distancing on workers' productivity estimating the size of the productivity loss to be about 5% in the short run, which suggests that $\xi = 0.05$ (Bloom et al., 2020). As in the previous section, following the macroeconomic literature, the rate of time preference is assumed to be about 4% on a yearly basis, the weight attached to diseases prevalence at the end of the planning horizon is normalized to unity and the diffusion parameter is assumed to be 1%, that is we set $\rho = 0.04/365$, $\phi = 1$ and $d = 0.01$ (La Torre et al., 2021a). As also in the case of COVID-19 there are no estimates of the of elasticities of the policy measures implemented, the parameters ω, β and θ are arbitrarily set and in particular $\omega = \beta = \theta = 0.5$. However, it is possible to show that changing these parameter values does not affect our qualitative conclusions but impact quantitatively the intensity of the optimal policy measures and thus epidemic dynamics. In particular, the higher the elasticities the higher the effectiveness of the epidemic control policies and the lower the disease prevalence at any moment in time.

The results of our numerical analysis are reported in Figures 5 and 6 where we show the spatio-temporal dynamics of the control (top panels) and state (bottom panels) variables. In the top panels we represent social distancing (left panel), treatment (middle panel) and vaccination (right panel), while in the bottom panels we represent the share of infectives (left panel) and the share of susceptibles (right panel). Figure 5

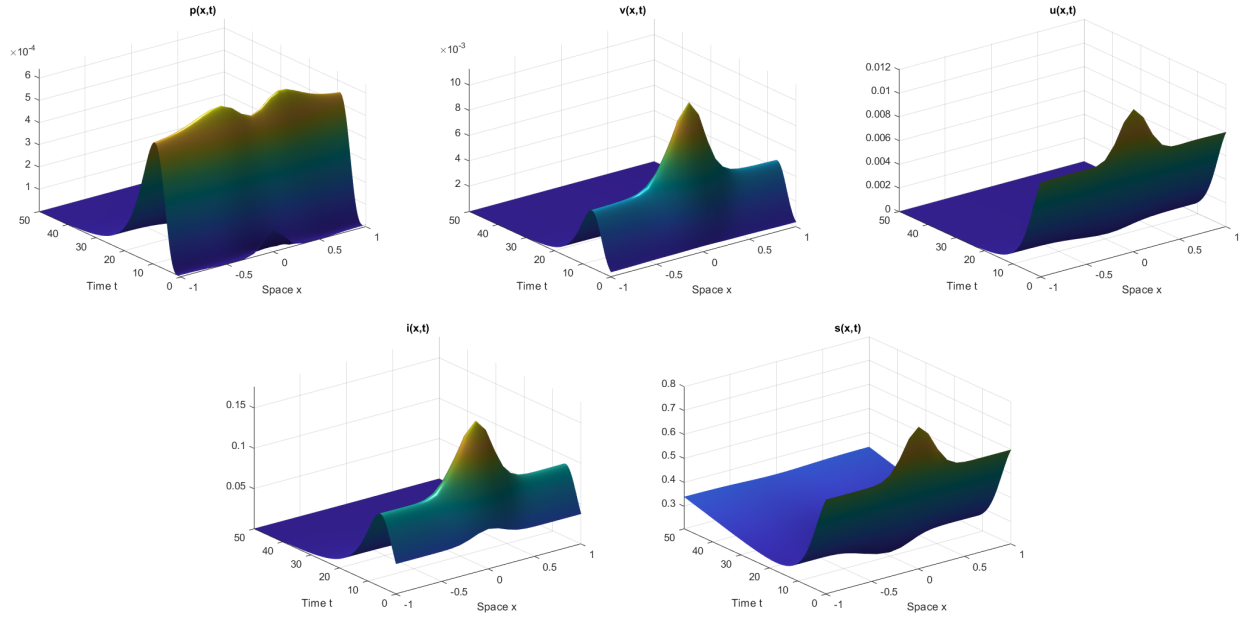

Figure 5: Evolution of the control variables (top) – prevention (left), treatment (center) and vaccination (right) – and state variables (bottom) – share of infectives (left) and share of susceptibles (right) – in the absence of the static externality.

