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Epidemics and macroeconomic outcomes: Social distancing intensity and duration

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ABSTRACT

We analyze the determination of the optimal intensity and duration of social distancing policy aiming to control the spread of an infectious disease in a simple macroeconomic–epidemiological model. In our setting the social planner wishes to minimize the social costs associated with the levels of disease prevalence and output lost due to social distancing, both during and at the end of epidemic management program. Indeed, by limiting individuals’ ability to freely move or interact with others (since requiring to wear face mask or to maintain physical distance from others, or even forcing some businesses to remain closed), social distancing has on the one hand the effect to reduce the disease incidence and on the other hand to reduce the economy’s productive capacity. We analyze both the early and the advanced epidemic stage intervention strategies highlighting their implications for short and long run health and macroeconomic outcomes. We show that both the intensity and the duration of the optimal social distancing policy may largely vary according to the epidemiological characteristics of specific diseases, and that the balancing of the health benefits and economic costs associated with social distancing may require to accept the disease to reach an endemic state. Focusing in particular on COVID–19 we present a calibration based on Italian data showing how the optimal social distancing policy may vary if implemented at national or at regional level.

1. Introduction

Communicable diseases have historically played (and still do today) a major role in shaping economic development in both industrialized and developing countries, by determining till today 30% of deaths and accounting for 45% of the related years of life lost worldwide (Lopez et al., 2006; WHO, 2009). A variety of channels through which this may occur, including education attainment, labor force participation, life expectancy, income and civil conflict, have been identified and extensively documented in literature (Acemoglu and Johnson, 2007; Bleakley, 2007; Adda, 2016; Cervellati et al., 2017; Klasing and Milionis, 2020). In the simplest and most intuitive way, communicable diseases are a major source of morbidity and mortality everywhere in the world, and thus they affect labor market outcomes along with saving and investment incentives yielding detrimental consequences on macroeconomic performance (Bouckx et al., 2009; Chakraborty et al., 2010). As health services are to a large extent publicly provided in most countries, the severity of such effects is amplified by the need to finance public health policies diverting resources from productive activities (Velenyi and Smitz, 2014; La Torre et al., 2020). The relevance of these findings and arguments has over the last two decades given birth to a growing economic epidemiology literature to accompany the mathematical epidemiology literature in analyzing the implications and dynamics of communicable diseases. While mathematical epidemiology aims to characterize the epidemic dynamics according to the biological features of specific diseases (Hethcote, 2000, 2008), economic epidemiology tries to understand the mechanisms through which health policy, in the form of preventive and treatment measures, may be used to contrast the spread of communicable diseases (Philipson, 2000; Gersovitz and Hammer, 2003). Most of the economic epidemiology papers rely on a microeconomic perspective focusing on a partial equilibrium analysis to examine the behavioral response of individual rational agents to the disease prevalence and to the health policy measures (Anderson et al., 2010; Gersovitz and Hammer, 2004; Goldman and Lightwood, 2002), and only few works have tried to
assess their macroeconomic implications (Goenka and Liu, 2012, 2020; Goenka et al., 2014; La Torre et al., 2020). Goenka and Liu (2012, 2020) and Goenka et al. (2014) discuss the long run effects of epidemics on economic growth, both in neoclassical and endogenous growth setups, showing that the health-income relationship may be a source of nontrivial macroeconomic dynamics, leading eventually to chaos. La Torre et al. (2020) focus on the short run effects of epidemics on income levels analyzing the feedback effects between the disease spread and the availability of resources to fight it, assessing whether preventive or treatment policy measures may be most effective. All these works, both those adopting a microeconomic and a macroeconomic perspective, share the same analytical framework to characterize the epidemic dynamics by relying on the susceptible–infected–susceptible (SIS) model, which is one of the simplest and most general mathematical epidemiology setups. In this paper we contribute to this literature adopting the same SIS framework to analyze the relation between epidemics and macroeconomic outcomes in light of the recent coronavirus pandemic experience. Indeed, since upon recovery from COVID-19 individuals return susceptible to a new infection (WHO, 2020b), the SIS model can be applied also to characterize the dynamics of the coronavirus epidemic.

The recent COVID-19 epidemic has spurred a large and shared interest in understanding the mutual relations between communicable diseases, macroeconomic outcomes and health-economic policy. COVID-19 is a highly contagious virus-induced communicable disease, transmitted via droplets and contaminated objects during close unprotected contact between an infecter and infectee (WHO, 2020a). The virus is currently spreading fast from human-to-human as transmission simply occurs when healthy individuals meet respiratory droplets from coughs or sneezes of an infected person. Transmission is also possible via contaminated objects or materials which act as a carrier of the virus, such as clothes, utensils, and furniture (Rothan and Byrareddy, 2020). The outbreak of the disease has origin in China in late 2019 and has reached a pandemic status in only a few months, resulting thus far (at the time of writing, in early July 2020) in over 9.3 million confirmed cases and over 480,000 deaths globally (Dong et al., 2020). The implemented policy responses to reduce the spread of the disease have been countless (i.e., more than 13,000 in more than 195 countries), including traditional preventive and treatment measures but also social distancing (Cheng et al., 2020). Social distancing refers to all those arrangements which aim at reducing the rate of transmission of a disease by limiting the exposure of single individuals to possible sources of infection. Social distancing is thus one of the most effective non-pharmaceutical disease control actions that can reduce the spread of a highly contagious disease (Maharaj and Kcleckowski, 2012). Different forms of social distancing have been introduced and promoted in most countries, ranging from quarantine to voluntary isolations, from restrictions on individual mobility to lockdowns, from the requirement to wear face masks to that of maintaining a certain physical distance from others. By imposing stringent restrictions on individual behavior that have forced firms to decrease the utilization of the workforce and by requiring individuals to limit their interactions with one another on the workplace, they have also generated devastating economic effects including a large number of job losses and a large drop in GDP. The effects have been particularly severe in those sectors in which social interactions are essential (such as tourism and education) or intensive-contacts are required (such as hospitality and manufacturing), and it has also affected the industries linked to them. According to their dependence on these sectors, some economies have been more exposed than others, and the forecasts for the drop in level of economic activities in 2020 in European countries range from about 10% (Greece, Italy, and Spain) to about 5% (Poland and Luxemburg) on a yearly basis (European Commission, 2020). Understanding thus the health and economic consequences of specific disease control policies, and in particular those of social distancing, is essential in order to minimize their social cost and support policymakers in the difficult task of effectively managing the pandemic.

Over the last few months several works have tried to analyze the macroeconomic implications of COVID-19 and the policy measures implemented to control its spread (Acemoglu et al., 2020; Alvarez et al., 2020; Atkeson, 2020; Eichenbaum et al., 2020; Ng, 2020). Atkeson (2020) analyzes a purely dynamic epidemiological setup in which the disease is described by a susceptible–infected–recovered (SIR) model, which is based on the assumption that recovery from the disease confers permanent immunity; through a comparative dynamic exercise he shows how different mitigation efforts affect the epidemic dynamics by reducing the disease transmission rate. Building on Atkeson’s epidemiological setup (2020), Alvarez et al. (2020) analyze the links between economy and epidemics in a setting where the lockdown policy determines how much output agents can produce; by focusing on a social planner’s framework they determine the optimal duration and intensity of the lockdown showing in their baseline scenario that it may need to be initially severe and implemented shortly after the outbreak. Acemoglu et al. (2020) rely on a similar centralized economic setup enriching the epidemiological framework to consider a multi-group SIR model to distinguish the disease impact among young, middle-aged and old individuals, showing that intuitively increasing the severity and the duration of the lockdown for the old may allow to relax it for younger agents. Eichenbaum et al. (2020) analyze the competitive equilibrium in a decentralized economy focusing on individuals’ behavioral response to the disease, showing that their decision to reduce consumption and work lowers the health costs of the epidemic but increases the economic ones, giving rise to an important trade off between health and macroeconomic outcomes. Ng (2020) focuses on a multi-group SIR framework to distinguish between different severity levels of the disease in a competitive framework, analyzing the effects of different intensity of lockdown policies through comparative dynamics. Bosi et al. (2020) analyze a centralized setup in which the social planner seeks to maximize social welfare, which is defined according to different criteria; different from other papers, they describe the epidemic setting as in a SIS model and characterize the optimal policy analytically, showing that the intensity of the lockdown crucially depends on individuals’ degree of altruism. Our paper contributes to this literature by taking a slightly different point of view and integrating some of the approaches presented thus far.

Despite most of the works are based on a SIR setup, since 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), it seems more reasonable to rely on a SIS framework in which after recovery people return susceptible and thus can get infected again, as in Bosi et al. (2020). This has also the advantage to allow for a direct comparison of our paper with the macroeconomic epidemiology literature (Goenka and Liu, 2012, 2020; Goenka et al., 2014; La Torre et al., 2020). Different from Bosi et al. (2020), since the epidemic management is a short run problem we abstract from saving and capital accumulation, considering a simple framework in which the disease prevalence determines the income level and disposable income is entirely consumed, as in Eichenbaum et al. (2020). Different from both Bosi et al. (2020) and Eichenbaum et al. (2020) in which the time horizon in infinite, we consider a finite time horizon to investigate the intensity and the duration of the optimal policy.
along the lines of Acemoglu et al. (2020) and Alvarez et al. (2020). Similar to them we focus on a centralized problem since in the real world the response to the disease has been mainly driven by public regulations and governmental measures rather than by behavioral changes. Different from them in our setup the social planner does not choose only the degree of social distancing but also that of therapeutic treatment: treatment is publicly provided and is financed through income taxation, thus the spread of the disease by lowering income reduces also the availability of resources to fund treatment measures, as in La Torre et al. (2020). In our setting the social planner aims to minimize the social costs associated with the disease prevalence and the strength of the policy tools employed to manage the epidemic, similar to Alvarez et al. (2020). However, different from them, rather than relying only on numerical simulations we aim to determine analytically the duration and the intensity of the optimal policies as in Bosi et al. (2020). Different from all extant works in which the epidemic is implicitly assumed to be in an advanced stage, we analyze both the early epidemic and non-early epidemic intervention strategies highlighting their implications for short and long run health and macroeconomic outcomes. Indeed, several papers in mathematical epidemiology show that in early stages of an epidemic outbreak the evolution of the disease prevalence can be described through sub-exponential or exponential growth dynamics giving rise to a setup substantially different from those considered in traditional mathematical epidemiology models (Chowell et al., 2016).