visualizes our results in the absence of the static externality, while Figure 6 those in the presence of such a static externality. From a qualitative point of view the dynamics is similar in the two scenarios and comparable with that shown in Figure 4. With respect to our previous seasonal flu calibration, the roles of social distancing and vaccination deserve some explanation. By affecting the epidemiological dynamics exactly as prevention, the evolution of social distancing closely resembles what seen in Figure 4. Vaccination instead is initially higher in the central region in which prevalence is higher at the beginning of the epidemic management program, and then it monotonically decreases to zero towards the end of the program. Despite social distancing entails an important reduction in output which makes the eradication goal (both at local and global levels) substantially costly, in the absence of the static externality, that is in the presence of travel restriction regulations, vaccination allows to prevent new infections to occur and thus to achieve eradication

everywhere in the spatial economy by quickly reducing the size of the susceptible population. However, in

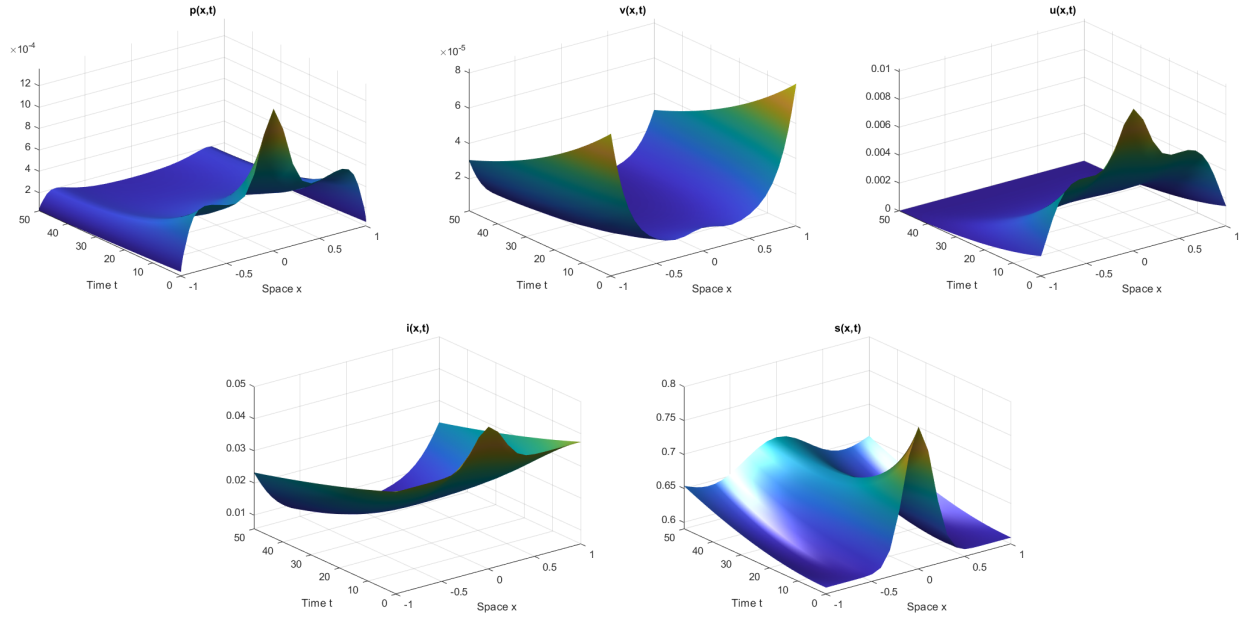

Figure 6: Evolution of the control variables (top) – prevention (left), treatment (center) and vaccination (right) – and state variables (bottom) – share of infectives (left) and share of susceptibles (right) – in the presence of the static externality.

the presence of the static externality, that is in the absence of travel restriction regulations, vaccination is not enough to counteract the new infections caused by the movements due to commuting and business trips from and to the lateral regions and thus eradication is not possible anywhere in the spatial economy. Consistent with what earlier discussed for the seasonal flu case, our analysis suggests that without the possibility to impose stringent travel restrictions it might be optimal to allow COVID-19 to become endemic in the Italian population. La Torre et al. (2021a) have achieved a similar conclusion in a framework without spatial effects and without considering immunity due to vaccination, and our results show that the existence of geographical externalities (in particular the movements due to the commuting needs) will not change this type of conclusion. In order not to excessively damage macroeconomic outcomes it might be convenient to tolerate the disease to persist indefinitely in our society.