Specifically, in our framework the social planner determines the optimal intensity and duration of social distancing by balancing its beneficial effects on health outcomes and its detrimental macroeconomic consequences. Indeed, by limiting individuals’ ability to freely interact with others social distancing allows to reduce the disease incidence but it also leads to a reduction in the economy’s productive capacity. Therefore, the planner wishes to minimize the social costs associated with the levels of disease prevalence and output lost due to the social distancing policy. Our setup is not strictly tailored to investigate the consequences of COVID-19 but it is applicable in more general terms, allowing us to consider the macroeconomic implications of a variety of communicable diseases. We show that in the early stages of an epidemic outbreak the disease dynamics can be proxied by a linear differential equation and thus it is possible to explicitly determine both the optimal intensity and duration of social distancing. In later stages such an approximation is not applicable and the nonlinearity of the epidemiological dynamics precludes us from the possibility to derive analytical solutions. Through numerical simulations we illustrate how the optimal social distancing may largely vary according to the epidemiological features of specific diseases. For example, in the case of the seasonal flu the social distancing policy needs to be stricter and to last for longer than in the case of the common cold, because of its higher infectivity rate. We also show that our model is applicable to COVID-19 (even if by abstracting from the disease-induced mortality our results need to be taken with some grain of salt) by presenting a calibration based on Italian data, distinguishing between national- and regional-level policy in which an early stage and an advanced stage epidemic model applies respectively, illustrating thus how the early stage epidemic setup may be a useful approximation in concrete real world circumstances.

The rest of the paper proceeds as follows. Section 2 presents our macroeconomic–epidemiological framework in which the social planner seeks to determine the optimal intensity and duration of the mitigation policy (i.e., social distancing) to minimize the social costs of the epidemic management program. Section 3 focuses on the early epidemic case in which the number of infectives is still very low and thus the number of susceptibles can be approximated with the entire population. Section 4 discusses the advanced epidemic case, in which the number of infectives is already non-negligible with respect to the population size. In both cases we focus on the determination of the optimal intensity and duration of social distancing showing its implications on macroeconomic and health outcomes, and illustrating how social distancing might need to be implemented to control the diffusion of widespread diseases such as the seasonal flu and the common cold. Section 5 presents a calibration of our early and advanced epidemic setups to the Italian COVID-19 experience, showing how social distancing should be differently applied at national and regional level. Section 6 as usual concludes and presents directions for future research. Technicalities are postponed to Appendices A and B.

2. The model

We now present our macroeconomic–epidemiological setup in which the disease prevalence drives output production and social distancing and treatment policy are used to manage the epidemic. The epidemiological side is described by a policy-extended SIS model while in the macroeconomic one social distancing affects output and income taxation determines the availability of resources to finance therapeutic treatment. This gives rise to feedback effects between health and macroeconomic outcomes.

2.1. The epidemiological setup

Before introducing our macroeconomic–epidemiological setup, we briefly review the basic SIS model, having its origin in the seminal work by Kermack and McKendrick (1927). The SIS model is one of the simplest and most discussed frameworks in mathematical epidemiology, widely applicable to a range of diseases not conferring permanent immunity, such as the seasonal flu, some sexually transmitted diseases and some vector-borne diseases (Hethcote, 2008). According to its simplest formulation abstracting from vital dynamics, the population which is assumed to be constant and normalized to unity without loss of generality, \( N = 1 \), is composed by healthy individuals who are susceptible to the disease, \( s \), and the infectives who have already contracted the disease and can transmit it by getting in contact with susceptibles, \( i \). Thus, at any moment in time we have that \( 1 = s + i \), and the interactions between susceptibles and infectives determines the evolution of the two subpopulation groups. Infectives spontaneously recover at the rate \( \delta > 0 \), and susceptibles become infective by interacting with infectives which occurs at the rate \( \alpha > 0 \), measuring the number of social contacts required to give rise to a new infection (i.e., the product between the number of contacts between infectives and susceptibles per unit of time and the probability that one contact leads to disease transmission). This implies that the dynamics of susceptibles and infectives can be described through a dynamic system as follows:

\[
\begin{align*}
\dot{s}_t &= \delta s_t - \alpha s_t i_t \\
\dot{i}_t &= \alpha (1 - i_t) s_t - \delta s_t.
\end{align*}
\]

Since the population is normalized to one the equations above describe the evolution of the shares of susceptibles and infectives. By recalling that \( s_t = 1 - i_t \), the evolution and equilibria of the above system can be characterized by focusing simply on one of the two equations, say that for infectives which can be rewritten as follows:

\[
\dot{i}_t = \alpha (1 - i_t) s_t - \delta i_t.
\]

It is straightforward to verify that there exists two equilibria: \( i_t = 0 \) and \( i_t = \frac{\alpha}{\alpha + \delta} \). The former represents the disease-free equilibrium in which the share of infectives is null, while the
latter the endemic equilibrium in which the share of infective is strictly positive. Note that while the disease-free equilibrium exists for all parameter values, the endemic equilibrium exists only if $\alpha > \delta$. Moreover, by analyzing the (local) stability properties of the equilibria, it is possible to show that $\frac{\partial i}{\partial t} \bigg|_{i=0} = \alpha - \delta$ and $\frac{\partial i}{\partial t} \bigg|_{i=-\delta} = -(\alpha - \delta)$, suggesting thus that when only the disease-free equilibrium exists (i.e., $\alpha < \delta$) this will be asymptotically stable, while when also the endemic equilibrium exists (i.e., $\alpha \geq \delta$) this will be asymptotically stable while the disease-free equilibrium will become unstable; thus $\alpha = \delta$, represents a bifurcation point at which the number of equilibria along with their stability properties change. This suggests that the long run epidemic outcome entirely depends on the relative size of the speed of recovery and the speed at transmission: intuitively, whenever the speed of recovery exceeds (falls short of) the speed at transmission, $\delta > \delta (\delta < \alpha)$, in the long run the disease will be completely eradicated (will persist becoming endemic in the population).

This outcome can be equivalently seen in terms of the so-called “basic reproduction number”, $R_0$. This parameter plays a crucial role in mathematical epidemiology, as it measures the average number of secondary infections produced by a typical infectious individual introduced into a completely susceptible population (Hethcote, 2000, 2008). In our SIS framework it is possible to show that the basic reproduction number is given by the following expression:

$$R_0 = \frac{\alpha}{\delta}. \tag{4}$$

thus the long run outcome completely depends on whether this takes a value larger or smaller than unity. Indeed, when $R_0 < 1 (R_0 > 1)$ in the long run the disease will be completely eradicated (will persist) and thus the share of infectives will be zero (positive); this happens whenever the speed of recovery is faster (slower) than the speed of transmission. In the case of an epidemic outbreak in which $R_0 > 1$, mitigating policies which affect the speed of recovery or the speed of transmission (or both) can be used to lower the basic reproduction number and bring it below unity to achieve the long run eradication goal. A more detailed review of the SIS model and the role of mitigating (preventive and therapeutic) policies can be found in La Torre et al. (2020).

### 2.2. The macroeconomic–epidemiological setup

After having recalled the basic SIS model, we now extend it to account for the role of social distancing and treatment policies and to analyze its relation with macroeconomic outcomes. Specifically, we consider a short and endogenous time horizon framework in which the social planner decides the policy measures to reduce the spread of a communicable disease, along with the duration of the implemented policies, in order to minimize the social cost associated with the epidemic management program. We consider therapeutic treatment, $0 < \nu_t < 1$ which increases the speed of recovery of the infectives and social distancing which limits the social contacts by a percentage $0 < u_t < 1$ reducing thus disease transmission. The short time horizon suggests that saving and capital accumulation are irrelevant, thus we simply assume that individuals entirely consume their disposable income as follows: $c_t = (1 - \tau)y_t$, where $c_t$ denotes consumption, $y_t$ income and $0 < \tau < 1$ the tax rate. Output is produced through a linear production function by the number of susceptibles as follows: $q_t = s_t = 1 - i_t$, but since only a certain share of the social contacts, $1 - u_t$, is allowed to regularly occur output net of social distancing is given by: $y_t = (1 - u_t)q_t$. The tax revenue, $\tau y_t$, is entirely used to finance treatment by maintaining a balanced budget at any moment in time: $\nu_t = \tau y_t$, meaning that health policy is completely publicly provided. The disease dynamics is described by a SIS equation as follows: $i_t = \alpha (1 - \beta u_t) (1 - i_t)q_t - \delta (1 + \omega \nu_t) i_t$ where the transmission rate depends on the amount of social interactions between individuals (either on the workplace or for daily life activities). Treatment reduces the disease prevalence by increasing the speed of recovery from the disease by a factor $\omega > 0$, while social distancing lowers disease incidence by reducing the number of possible contacts between susceptibles and infectives by an amount $\beta > 0$.\(^1\) The social cost is the weighted sum of two terms: the discounted sum ($\rho > 0$ is the discount rate) of the instantaneous losses associated with the epidemic management program during its duration and the discounted final damage associated with the remaining prevalence level of the disease. The instantaneous loss function is assumed to depend on the spread of the disease and the output lost due to social distancing, and to take a quadratic non-separable form (La Torre et al., 2017, 2020) as follows: $c_t (i_t, u_t q_t) = \frac{1}{2} (\frac{\beta}{\delta} u_t q_t i_t + \beta (1 - \beta u_t) (1 - i_t)q_t)$, penalizing deviations from the disease-free status and from the no-production-loss scenario. The final damage function is assumed to depend only on the share of infectives remaining at the end of the epidemic management program and to take a linear form as follows: $d_f = d_f (\beta (1 - \beta u_t) (1 - i_t)q_t)$. The relative weight of the final damage in terms of the instantaneous losses is given by $\psi > 0$, which measures the concerns for long run health outcomes and depends on the degree of health concern, $\phi > 0$, and the final time period, $T$. This means that, independently of the degree of health concern, the weight attached to long run health outcomes critically depends on today’s distance from the long run date: if $T = 0$ the short and long runs coincide and thus an infinitely large weight is attached to the final damage, if $T \to \infty$ the long run is infinitely far away and thus the weight attached to the final damage is null; for positive but finite values of $T$ a positive and finite value is attached to the final damage giving rise to a clear tradeoff between the discounted sum of the instantaneous losses (which are minimized with $T \to 0$) and the discounted final damage (which is minimized with $T \to \infty$) ensuring thus that optimality will require that the epidemic mitigation policies will be used for a positive and finite amount of time. The social planner problem reads as follows:

$$\min_{u_t} c = \int_0^T \left\{ \frac{\beta}{\delta} u_t (1 - i_t^2) \right\} e^{-\rho t} dt + \frac{1}{T} \left( \int_0^T i_t \right) e^{-\rho T} \tag{5}$$

s.t. $\dot{i}_t = \alpha (1 - \beta u_t) (1 - i_t)q_t - \delta (1 + \omega \nu_t) i_t \tag{6}$

$0 \leq i_t, u_t \leq 1 \tag{7}$

$0 < \rho_0 < 1 \text{ given} \tag{8}$

$T > 0 \text{ free} \tag{9}$

Note the role of the degree of health concern in determining the relation between health and macroeconomic outcomes. If this is null the planner will not be much willing to reduce economic activity to lower disease incidence and thus prevalence in the short run as well as in the long run. If this is particularly high the planner will be very well willing to incur short run economic

\(^1\) Note that we have assumed that both treatment and social distancing affect linearly the epidemic dynamics, while we could also assume the effect to be nonlinear. Indeed, some economic epidemiology works conjecture that health policy is subject to decreasing returns and thus their effect on disease prevalence is less than linear (Gersovitz and Hammer, 2004). Such alternative formulation would yield an analytically tractable model making it thus impossible to derive closed-form solutions. Despite the lack of analytical tractability, it is reasonable to believe that the presence of nonlinear effects would give rise to results qualitatively similar to ours, apart from the fact that the lower effectiveness of health policy would lead from a quantitative point of view to smaller reductions in the share of infectives, which would thus result to be higher than in our setup at any moment in time.
losses (i.e., to increase the severity of social distancing) in order to slow down the epidemic dynamics and achieve at the end of the planning horizon a lower level of disease prevalence. Understanding thus how the intensity and the duration of the optimal policy depend on the degree of health concern may provide us with important information regarding the consequences of real world policymakers’ preferences on health and macroeconomic outcomes.

Before proceeding into the determination of the optimal policy it may be useful to comment on some features and implications of our modeling approach. (i) Consistent with the economic epidemiology literature, our setup allows to consider two different forms of health policy measures, prevention and treatment, where the former acts on incidence and the latter on prevalence. While therapeutic treatment is defined similar to extant literature (Gersovitz and Hammer, 2004; La Torre et al., 2020), prevention is determined by social distancing as in the recent COVID-19 experience (Acemoglu et al., 2020; Alvarez et al., 2020). Different from other preventive measures, such as vaccines or prophylactic interventions, which act only on the health status by lowering the disease transmission rate, social distancing has also important economic implications by reducing production. (ii) The interpretation of our setup in terms of COVID-19 requires careful considerations of its underlying assumptions. By abstracting from vital dynamics, we cannot account for the disease-induced mortality which represents an important element of the recent coronavirus epidemic experience since about half a million of deaths worldwide are attributable (either directly, through its related pneumonia complications, or indirectly, through the stress caused on healthcare facilities) to the disease. Therefore, we may expect the optimal policy in our setup to be substantially less strict than in other frameworks in which mortality effects have been explicitly taken into account (Acemoglu et al., 2020; Alvarez et al., 2020). Moreover, consistent with previous studies, given the large degree of uncertainty on parameter values which our calibrated optimal policies depend on, our conclusions can only be considered as a stylized benchmark for real world policymaking (Acemoglu et al., 2020; Manski and Molinari, 2020).

3. Early epidemic growth: Outbreak of the disease

The outbreak of a new epidemic does not generally translate in an immediate response from policymakers and thus the disease spreads freely among the population. Several studies in mathematical epidemiology document that in the early phase of an epidemic the number of infectives tends to grow at a constant (or even decreasing) rate, and thus the evolution of the disease can be described through an exponential growth (or sub-exponential growth) dynamics (Chowell et al., 2016; Ma, 2020). This argument is independent of the specific epidemiological framework used to characterize the disease spread and it is equally applicable to SIS, SIR or more sophisticated epidemiological models. In fact, for any of the above mentioned epidemic models, given $i_t$, the evolution of the epidemics is driven by a differential equation taking the form:

$$\dot{i}_t = f(i_t, s_t), \quad (10)$$

where $f(\cdot, \cdot)$ is a continuous and differentiable function. Similar to the SIS model seen in the previous section, the disease-free equilibrium is characterized by the pair $(0, 1)$ such that the absence of infectives results in no spread of the disease. If we take the first-order expansion of the right-hand side of the above equation, we get:

$$\dot{i}_t = f(i_t, s_t) \approx f(0, 1) + \frac{\partial f}{\partial i_t}(0, 1)(i_t - 0) + \frac{\partial f}{\partial s_t}(0, 1)(s_t - 1) \quad (11)$$

which, using the fact that $f(0, 1) = 0$ along with $s_t - 1 = -i_t$, implies that:

$$\dot{i}_t \approx \frac{\partial f}{\partial i_t}(0, 1)i_t - \frac{\partial f}{\partial s_t}(0, 1)i_t = \left[ \frac{\partial f}{\partial i_t}(0, 1) - \frac{\partial f}{\partial s_t}(0, 1) \right] i_t \quad (12)$$

This clearly explains why, in an early epidemic stage, the epidemic growth can be modeled via an exponential equation.

By using such a linear approximation and the assumption that in an early epidemic stage the share of infectives in the population is negligibly small, we can approximate the share of susceptibles with the entire population, that is $s_t = 1 - i_t \approx 1$. In such a setup our optimization problems (5)–(9) can be rewritten as follows:

$$\min_c \int_0^T \left\{ \frac{\dot{i}_t^2(1 + u_t^2)}{2} \right\} e^{-\rho t} dt + \phi_T i_T e^{-\rho T} \quad (13)$$

s.t. $\dot{i}_t = \alpha(1 - \beta u_t)i_t - \delta[1 + \omega t(1 - u_t)]i_t$

$$0 \leq i_t, u_t \leq 1 \quad (14)$$

$$0 < \theta_0 < 1 \text{ given} \quad (15)$$

$$T > 0 \text{ free} \quad (16)$$

Before proceeding with the determination of the optimal intensity and duration of the epidemic prevention policies, note that the evolution of the infectives in (14) is now given by the following differential equation:

$$\dot{i}_t = \alpha(1 - \beta u_t)i_t - \delta[1 + \omega t(1 - u_t)]i_t$$

where $\theta = \alpha - \delta - \delta\omega t$ represents the net infectives growth rate (i.e., the infectivity rate, $\alpha$, adjusted for recovery, $\delta$, and the treatment-augmented recovery, $\delta\omega t$) and $\mu = \alpha\beta - \delta\omega t$ the effectiveness of the social distancing policy (i.e., the social-distancing-reduced infectivity rate, $\alpha\beta$, net of the treatment-augmented recovery). Intuitively, given $\theta$ and $\mu$, the disease eradication goal will be eventually achieved only if the social distancing policy is stringent enough to reverse the growth trend of the infectives share. However, eradication does not necessarily need to be the most desirable outcome, since the economic costs of social distancing might lead the planner to prefer an endemic state by accepting a certain level of disease prevalence.

For the time being, let us suppose that $T$ is fixed (but unknown) and later we will determine a value of $T$ that minimizes the social cost functional in (13). Our analytical results, made possible by the quasi quadratic–linear structure of our problem, are summarized in Theorem 1 and Proposition 1. Theorem 1 states necessary optimality conditions for the existence of a optimal solution for the problem above. Proposition 1 instead determines some sufficient parameter conditions ensuring that the feasible solution is optimal, by exploiting the fact that the optimal control and the state and costate equations are bounded, meaning that the derived system of forward–backward differential equations has a Lipschitz structure (Jung et al., 2002; La Torre et al., 2020).