Note that the results of our model applied to COVID-19 need to be taken with some grain of salt as some of its underlying assumptions may not entirely reflect the true consequences of the disease. In particular, the absence of vital dynamics precludes us from accounting for the effects associated with disease-induced mortality, which in reality represents an essential aspect of the recent coronavirus epidemic experience, as deaths counts exceed one million and half worldwide (Dong et al., 2020). Therefore, we may expect that in our analysis the optimal level of policy intervention results to be more lenient than in other frameworks in which mortality effects are effectively taken into account, and thus our conclusions can only be considered as a stylized benchmark for real world policymaking.

6 Conclusions

This paper analyzes the implications of geographical heterogeneities and externalities on health outcomes by focusing on the dynamics of infectious diseases. Understanding the evolution of communicable diseases and how policies can be used to control them is an important actual problem, as suggested by the fact that the UN in its sustainable development goals for 2030 have specifically included the eradication of

HIV, tuberculosis, malaria and other communicable diseases. In order to understand the role of growing economic integration and human movements (i.e., migration and commuting) in this context, we develop a spatial epidemiological model to account for differences in the effectiveness of health services and in the initial spread of the disease across regions, along with the implications of geographical externalities. We distinguish between static and dynamic externalities, where the former type represents the effects of migration while the latter those of commuting and business trips. We show that the presence of only the dynamic externality leads in the long run to a geographically homogeneous health outcome, while the static externality allows some geographical heterogeneity to persist also in the long run. Moreover, we show that neglecting the existence of geographical externalities may lead to misleading conclusions about the long run health outcome not only in single regions but also in the entire economy, suggesting thus that in order to achieve disease eradication single regions need to carefully account for the policies implemented in other regions and eventually promote cross-regional coordination. We analyze such a coordination by analyzing a macroeconomic-epidemiological control problem in which the social planner determines at global level the health policy measures by considering the availability of resources determined at macroeconomic level, showing that in order to achieve long run eradication globally it is essential to accompany traditional health policies (i.e., prevention and treatment) with economic regulations limiting people's movements (i.e., imposing travel bans). Focusing on the specific case of COVID-19 we develop an extension of our baseline model to account for the peculiarities of the disease and of the policy measures employed to fight it (social distancing and vaccination), and present a calibration of such an extended model based on Italian data, showing that, because of the infections generated by cross-regional commuting, even vaccination may not be enough to achieve disease eradication, and limitations on people's movements need to accompany vaccination in order to preclude COVID-19 from reaching an endemic state.

To the best of our knowledge, no other study has tried to analyze the geographical implications of infectious diseases in a way comparable to ours. Therefore, our modeling framework has been quite simplistic in order to show in the most intuitive way the importance of taking into account geographical characteristics to effectively achieve desired health goals. As a result, some of the assumptions underlying our basic spatial SIS model would need to be relaxed in order to improve its ability to effectively describe real world experiences. Specifically, the movements of individuals from one region to the next is assumed to be completely exogenous but with the outbreak of new diseases people may try to escape the disease moving towards healthier regions; endogeneizing the speed of spatial diffusion to account for this potential effect may provide us with a more rigorous characterization of the geographical implications of epidemics (Ramalingaswami, 2001). Also, the time horizon has been assumed to be finite such that saving and capital accumulation do not play any role, however the outbreak of certain diseases may require several decades to be brought under control; introducing capital accumulation may be important to characterize the medium-long run consequences of epidemics and their geographical effects (Goenka et al., 2014). These further issues are left for future research.

Compliance with Ethical Standards

Conflict of interest: The authors declare that they have no conflict of interest.

Data sharing and accessibility: Data sharing is not applicable to this article as no new data were created or analyzed in this study.

References

1. Acemoglu, D., Chernozhukov, V., Werning, I., Whinston, M.D. (2021). "Optimal targeted lockdowns in a multigroup SIR model", American Economic Review: Insights 3, 487–502

2. Acemoglu, D., Johnson, S. (2007). "Disease and development: the effect of life expectancy on economic growth", Journal of Political Economy 115, 925–985
3. Adda, J. (2016). "Economic activity and the spread of viral diseases: evidence from high frequency data", Quarterly Journal of Economics 131, 891—941
4. Alvarez, F.E., Argente, D., Lippi, F. (2021). "A simple planning problem for COVID-19 lockdown", American Economic Review: Insights 3, 367–382
5. Anderson, S.T., Laxminarayan, R, Salant, S.W. (2010). "Diversify or focus? Spending to combat infectious diseases when budgets are tight", Journal of Health Economics 31, 658—675
6. Anita, S., Capasso, V. (2017). "Reaction-diffusion systems in epidemiology", arXiv:1703.02760
7. Aspri, A., Beretta, E., Gandolfi, A., Wasmer, E. (2021). "Mortality containment vs. economics opening: optimal policies in a SEIARD model", Journal of Mathematical Economics 93, 10249
8. Barrett, S. (2003). "Global disease eradication", Journal of the European Economic Association 1, 591–600
9. Barro, R.J., Sala-i-Martin, X. (2004). "Economic growth" (Cambridge, Massachusetts: MIT Press)
10. Bloom, N., Bunn, P., Mizen, P., Smietanka, P., Thwaites, G. (2020). "The Impact of Covid-19 on productivity", NBER Working Paper 28233
11. Brodeur, A., Gray, D., Islam, A., Bhuiyan, S. (2021). "A literature review of the economics of COVID-19", Journal of Economic Survey 35, 1007–1044
12. Boucekkine, R., Camacho, C., Zou, B. (2009). "Bridging the gap between growth theory and economic geography: the spatial Ramsey model", Macroeconomic Dynamics 13, 20–45
13. Boucekkine, R., Carvajal, A., Chakraborty, S., Goenka, A. (2021). "The economics of epidemics and contagious diseases: an introduction", Journal of Mathematical Economics 93, 102498
14. Brock, W.A., Xepapadeas, A. (2010). "Pattern formations, spatial externalities and regulation in a coupled economic–ecological system", Journal of Environmental Economics and Management 59, 149–164
15. Bucci, A., La Torre, D., Liuzzi, D., Marsiglio, S. (2019). "Financial contagion and economic development: an epidemiological approach", Journal of Economic Behavior & Organization 162, 211–228
16. Camacho, C., Pérez–Barahona, A. (2015). "Land use dynamics and the environment", Journal of Economic Dynamics & Control 52, 96–118
17. Caulkins, J.P., Grass, D., Feichtinger, G., Hartl, R.F., Kort, P.M., Prskawetz, A., Seidl, A., Wrzaczek, S. (2021). "The optimal lockdown intensity for COVID-19", Journal of Mathematical Economics 93, 102489
18. Cervellati, M., Sunde, U., Valmori, S. (2017). "Pathogens, weather shocks and civil conflict", Economic Journal 127, 2581–2616
19. Chakraborty, S., Papageorgiou, C., Pérez Sebastián, F. (2010). "Diseases, infection dynamics and development", Journal of Monetary Economics 57, 859–872
20. Cheng, C., Barceló, J., Hartnett, A.S., Kubinec, R., Messerschmidt, L. (2020). "COVID-19 government response event dataset (CoronaNet v.1.0)", Nature Human Behavior 4, 756—768
21. Dong, E., Du, H., Gardner, L. (2020). "An interactive web-based dashboard to track COVID-19 in real time", Lancet Infectious Diseases 20, 533–534
22. Ducasse, R. (2020). "Qualitative properties of spatial epidemiological models", arXiv:2005.06781
23. Eichenbaum, M., Rebelo, S., Trabandt, M. (2021). "The macroeconomics of epidemics", Review of Financial Studies 34, 5149-–5187
24. Feng, Z., Huang, W., Castillo-Chavez, C. (2005). "Global behavior of a multi-group SIS epidemic model with age structure", Journal of Differential Equations 218, 292–324