**Theorem 1.** Suppose that $T > 0$ is fixed and define $\psi = \sqrt{\rho - 2\theta^2} + 4\mu^2$. If the pair $(i_0, u_0)$, $0 < i_0 < 1$ and $0 < u_0 < 1$, is the optimal control problem given by (13)–(15) solves the following system of forward–backward differential equations:

$$\begin{align*}
\dot{u}_t &= \frac{\mu u_t e^{-\rho t}}{\rho} \\
\dot{i}_t &= -i_t(1 + u_t^2)e^{-\rho t} - (\theta - \mu u_t)\lambda_t \\
\lambda_t &= \frac{\psi}{e^{-\rho t}} \\
0 < \theta_0 &< 1 \text{ given}
\end{align*} \quad (18)$$

where $\lambda_t$ is the costate variable. The closed-form expressions of $i_t$ and $u_t$ are given by:

$$i_t = C_1 e^{1/2(\rho + \psi) t} + C_2 e^{1/2(\rho - \psi) t} \quad (19)$$
where

\begin{equation}
U_t = \frac{1}{2\mu} \left[ \frac{Ci e^{\xi T} (2\theta - \psi - \rho) + Cj e^{-\eta T} (2\theta + \psi - \rho)}{Ci e^{\xi T} + Cj e^{-\eta T}} \right] 
\end{equation}

Proposition 1. Assume $T \geq \mu \phi$ and $\psi > \rho$. If either of the following conditions:

1. $\phi > \frac{\delta_0 T e^{\xi T} (\rho - \psi + 2\theta)}{2\mu e^{\xi T}}$
2. $\phi < \frac{\delta_0 T e^{\xi T} (\rho - \psi + 2\theta)}{2\mu e^{\xi T}}$


\begin{equation}
C_1 = \frac{i_0 (\rho - \psi - 2\theta) e^{\xi T} (\rho - \psi + 2\theta) + 2\mu \phi}{(\rho - \psi - 2\theta) e^{\xi T} (\rho - \psi + 2\theta) + 2\mu \phi} \left[ (2\theta - \psi - \rho) i_0 - 2\mu \phi \right]
\end{equation}

\begin{equation}
C_2 = \frac{(\rho - \psi - 2\theta) e^{\xi T} (\rho - \psi + 2\theta) + 2\mu \phi}{(\rho - \psi - 2\theta) e^{\xi T} (\rho - \psi + 2\theta) + 2\mu \phi} \left[ (2\theta - \psi - \rho) i_0 - 2\mu \phi \right]
\end{equation}

or the following ones:

3. $\rho > 2\theta$
4. $\phi > \frac{\delta_0 T e^{\xi T} (\rho - \psi + 2\theta)}{2\mu e^{\xi T}}$
5. $\phi \leq \frac{\delta_0 T e^{\xi T} (\rho - \psi + 2\theta)}{2\mu e^{\xi T}}$

\begin{equation}
\frac{i_0 (\rho - \psi - 2\theta) e^{\xi T} (\rho - \psi + 2\theta) + 2\mu \phi}{(\rho - \psi - 2\theta) e^{\xi T} (\rho - \psi + 2\theta) + 2\mu \phi} \left[ (2\theta - \psi - \rho) i_0 - 2\mu \phi \right]
\end{equation}

are satisfied, then the solution $(i_t, u_t)$ given by (19) and (20), respectively, satisfies the inequalities $0 < i_t < 1$, $0 < u_t < 1$ and it is optimal for the control problem given by (13)–(15). Furthermore, $i_t < 0$ for any $t \in [0, T]$.

Theorem 1 determines explicitly the optimal time evolution of the control, $u_t$, and state, $i_t$, variables, which given the quasi quadratic–linear structure of the problem, are given by exponential functions. Proposition 1 identifies two alternative parameter configurations ensuring that such a solution is well defined, by determining a range of values for degree of health concern $\phi$ and for the rate of time preference $\rho$. A simple inspection of the optimality conditions in (36) jointly with the inequality constraints on $i_t$ and $u_t$ ensured by Proposition 1 allows to show that the following condition (which we will assume to hold true in what follows) needs to be satisfied:

\begin{equation}
T \geq \mu \phi.
\end{equation}

which suggests that the social distancing duration needs to be large enough and that such a duration increases with the effectiveness of the social distancing policy $\mu$ and the degree of health concern $\phi$. Under the parameter restrictions in Proposition 1, it directly follows that the share of infectives monotonically decreases over time. Intuitively, since in our SIS framework both the incubation and latent periods are null, the social distancing policy is optimal if reducing the disease incidence enough to bring the number of new infectives below the number of recovered ones such that disease prevalence will decrease during the entire duration of the epidemic management program. This also implies that during the whole planning horizon the share of infectives will always be lower than its initial value and thus the share of susceptibles can continue to be approximated with the entire population, suggesting that our linearization approach is applicable for the entire life of the social distancing policy. Even if the optimal share of infectives decreases over time, it is bounded from below at a null level thus it never reach zero suggesting that it will never be optimal to achieve complete disease eradication. The next proposition summarizes such a result.

Proposition 2. Let $(i_t, u_t)$, with $0 < i_t < 1$ and $0 < u_t < 1$, given by (19) and (20), be the optimal solution to the control problem given by (13)–(15). Then the disease eradication is not optimal for any $T > 0$.

Thus far, in our analysis we have assumed that $T$ is fixed (and thus our above conclusions apply independently of its specific value) while this needs to be endogenously derived as part of the optimization problem. Indeed, the optimal duration of the social distancing policy, other than satisfying the constraint $T \geq T_{\text{min}} \geq \mu \phi$ ($T_{\text{min}}$ could even be larger than $\mu \phi$ due to the presence of the other constraints implied by Proposition 1), is determined by minimizing the social cost functional, thus the determination of the optimal $T$ reduces to solving the following static problem:

\begin{equation}
\min_{T} C(T)
\end{equation}

s.t. $T \in [T_{\text{min}}, \infty)$.

The above problem may be characterized by either an interior or a corner solution according to the model’s parametrization. In particular, the expression of $C(T)$ is known and provided by the following expression:

\begin{equation}
C(T) = \phi e^{-T_1 T} + \frac{1}{4\mu^2} \left[ (\rho - 2\theta) (e^{-T_1 T} - \frac{\rho}{2}) \right. \\
\left. \left. + \psi \left[ C_1^2 \left( e^{-T_1} - 1 \right) + C_2^2 \left( 1 - e^{-T_1} \right) \right] \right],
\end{equation}

where $T_1 = \frac{2\mu \phi e^{-T_1 T} - 4\mu^2 \sinh(T_1 T)}{2\mu \phi e^{-T_1 T} + 4\mu^2 \cosh(T_1 T)}$, while $C_1$ and $C_2$ (which are functions of $T$ as well) are given in (21) and (22), respectively. Clearly, an interior solution of the above minimization problem can be determined by setting the derivative of $C(T)$ with respect to $T$ to zero, however, this expression is particularly cumbersome and it is not possible to determine it explicitly. The only conclusion that we can derive without a closed-form expression for the optimal $T$ is that this necessarily increases with the effectiveness of the social distancing policy and the degree of health concern, since these parameters determine a lower bound for the above optimization problem. Moreover, note that also all the analytical expressions for the optimal intensity of social distancing and the optimal share of infectives, given in Theorem 1, are particularly cumbersome due to the presence of several parameters and thus it is not possible a priori to perform any sort of comparative statics or dynamics exercise. We will thus proceed by illustrating how the dynamics of the share of infectives and the social distancing intensity, along with the optimal social distancing duration, may change under different model’s parametrizations.

Fig. 1 shows the determination of the optimal social distancing duration, $T$, by displaying the behavior of the cost functional $C$ (left panels), the dynamics of the share of infectives, $i_t$ (middle panels), and that of the social distancing intensity, $u_t$ (right panels), under two different disease configurations representing the cases of two widespread diseases, namely the seasonal flu (top panels) and the common cold (bottom panels). The value of the basic reproduction number and the time of recovery for each of these diseases are well known, and this information allows us to determine the relevant epidemiological parameters. Specifically, the recovery rate can be computed as the inverse of the average number of days to recover from the disease and, given the basic reproduction number and the recovery rate, the infectivity rate can be computed from (4) as: $\alpha = \delta R_0$. Estimates on seasonal flu suggest that $R_0 = 1.5$ and people recover in about seven days which implies that $\delta = 0.14$ and thus $\alpha = 0.21$; estimates on the common cold instead show that $R_0 = 2$ and in this case people recover in about ten days which implies that $\delta = 0.1$ and thus $\alpha = 0.2$ (Bailey, 1975; Coburn et al., 2008). In order to understand the effects of different epidemiological parameters on social distancing policy, we set the initial share of infectives to 5% of the susceptible population for both the diseases. We therefore set the epidemiological parameters as follows: $R_0 = 1.5, \alpha = 0.21, \delta = 0.14, i_0 = 0.05$ in the seasonal flu case and $R_0 = 2.0, \alpha = 0.2, \delta = 0.1$, $i_0 = 0.05$ in the common cold case.
\(\alpha = 0.2, \delta = 0.1, i_0 = 0.05\) in the common cold case. For both cases, the economic parameter values are set according to the macroeconomics literature as follows: \(\rho = 0.04/365\) (in order to transform the annual rate of time preference in a daily measure), \(\tau = 0.3, \beta = 0.6\) and \(\phi = [0.8, 1, 1.2]\), while we set \(\omega = 2.13\) for the seasonal flu and \(\omega = 3.94\) for the common cold. In such a parametrization \(\beta\) reflects the consumption share of GDP (as consumption needs exist also under social distancing, which thus can only lower the transmission rate by the amount that social contacts do not entail consumption activities), \(\phi\) is varied across three different values to understand the implications of the degree of health concerns on the optimal policy, while \(\omega\), quantifying the effectiveness of treatment measures which may differ from one disease to the next, is calibrated to ensure that the parameter restrictions in Proposition 1 are verified. Note that in our two different parametrizations the only source of difference is the epidemiological parameters and the effectiveness of disease-specific treatment measures in order to focus on the implications of different disease characteristics on social distancing. In particular, the common cold is characterized by a higher basic reproduction number and a higher effectiveness of treatment, along with a lower degree of infectivity and a lower recovery rate than the seasonal flu.