25. Gersovitz, M., Hammer, J.S. (2004). "The economical control of infectious diseases", Economic Journal 114, 1–27
26. Goenka, A., Liu, L. (2012). "Infectious diseases and endogenous fluctuations", Economic Theory 50, 125–149
27. Goenka, A., Liu, L., Nguyen, M.H. (2014). "Infectious diseases and economic growth", Journal of Mathematical Economics 50, 34–53
28. Goldman, S.M., Lightwood, J. (2002). "Cost optimization in the SIS model of infectious disease with treatment", Topics in Economic Analysis and Policy 2, Article 4
29. Gollier, C. (2020). "Cost–benefit analysis of age-specific deconfinement strategies", Journal of Public Economic Theory 22, 1746–1771
30. Gori, L., Mammana, C., Manfredi, P., Michetti, E. (2021a). "Economic development with deadly-communicable diseases and public prevention", Journal of Public Economic Theory, forthcoming
31. Gori, L., Manfredi, P., Marsiglio, S., Sodini, M. (2021b). "COVID-19 epidemic and mitigation policies: positive and normative analyses in a neoclassical growth model", Journal of Public Economic Theory, forthcoming
32. Guan, Y., Zheng, B.J., He, Y.Q., Liu, X.L., Zhuang,Z.X., Cheung, C.L., Luo, S.W., Li, P.H., Zhang, L.J., Guan, Y.J., Butt, K.M., Wong, K.L., Chan, K.W., Lim, W., Shortridge, K.F., Yuen, K.Y., Peiris, J.S.M., Poon, L.L.M. (2003). "Isolation and characterization of viruses related to the SARS coronavirus from animals in southern China", Science 302, 276—278
33. Hansen, C.H., Michlmayr, D., Gubbels, S.M., Molbak, K. Ethelberg, S. (2021). "Assessment of protection against reinfection with SARS-CoV-2 among 4 million PCR-tested individuals in Denmark in 2020: a population-level observational study", The Lancet 397, P1204–1212
34. Hethcote, H.W. (2000). "The mathematics of infectious diseases", SIAM Review, 42, 599–653
35. Hotelling, H.(1929). "Stability in competition", Economic Journal 39, 41–57
36. Hritonenko, N., Yatsenko, O., Yatsenko, Y., (2021). "Model with transmission delays for COVID-19 control: theory and empirical assessment", Journal of Public Economic Theory, forthcoming
37. Kermack, W.O., McKendrick, A.G. (1927). "A contribution to the mathematical theory of epidemics", Proceedings of the Royal Society of London Series A 115, 700—721
38. Kimball, A.M. (2006). "Risky trade: infectious disease in the era of global trade" (Ashgate Publishing: Farnham)
39. Klasing, M.J., Milionis, P. (2020). "The international epidemiological transition and the education gender gap", Journal of Economic Growth 25, 37—86
40. Klein, E., Laxminarayan, R., Smith, D.L., Gilligan, C.A. (2007). "Economic incentives and mathematical models of disease", Environment and Development Economics 12, 707–732
41. La Torre, D., Liuzzi, D., Marsiglio, S. (2015). "Pollution diffusion and abatement activities across space and over time", Mathematical Social Sciences 78, 48–63
42. La Torre, D., Liuzzi, D., Marsiglio, S. (2019). "Population and geography do matter for sustainable development", Environment and Development Economics 24, 201-–223
43. La Torre, D., Liuzzi, D., Marsiglio, S. (2021a). "Epidemics and macroeconomic outcomes: social distancing intensity and duration", *Journal of Mathematical Economics* 93, 102473
44. La Torre, D., Liuzzi, D., Marsiglio, S. (2021b). "Transboundary pollution externalities: think globally, act locally?", *Journal of Mathematical Economics*, forthcoming
45. La Torre, D., Malik, T., Marsiglio, S. (2020). "Optimal control of prevention and treatment in a basic macroeconomic-epidemiological model", Mathematical Social Sciences 108, 100–108
46. Ledford, H. (2021). "COVID reinfections are unusual – but could still help the virus to spread", Nature, January 2021