The figure shows that behavior of the optimal social distancing policy (which in this setting can be interpreted as the requirement to wear face masks or maintain physical distance from others) is similar for both the seasonal flu and the common cold, starting high and then decreasing over time. In particular, we can observe that both the intensity and the duration of social distancing are higher in the flu case and this leads the share of infectives to decrease by a larger amount in the case of the flu. Consistently with what discussed in our theoretical analysis, for both diseases the optimal policy does not lead to disease eradication, but rather to the convergence to an endemic state in which the share of infectives reaches a value of about 0.041 in the case of the flu or 0.0445 in the case of the cold, associated with a reduction in disease prevalence ranging from about 18 to 10 percent with respect to its initial level. In both cases the effects of the degree of health concern are intuitive: the higher the parameter the higher the intensity and the duration of social distancing, and thus the lower the level of disease prevalence at any moment in time.

### 4. Non-early epidemic growth: Spread of the disease

Apart from the early stage of an epidemic outbreak in which the disease dynamics is characterized by exponential growth, in later phases the evolution of the disease can be described by a fully-fledged epidemiological model. In our setup this implies that the spread of the disease follows a nonlinear SIS equation as discussed in the economic epidemiology literature. Therefore, in this context our optimization problem reads as follows:

\[
\min_{u_t} C = \int_0^T \left\{ \frac{t^2 [1 + u_t^2 (1 - i_t)]}{2} \right\} e^{-\omega t} dt + \frac{\phi}{T} e^{-\omega T} i_T \tag{27}
\]

subject to:

\[
i_t = \omega (1 - \beta u_t) (1 - i_t) i_t - \delta [1 + \omega \tau (1 - u_t) (1 - i_t)] i_t \quad \text{for } 0 \leq i_t, u_t \leq 1, \quad 0 < i_0 < 1 \text{ given} \quad T > 0 \text{ free} \tag{28, 29, 30, 31}
\]

As in the previous section, we first consider the case in which \(T\) is fixed (but unknown) and later we will determine a value of \(T\) that minimizes the social cost functional. Our analytical results are summarized in Theorem 2 and Proposition 3. By applying the same arguments as in the previous section, Theorem 2 states a necessary optimality condition for the existence of an optimal solution to models (27)–(31). Proposition 3 states some sufficient conditions ensuring that the feasible solution is effectively optimal.

**Theorem 2.** Suppose that \(T > 0\) is fixed. Then, the optimal solutions \((i_t, u_t), 0 < i_t < 1\) and \(0 < u_t < 1\), of the optimal control problem...
given by (27)–(29) solve the following system of forward–backward differential equations:

\[
\begin{align*}
\dot{i}_t & = (\alpha - \delta \omega \tau - \mu_i)(1 - i_t)i_t - \delta_i i_t \\
\dot{\lambda}_t & = -\lambda_t(\alpha - \delta \omega \tau)(1 - 2i_t) - i_t e^{-\rho t} + \delta \lambda_t \\
u_t & = \frac{\lambda_t e^{-\rho t}}{i_t(1 - i_t)} \\
\lambda_T & = \frac{\phi}{T} e^{-\rho T} \\
0 & < i_0 < 1 \text{ given,}
\end{align*}
\]

where \(\lambda_t\) is the costate variable.

**Proposition 3.** Assume \(T \geq \mu \phi\) and let \((i_t, u_t)\) be the solution of the system (32). Then \((i_t, u_t)\) satisfies the inequalities \(0 < i_t < 1\) and \(u_t > 0\) for any \(t \in [0, T]\). Furthermore, if \(u_t > 1\) for any \(t \in [0, T]\) then the pair \((i_t, u_t)\) is optimal for the control problem given by (27)–(29). Finally, if either \(u_t \leq \frac{\phi}{T} e^{-\rho t}\) or \(i_t > 1 - \frac{T}{\alpha - \delta \omega \tau}\) then \(i_t < 0\).

Theorem 2 shows that the control and state variables are determined as the solution of a simultaneous system of differential equations, which, different from what seen in the previous section, by being highly nonlinear cannot be solved explicitly. Exactly as in the previous section, a simple analysis of the optimality conditions in (32) allows to show that the following condition needs to be satisfied:

\[T \geq \mu \phi,\]  

which requires the social distancing duration to be large enough. Despite the absence of a closed-form expression for the optimal solution, **Proposition 3** states that, even without imposing any additional parameter restriction, the optimal share of infectives lies in the unit interval and the optimal social distancing intensity is nonnegative,\(^2\) meaning that results similar to those earlier discussed in the previous section apply. In particular, the fact that the share of infectives needs to be strictly positive implies that also in a non-early epidemic stage complete disease eradication is not optimal, and thus conclusions similar to those outlined in **Proposition 2** hold true. However, the lack of a closed-form solution for the above problem precludes us from the possibility to explicitly derive some analytical result not only about the intensity of the social distancing policy but also about its dynamics. In particular, we cannot state a priori whether the share of infectives will monotonically fall as in the previous section, and we can only determine some quite stringent conditions (depending on the value of either the intensity of social distancing or the share of infectives itself) under which this may be the case. Moreover, thus far we have not said anything about the optimal determination of \(T\) yet, but exactly as in the previous section this needs to be chosen by minimizing the social cost functional in (27). In order to shed some light on this, we will proceed by exemplifying the behavior of the optimal solution along with the determination of the optimal \(T\) under different model’s parametrizations, by applying the numerical algorithm briefly described in Appendix B.

Fig. 2 shows the determination of the optimal social distancing duration, \(T\), by displaying the behavior of the cost functional \(C\) (left panels), the dynamics of the share of infectives, \(i_t\) (middle panels), and that of the social distancing intensity, \(u_t\) (right panels), under two different disease configurations representing the cases of the seasonal flu (top panels) and the common cold (bottom panels). The economic and epidemiological parameters are set exactly as in the previous section, and we can observe that from a qualitative point of view the results are identical to those presented earlier. For both diseases the optimal social distancing policy starts high and then decreases over time, and both its intensity and duration are higher in the case of the seasonal flu and numerical analysis we will check that this further condition is verified ex-post in order to ensure that the optimal solution is effectively feasible.
this leads the share of infectives to decrease by a larger amount in the flu case. Also in an advanced epidemic stage framework, for both diseases the optimal policy implies convergence to an endemic state, in which the share of infectives reaches a value of about 0.039 in the case of the flu or 0.043 in the case of the cold, associated with a reduction in disease prevalence ranging from about 22 to 14 percent with respect to its initial level. Intuitively, even if the qualitative effects are similar, from a quantitative point of view in an advanced epidemic stage social distancing is stricter and lasts for longer than in the early epidemic stage, and thus the final level of prevalence results to be lower in the case of both diseases. The effects of the degree of health concern are exactly the same as in the previous section.

5. A COVID-19 application: the Italian case

Even if our model is not specifically designed to capture the peculiarities of COVID-19, we now show that our general framework can be applied (with the limitations following to the lack of disease-induced mortality) also to determine the optimal social distancing policy in the case of this emerging disease. Italy has been strongly affected by the COVID-19 outbreak and it has registered thus far the highest number of deaths and the second highest number of reported cases among European continental countries (Dong et al., 2020). The first reported case has been filed in late January and by the end of February more than 1000 other cases have been reported. In early March the Italian government has introduced its first social distancing policies imposing the lockdown of a number of non-essential businesses and the wearing of face mask to reduce the number of contacts between individuals and the probability that each single contact may lead to disease transmission, respectively. Similar and more stringent policies and regulations have been introduced between March and April, leading on the one hand to a substantial reduction in the epidemic growth and on the other hand to a dramatic drop in economic activity. In order to get a sense of these effects, in Fig. 3 we illustrate the daily evolution of the disease incidence (left panel) and quarter-to-quarter GDP growth (right panel) since late January to late June 2020. This clearly shows that while it took about one month for the effects of social distancing to be visible on the disease incidence, those on GDP growth became evident in just a matter of days. GDP growth has fallen rapidly since late March while incidence has slowed down significantly only since early May.

In order to determine the optimal balancing between its health benefits and economic costs in the case of COVID-19, we now determine the optimal intensity and duration of social distancing (which can be interpreted not only in terms of the requirement to wear face mask or to maintain physical distance from others, but also as the introduction of a lockdown measure) by calibrating our theoretical framework to Italian data. By following the same procedure discussed earlier, we use estimates of the basic reproduction number and the recovery rate to infer the infectivity rate. Italian estimates show that $R_0 = 2.79$ and that recovery takes about three weeks (Remuzzi and Remuzzi, 2020), thus we set $\delta = 0.0476$ from which it follows that $\alpha = 0.1328$. The instantaneous rate of time preference is set at $\rho = 0.04/365$ (Barro and Sala-i Martin, 2004) and the tax rate at $\tau = 0.3$ (Di Nicola et al., 2017), the effectiveness of treatment measures is calibrated as $\omega = 8.23$ in order to ensure that the optimization problem is well defined (i.e., the constraints $0 \leq i \leq 1$ and $0 \leq u_t \leq 1$ are met), the degree of health concerns and the effectiveness of social distancing have been normalized to unity ($\beta = 1$ and $\phi = 1$ for simplicity, while $i_0$ is varied between different levels to show the implications of different initial conditions on the optimal policy. As today, the number of COVID-19 cases at national level (about 300,000) is completely negligible (i.e., considering that Italy is populated by 60,461,826 people, it amounts to a share of infectives of 0.0049) thus we can apply our early epidemic stage model to characterize the national dynamics. However, the same is not true at regional level where we can see that in specific regions at the epicenter of the outbreak the number of cases has been substantially large, thus we can apply our advanced epidemic stage model to characterize the regional dynamics.

In the first phase of the Italian coronavirus epidemic at national level the disease has evolved according to an exponential pattern (Remuzzi and Remuzzi, 2020), consistent with the assumption underlying our early epidemic growth setup. By considering different initial share of infectives ($i_0 = [0.02, 0.03, 0.04]$), we account for the large uncertainty (due to existence of asymptomatic individuals that have never been tested) regarding the exact number of infectives present today in the Italian economy (Lazzerini and Putoto, 2020). Fig. 4 shows the determination of the optimal social distancing duration, $T$ (left panel), the dynamics of the share of infectives, $i_t$ (middle panel), and of the social distancing intensity, $u_t$ (right panel) in the case of COVID-19 at Italian national level. We can observe that the social distancing policy needs to be applied for about 9–16 days according to the initial share of infectives, and in particular the higher the initial share of infectives the lower the duration and the intensity of the social distancing policy. This counterintuitive result is driven by the fact that the higher the initial infectives share the higher the social cost associated with inaction, and some policy intervention increases this social cost further; therefore, with a higher initial share of infectives it is optimal to implement a softer and shorter-living policy. Independently, of the initial infectives share the social distancing policy starts high to then gradually decrease, generating a smooth reduction in the level of disease prevalence to reach a final value ranging from 0.01 to 0.03: the optimal policy requires COVID-19 to become endemic in the Italian population.
At a regional level, the outbreak has hit particularly strong certain local areas, such as the Bergamo region where some studies estimate that more than 40% of the local population has contracted the virus (Buonanno et al., 2020), thus in such a setting our advanced epidemic growth setup seems more appropriate. According to these rough estimates and in order to account for some uncertainty along the lines of our earlier approach for the national level, we assume different initial share of infectives \( i_0 = \{0.2, 0.3, 0.4\} \). Fig. 5 shows the outcome of our numerical analysis for the case of COVID-19 at regional level in Bergamo. The social distancing policy needs to be applied for about 2–4 days according to the initial share of infectives with a mild intensity, decreasing from about 0.06–0.07 to 0.005. Such a mild policy is however enough to reverse the disease growth pattern, leading thus the share of infectives to reach a final value ranging from about 0.2 to 0.35. Also at a regional level the optimal policy requires COVID-19 to become endemic in the population, and the effects of the initial share of infectives on both the intensity and duration of social distancing are similar to those earlier discussed at national level.

Both in the national- and regional-level calibrations, our optimal policy turns out to be substantially different from those derived in other papers analyzing the optimal intensity and duration of social distancing policies aiming to manage the COVID-19 epidemic, and such differences are reflected also in the evolution of the infectives (Acemoglu et al., 2020; Alvarez et al., 2020). Indeed, while our social distancing policy is characterized by a monotonically decreasing intensity and a short duration ranging from 2 to 16 days leading to a monotonically decreasing share of infectives, the optimal policies both in Acemoglu et al. (2020) and Alvarez et al. (2020) are characterized by bell-shaped intensities and long durations ranging from 100 to 400 days in their baseline scenarios, which in turn yields a non-monotonic dynamic in the spread of the disease. Such neat dissimilarities are the result of the differences in the economy under consideration in the calibration exercise (Italy vs US), in the analytical frameworks (SIS vs SIR contexts) and in the assumptions underlying the disease characteristics (absence vs presence of disease-induced deaths). Moreover, the types of social distancing policy under consideration are quite different as well. Indeed, we focus on a policy that only reduces the number of contacts between infectives and susceptibles and thus disease incidence along with its implications on treatment through the economic–epidemiological feedback effects. Alvarez et al. (2020) instead analyze the implications of a lockdown policy which resembles more a quarantine than a true lockdown since a certain share of both infectives and susceptibles is isolated from the rest of the population, while Acemoglu et al. (2020) investigate the mutual links between lockdown measures and other forms of policy interventions such as vaccination. Given such discrepancies between our approach and that employed in other related papers, it is difficult to perform a true comparative analysis but nevertheless the difference in the optimal duration of the social distancing policies is striking, and we believe that the short social distancing duration underlying our analysis appears to be consistent with real-world experiences.

By comparing our optimal policy associated with the examples discussed in Sections 3 and 4 with that derived from the COVID-19 case at Italian national level (since the initial share of infectives are comparable in these cases), even if the economic parameters are different and thus such a comparison may be misleading, we can observe that social distancing is characterized by a larger variability in the evolution of its intensity and a longer duration in the case of COVID-19 than in the seasonal flu and common cold cases. Even without considering its eventual effects on mortality, the epidemiological peculiarities of COVID-19 (a high basic reproduction number and a low recovery rate) require social distancing measures to be applied for longer periods than in the case of more common diseases.

5.1. Social distancing vs treatment

In order to better understand the working mechanisms underlying our epidemic management program, which is characterized by a simultaneous application of social distancing and treatment measures, we now compare the effectiveness of social distancing and treatment in isolation. In particular, we focus on the Bergamo area which is probably the most interesting case to understand the validity of the different health policy measures, and we rely on the same parametrization earlier employed in which the initial share of infectives is assumed to be equal to 0.2. The effectiveness of social distancing and treatment is captured by the parameters \( \beta \) and \( \omega \) respectively, thus by setting one of these parameters to zero we can analyze how our model would perform in the absence of one of the two policy measures. Therefore, we compare the results we have obtained earlier in which social distancing and treatment are applied simultaneously (i.e., \( \beta > 0 \) and \( \omega > 0 \)), with those derived in a setting in which either only social distancing (\( \omega = 0 \)) or only treatment (\( \beta = 0 \)) is implemented.

Fig. 6 shows the outcome of our analysis distinguishing between situations in which \( \beta > 0 \) and \( \omega > 0 \) (solid curve), \( \omega = 0 \) (dashed curve), and \( \beta = 0 \) (dashed and dotted curve). With both treatment and social distancing (\( \beta > 0 \) and \( \omega > 0 \)) the optimal social distancing policy is characterized by a mild intensity, which however is enough to reduce the disease prevalence over time. In the absence of social distancing (\( \beta = 0 \), which leads to \( u_t = 0 \), for all \( t \in [0, T] \)), health policy is entirely driven by treatment which is enough per se to lead to a monotonic reduction in the disease prevalence, even if the share of infectives results to be higher than in the previous case with the additional help of social distancing. In the absence of treatment (\( \omega = 0 \)), the optimal social distancing
policy is characterized by a very high intensity but nevertheless it is not enough to reverse the growth pattern in the disease dynamics and thus the share of infectives monotonically increases over time. The optimal duration of the epidemic management program is similar in the three cases, thus we can focus our comparison on the differences in the social distancing intensity and in the share of infectives.

The comparison among these three cases under investigation allows us to stress the importance of treatment: social distancing alone is not enough to reduce the disease prevalence over time while treatment alone can be. This result may be explained by the specific epidemiological features of COVID-19. Since such a disease is characterized by a high basic reproduction number and a low recovery rate, the key element to successfully reduce its spread consists of increasing the speed of recovery which is exactly the role of treatment. Since social distancing acts by reducing the number of contagious contacts and thus does not affect the speed of recovery it plays only a secondary role in this context, but nevertheless this does not mean that social distancing is not important. Indeed, by computing the social costs associated with the three scenarios analyzed, which turn out to be equal to \( C = 0.1123 \) in the \( \beta > 0 \) and \( \omega > 0 \), \( C = 0.1390 \) in the \( \omega = 0 \) case, and \( C = 0.1625 \) in the \( \beta = 0 \) case, we can conclude that social distancing plays an important role in reducing the social costs of the epidemic management program. The scenario with no social distancing is clearly the least desirable from the social cost perspective while that with no treatment outperforms the previous one but it is less desirable than the scenario in which both treatment and social distancing are simultaneously applied. By reducing disease incidence, social distancing permits to lower the epidemic growth pattern allowing treatment to speed up the recovery of a lower number of infective individuals, reaching thus the lowest social costs among the three scenarios.

6. Conclusion

The recent COVID-19 epidemic experience has spurred a growing interest in understanding the mutual relation between macroeconomic and epidemiological outcomes, along with the role of health-economic policy in affecting such a relation. Different from previous epidemics, policymakers have tried to control the spread of the disease through social distancing policies, implemented by imposing the requirement to wear face masks or to maintain a certain physical distance from others, by forcing individuals to quarantine and voluntary isolations, or by introducing restrictions on individual mobility along with (complete or partial) lockdowns. Due to the widespread use of social distancing policies, which have never been implemented on such a large scale before, it is essential to understand their implications on both health and economic outcomes. This paper wishes to contribute to this goal by developing a macroeconomic–epidemiological model in which the social planner aims to minimize the social costs of an epidemic management program by considering that social distancing on the one hand lowers the disease incidence by reducing social contacts and on the other hand it reduces output by hindering economic production activities.

Our framework is general enough to allow for the determination of the optimal intensity and the duration of social distancing in the cases of a number of diseases, some very common (such as the seasonal flu and common cold) and others emerging (such as COVID-19), identifying how the optimal policy may change in early or advanced epidemic stages. We show that both the optimal intensity and duration of social distancing may largely vary according to the epidemiological characteristics of specific diseases, and that the balancing between health benefits and the economic costs associated with social distancing requires to accept that the disease will reach an endemic state in the
population, as confirmed in all our model’s parametrizations aiming to capture the peculiarities of the seasonal flu, the common cold and COVID-19. In our analysis we have focused on a SIS framework in which individuals never acquire immunity from the disease, but we could alternatively consider a SIR framework to allow for the possibility that infectives gain permanent immunity upon recovery. Apart from making the structure of the model a bit more complicated, since this requires to consider the joint evolution of three different population subgroups (i.e., infective, susceptible and recovered individuals), this will not modify our main conclusions from a qualitative point of view. From a quantitative perspective, the main difference would rest in the duration of the optimal social distancing policy, which will tend to be shorter in a SIR than in a SIS setting. Indeed, since in a SIR context comparable to our SIS model (that is, abstracting from vital dynamics) the share of susceptibles monotonically decreases over time as individuals gain immunity, the need to prevent new infections naturally falls as time goes by and this translates into a shorter duration of the epidemic management program.