47. Lotfi, E.M., Maziane, M., Hattaf, K., Yousfi, N. (2014). "Partial differential equations of an epidemic model with spatial diffusion", International Journal of Partial Differential Equations, article 186437
48. Martcheva, M. (2009). "A non-autonomous multi-strain SIS epidemic model", Journal of Biological Dynamics, 3, 235–251
49. Martcheva, M. (2015). "An introduction to mathematical epidemiology" (New York: Springer)
50. Philipson, T. (2000). "Economic epidemiology and infectious disease", in (Cuyler, A.J., Newhouse, J.P., eds.) "Handbook of Health Economics", vol. 1B, 1761–1799 (Amsterdam: North Holland)
51. Polyanin, A.D. (2002). "Handbook of linear partial differential equations for engineers and scientists" (New York: Chapman and Hall/CRC)
52. Ramalingaswami, V. (2001). "Psychosocial effects of the 1994 plague outbreak in Surat, India", Military Medicine 166, 29-–30
53. Remuzzi, A., Remuzzi, G. (2020). "COVID-19 and Italy: what next?", Lancet 395, 1225—1228
54. Rowthorn, R., Toxvard, F. (2012). "The optimal control of infectious diseases via prevention and treatment", CEPR Discussion Papers 8925
55. Rothert, J. (2021). "Optimal federal transfers during uncoordinated response to a pandemic", Journal of Public Economic Theory, forthcoming
56. Sattenspiel, L., Dietz, K. (1995). "A structured epidemic model incorporating geographic mobility among regions", Mathematical Biosciences 128, 71–91
57. Skinner, J. (2012). "Causes and consequences of regional variations in health care", in (Pauly, M.V., Mcguire, T.G., Barros, P.P., eds.) "Handbook of Health Economics", vol. 2, 45–93 (Amsterdam: North Holland)
58. Tsori, Y., Granek, R. (2021). "Epidemiological model for the in-homogeneous spatial spreading of COVID-19 and other diseases", PLoS ONE 16, e0246056
59. United Nations (2015). "Resolution adopted by the General Assembly on 25 September 2015", available at: http://www.un.org/ga/search/view_doc.asp?symbol=A/RES/70/1&Lang=E
60. Weil, D. (2014). "Health and economic growth", in (Aghion, P., Durlauf, S.N., eds.) "Handbook of Economic Growth", vol. 2B, 623–682 (Amsterdam: North Holland)
61. Wang, T. (2014). "Dynamics of an epidemic model with spatial diffusion", Physica A 409, 119-–129
62. World Bank (1993). "Investing in health", World Bank Development Report (Oxford University Press: London)
63. World Health Organization (2009)."World Health Statistics 2009", available at: http://www.who.int/gho/publications/world_health_statistics/EN_WHS09_Full.pdf
64. World Health Organization (2018). "Influenza (seasonal)", available at: https://www.who.int/news-room/fact-sheets/detail/influenza-(seasonal)
65. World Health Organization (2020a). "Coronavirus disease (COVID-19) outbreak", available at: https://www.who.int/emergencies/diseases/novel-coronavirus-2019
66. World Health Organization (2020b). "Immunity passports" in the context of COVID-19 - Scientific brief, available at: https://www.who.int/news-room/commentaries/detail/immunity-passports-in-the-context-of-covid-19
67. Wu, J.T., Leung, K., Leung, G.M.. (2020). "Nowcasting and forecasting the potential domestic and international spread of the 2019-nCoV outbreak originating in Wuhan, China: a modelling study", The Lancet 395, 689—697