To the best of our knowledge no paper has tried to analyze the implications of social distancing in a way comparable to ours. Several papers analyze the determination of the optimal lockdown intensity and duration in the case of COVID-19 by numerical simulations, and only few works investigate the issue analytically by treating the lockdown policy as a mere parameter. Our attempt to determine analytically the optimal social distancing intensity and duration, not only focusing on the case of COVID-19 but on communicable disease in general, is to some extent unique. In order to develop an analytically tractable framework, we have had to introduce a number of simplifying assumptions which however limit the ability of our model to capture specific issues brought to light by the recent COVID-19 experience. In particular, by abstracting from vital dynamics and disease-induced mortality, our model cannot properly quantify the true social costs associated with the coronavirus outbreak, and thus it should come to no surprise that in our setup the optimal policy requires social distancing to be mild and short-lived. Moreover, by assuming that treatment is entirely funded through income taxation via balanced-budget considerations our model cannot capture the long run consequences on the public finance sustainability of the growing need to issue public debt to fund the high coronavirus treatment expenses. Extending the analysis to address these further issues is left for future research.

**Declaration of competing interest**

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

**Appendix A. Technical appendix**

**A.1. Proof of Theorem 1**

The Hamiltonian $H(i, u, λ_t)$, associated with the optimal control problems (13)-(15) reads as:

$$H = \frac{1}{2} [1 + u_i^2] e^{-\rho t} + \lambda_t (θ - μ u_i) i_t$$

where $θ = α - δ - δ \sigma, μ = α β - δ \omega t$, and $λ_t$ is the costate variable. The optimality conditions can be stated as follows:

$$\begin{align*}
u_i^2 e^{-\rho t} &= μ λ_t i_t \\
-λ_t &= i_t [1 + u_i^2] e^{-\rho t} + (θ - μ u_i) λ_t \\
i_t &= (θ - μ u_i) i_t \\
λ_t &= \frac{θ}{T} e^{-\rho T} \\
i_0 \text{ given}
\end{align*}$$

(34)

Note that in order to derive the expression of $u_t$, we suppose that $i_t \neq 0$. The degenerate case in which $i_t = 0$ implies that any $u_t$ can be chosen. By plugging the expression of $u_t$ into the equation of $λ_t$, we get:

$$\dot{λ}_t = -λ_t \left[1 + \left(\frac{μ λ_t e^{ρt}}{i_t}\right)^2\right] e^{-ρt} - \left[θ - μ \left(\frac{μ λ_t e^{ρt}}{i_t}\right)\right] λ_t$$

which, after simplifications, leads to the following expression:

$$\dot{λ}_t = -i_t e^{-ρt} - θ λ_t$$

By plugging the expression of $u_t$ into the equation of $i_t$ we obtain:

$$i_t = \theta i_t - μ^2 λ_t e^{ρt}$$

By differentiating the expression of $i_t$ with respect to time, we get:

$$\dot{i}_t = \theta i_t - μ^2 (i_t e^{ρt} + ρ e^{ρt} λ_t)$$

and plugging the expression of $\dot{λ}_t$ yields:

$$\dot{i}_t = \theta i_t - μ^2 \left[-i_t e^{-ρt} - θ λ_t\right] e^{ρt} + ρ e^{ρt} λ_t]
\dot{i} = θ i_t - μ^2 i_t + (θ - ρ) (i_t - θ i_t)
= ρ i_t + μ^2 i_t - (θ - ρ) |i_t|
that leads to:

$$\dot{i}_t - ρ i_t + (μ^2 + θ^2 - θ ρ) i_t = 0$$

The characteristic polynomial is given by the following expression:

$$\alpha^2 - ρ α + (μ^2 + θ^2 - θ ρ) = 0$$

whose solutions are given by:

$$α_1 = \frac{ρ + \sqrt{ρ^2 + 4μ^2 + 4θ^2 - 4θ ρ}}{2}
α_2 = \frac{ρ - \sqrt{ρ^2 + 4μ^2 + 4θ^2 - 4θ ρ}}{2}$$

which implies that the solution of the above second-order differential equation is the following:

$$i_t = C_1 e^{\frac{ρ + \sqrt{ρ^2 + 4μ^2 + 4θ^2 - 4θ ρ}}{2 t}} + C_2 e^{\frac{ρ - \sqrt{ρ^2 + 4μ^2 + 4θ^2 - 4θ ρ}}{2 t}}$$

where $ψ^2 = (ρ - 2θ)^2 + 4μ^2$. In order to determine the values of the two unknown constants $C_1$ and $C_2$, note that when $t = 0$ we have $i_{t=0} = i_0$ which implies that $C_1 + C_2 = i_0$. To get another equation, let us plug the expression of $i_t$ into the following:

$$λ_t = \frac{e^{-ρt}}{-μ^2 [i_t - θ i_t] = \frac{e^{-ρt}}{-μ^2} \left[C_1 (ρ + ψ) i_t e^{\frac{ρ + ψ}{2 t}} + C_2 (ρ - ψ) i_t e^{\frac{ρ - ψ}{2 t}}\right]}
+ C_2 (ρ - ψ) i_t e^{\frac{ρ - ψ}{2 t}} - θ \left(C_1 e^{\frac{ρ + ψ}{2 t}} + C_2 e^{\frac{ρ - ψ}{2 t}}\right)\right]\right]$$

which by plugging $t = T$ and using the terminal condition for $λ_T$ yields:

$$\begin{align*}
\dot{e}^{-ρT} &\left[C_1 (ρ + ψ) i_t e^{\frac{ρ + ψ}{2 t}} + C_2 (ρ - ψ) i_t e^{\frac{ρ - ψ}{2 t}}\right] = \frac{θ}{T} e^{-ρT},
\end{align*}$$
that is:

\[ C_1 \left( \frac{\rho + \psi}{2} - \theta \right) e^{(\rho + \psi) T} + C_2 \left( \frac{\rho - \psi}{2} - \theta \right) e^{-(\rho - \psi) T} = -\frac{\mu^2 \phi}{T} \]

By using \( C_1 = i_0 - C_2 \), we finally get the following expressions of \( C_1 \) and \( C_2 \):

\[
C_1 = \frac{i_0 (\rho - \psi - 2 \theta) e^{1/2(\rho - \psi) T} + 2 \mu^2 \phi}{(\rho - \psi - 2 \theta) e^{1/2(\rho - \psi) T} + e^{1/2(\rho + \psi) T} (2 \theta - \psi - \rho)}.
\]

\[
C_2 = i_0 - C_1 = \frac{(\rho - \psi - 2 \theta) e^{1/2(\rho + \psi) T} + e^{1/2(\rho - \psi) T} (2 \theta - \psi - \rho)}{(\rho - \psi - 2 \theta) e^{1/2(\rho + \psi) T} + e^{1/2(\rho - \psi) T} (2 \theta - \psi - \rho)}.
\]

Note that, after plugging in the expressions of \( C_1 \) and \( C_2 \), the equation of \( i_t \) takes the form:

\[
i_t = e^{u_T(\rho - \psi)} \left[ (i_0 - C_2) e^{\theta T} + C_1 \right] = e^{u_T(\rho - \psi)} \left( [i_0 - C_2] e^{\theta T} + C_1 (1 - e^{\theta T}) \right)
\]

\[
= e^{u_T(\rho - \psi)} \left[ \frac{\mu \lambda_T e^{\rho T}}{\tilde{\lambda}} \right] + e^{u_T(\rho - \psi)} \left( \frac{\mu \lambda_T e^{\rho T}}{\tilde{\lambda}} \right),
\]

\[
= \left( \frac{\mu \lambda_T e^{\rho T}}{\tilde{\lambda}} \right) C_1 e^{\rho T}(2 \theta - \psi - \rho) + C_2 e^{-\rho T}(2 \theta + \psi - \rho)
\]

The optimal level of the control variable is instead determined by the optimality condition:

\[
u_t = \frac{\mu \lambda_T e^{\rho T}}{\tilde{\lambda}}
\]

\[
= \frac{1}{2} \left( \frac{C_1 e^{\rho T}(2 \theta - \psi - \rho) + C_2 e^{-\rho T}(2 \theta + \psi - \rho)}{C_1 e^{\rho T} + C_2 e^{-\rho T}} \right)
\]

A.2. Proof of Proposition 1

First of all, note that simple considerations about the expression of \( u_t \) in (34) and the constraints on \( i_t \) and \( u_t \) imply that:

\[
\lambda_T = \frac{u_T i_T e^{-\rho T}}{\mu} = \frac{\phi e^{-T}}{T}
\]

\[
\phi = \frac{u_T i_T}{\mu} \leq \frac{1}{\mu}
\]

from which it follows that \( T \geq \mu \phi \). Let us now suppose that conditions (1)–(2) are satisfied.

1. We first prove that \( C_1 < 0 \). The sign of \( C_1 \) depends on the sign of its numerator, as its denominator is always negative. By simple calculations we get the following chain of equivalent inequalities:

\[
i_0 (\rho - \psi - 2 \theta) e^{1/2(\rho - \psi) T} + 2 \mu^2 \phi > 0
\]

\[
i_0 (\rho - \psi - 2 \theta) e^{1/2(\rho - \psi) T} > -\frac{2 \mu^2 \phi}{T}
\]

\[
T e^{1/2(\rho - \psi) T} < -\frac{2 \mu^2 \phi}{i_0 (\rho - \psi - 2 \theta)}
\]

which implies the conclusion. Elementary considerations about the sign of all involved parameters imply that \( i_t < 0 \) for all \( t \in [0, T] \). This also implies that \( i_T < 1 \).

2. We now prove that \( i_t > 0 \). In order to determine the sign of \( i_t \), let us proceed by noticing that:

\[
C_1 e^{\rho T}(2 \theta - \psi - \rho) + C_2 e^{-\rho T}(2 \theta + \psi - \rho) = C_2 e^{\rho T}(2 \theta - \psi - \rho) \left[ \frac{C_1}{C_2} e^{\rho T} + 1 \right]
\]

and, due to the negativity of \( C_1 \), to demonstrate the positivity of \( i_t \) for any \( t \in [0, T] \), it is enough to prove that:

\[
\frac{C_1}{C_2} e^{\rho T} + 1 > 0
\]

The following result provides a sufficient condition for this purpose. The following inequality:

\[
\frac{C_1}{C_2} e^{\rho T} + 1 > 0
\]

can be written in equivalent form as follows:

\[
i_0 (\rho - \psi - 2 \theta) e^{1/2(\rho - \psi) T} + 2 \mu^2 \phi > -2 \mu^2 \phi + e^{1/2(\rho + \psi) T} (2 \theta - \psi - \rho) i_0
\]

which is equivalent to

\[
i_0 (\rho - \psi - 2 \theta) e^{1/2(\rho + \psi) T} + 2 \mu^2 \phi
\]

\[
< i_0 (\rho + \psi - 2 \theta) e^{1/2(\rho + \psi) T} + 2 \mu^2 \phi T
\]

By simple calculations and rearranging the terms we get:

\[
-2 \psi i_0 e^{1/2(\rho + \psi) T} < 2 \mu^2 \psi e^{T} (1 - e^{T})
\]

and then the thesis follows. Same calculations show that \( \lambda_T > 0 \) for all \( t \in [0, T] \).

3. It remains to prove that \( u_t < 1 \). This is equivalent to prove that:

\[
\frac{C_1 e^{\rho T}(2 \theta - \psi - \rho) + C_2 e^{-\rho T}(2 \theta + \psi - \rho)}{C_1 e^{\rho T} + C_2 e^{-\rho T}} < 2 \mu
\]

that is:

\[
\frac{C_1 e^{\rho T}(2 \theta - \psi - \rho) + C_2 e^{-\rho T}(2 \theta + \psi - \rho)}{C_1 e^{\rho T} + C_2 e^{-\rho T}}< 2 \mu \left[ \frac{C_1 e^{\rho T} + 1}{C_2} \right]
\]

\[
\frac{C_1 e^{\rho T}(2 \theta - \psi - \rho) + (2 \theta + \psi - \rho)}{C_1 e^{\rho T} + C_2 e^{-\rho T}}< 2 \mu \left[ \frac{C_1 e^{\rho T} + 1}{C_2} \right]
\]

\[
\frac{C_1 e^{\rho T}(2 \theta - \psi - \rho - 2 \mu)}{C_1 e^{\rho T} + C_2 e^{-\rho T}} < (2 \mu - 2 \theta - \psi - \rho)
\]

and finally:

\[
\frac{C_1 e^{\rho T}}{C_2} > \frac{2 \mu - 2 \theta - \psi + \rho}{2 \theta - \psi - \rho - 2 \mu}
\]

and the negativity of \( C_1 \) implies that this inequality has to be satisfied at \( t = T \), leading to the following expression:

\[
\frac{C_1 e^{\rho T}}{C_2} + 1 > \Omega
\]

where

\[
\Omega = \frac{-2 \psi}{2 \theta - \rho - 2 \mu - \psi} \geq 0
\]

Easy calculations similar to those done in the previous point allow to conclude that \( u_t < 1 \) for any \( t \in [0, T] \).

Now let us suppose that conditions (3)–(5) are satisfied instead. Note that condition (3) implies that \( \Omega > 1 \).

1. We first prove that \( C_1 > 0 \). The calculations on its sign are similar to those presented above and thus we omit them. The positivity of \( C_1 \) trivially implies that \( i_t > 0 \) and \( \lambda_T > 0 \) for all \( t \in [0, T] \).

2. To prove that \( i_t < 0 \) is equivalent to show that:

\[
\frac{C_1}{2} e^{\rho T} + \frac{2}{2} e^{1/2(\rho + \psi) T} + C_2 \frac{\rho - \psi}{2} e^{1/2(\rho - \psi) T} > 0
\]
which is equivalent to require that:
\[
\frac{C_1 e^{\phi t}}{C_2} < \frac{\psi - \rho}{\psi + \rho}
\]
which, by elementary algebra, equivalent to:
\[
-4\psi \theta i_0 e^{1/2(\rho + \psi)} > -\frac{2\phi \mu^2}{T} \left[ (\psi + \rho) e^{\psi T} + \psi - \rho \right]
\]
and then the thesis follows. This also implies that \(\dot{i}_T < 1\).

3. The proof that \(u_t < 1\) is very similar to the previous one and we omit it.

A.3. Proof of Proposition 2

Since \(i_t\) satisfies the following differential equation: \(\dot{i}_t = (\theta - \mu u_t) i_t\), it follows that:
\[
i_t = i_0 e^{\int_0^t (\theta - \mu u_t) dt}
\]
which, thanks to the fact that \(i_0 > 0\), also implies that \(\dot{i}_t > 0\).

Note that the result that eradication is never optimal in our setting is due to the fact that the time horizon is finite, but in a different infinite-horizon context the results may be different. Indeed, since the closed-form solution of \(i_t\) is provided by the following expression:
\[
\dot{i}_t = \frac{2i_t \psi e^{\psi T} - 4\mu^2 \sinh(\frac{1}{2} \psi T)}{2 \psi \cosh(\frac{1}{2} \psi T) + 2(\rho - 2\theta) \sinh(\frac{1}{2} \psi T)}
\]
it is easy to verify that \(\lim_{T \to +\infty} \dot{i}_t = 0\), thanks to the condition \(\psi > \rho\).

A.4. Proof of Theorem 2

The Hamiltonian \(H(i_t, u_t, \lambda_t)\), associated with the optimal control problem (27)–(29) reads as:
\[
H = \frac{i_t^2}{2} \left[ 1 + u_t^2 (1 - i_t)^2 \right] e^{-\rho t} + \lambda_t \left[ (i(1 - i_t) - \delta) i_t \right]
- (\alpha \beta - \delta \omega \tau) u_t (1 - i_t) - \delta \omega \tau i_t (1 - i_t)\]
\[
= \frac{i_t^2}{2} \left[ 1 + u_t^2 (1 - i_t)^2 \right] e^{-\rho t} + \lambda_t \left[ (\alpha - \delta \omega \tau) i_t (1 - i_t) \right]
- \delta i_t - (\alpha \beta - \delta \omega \tau) u_t i_t (1 - i_t)\]
\]
and the optimality conditions can be stated as follows:
\[
\begin{align*}
\dot{i}_t &= (\alpha - \delta \omega \tau - \mu u_t) (1 - i_t) i_t - \delta i_t \\
\dot{\lambda}_t &= -\lambda_t (\alpha - \delta \omega \tau) (1 - 2i_t) - i_t e^{-\rho t} + \delta \lambda_t \\
u_t &= \frac{\lambda_t e^{-\rho t}}{i_t (1 - i_t)} \\
\lambda_T &= \frac{\phi}{T} e^{-\rho T} \\
i_0 & \text{ given}
\end{align*}
\]
(35)

A.5. Proof of Proposition 3

Let us first note that since \(u_T = \frac{\lambda_T e^{-\rho T}}{i_T (1 - i_T)}\), we get the following condition:
\[
\lambda_T = \frac{u_T e^{-\rho t} i_t (1 - i_t)}{\mu} = \frac{\phi}{T} e^{-\rho T}
\]
which implies that:
\[
\phi = \frac{u_T i_t (1 - i_t)}{\mu} \leq \frac{1}{\mu T}
\]
(36)
from which it follows that \(T \geq \phi\mu\).

In order to prove that \(i_t > 0\) let us notice that since \(i_t\) solves the following differential equation:
\[
\dot{i}_t = \frac{\partial H}{\partial \lambda_t} = (\alpha - \delta \omega \tau - \mu u_t) (1 - i_t) i_t - \delta i_t
\]
then the following result follows:
\[
\dot{i}_t = i_0 e^{\int_0^t (\alpha - \delta \omega \tau - \mu u_t) (1 - i_t) - \delta dt}
\]
which implies that \(i_t > 0\). The same argument applies to the equation of \(\dot{\lambda}_t\) after having replaced \(\lambda_t\) with \(i_t = \frac{\lambda_t e^{-\rho t}}{i_T (1 - i_T)}\), which implies that \(\lambda_t > 0\) for all \(t \in [0, T]\). Then \(u_t > 0\) for all \(t \in [0, T]\). To prove that \(i_t < 1\), let us suppose that \(i_t\) is tending to 1 from below. Then there exists some \(t^*\) such that \(i_{t^*} < 1\) and it is sufficiently near to 1 such that the following inequality is also satisfied:
\[
\frac{\delta}{1 - i_{t^*}} > \alpha - \delta - \mu u_{t^*}
\]

Then \(i_{t^*}\) is given by:
\[
i_{t^*} = ((\alpha - \delta \omega \tau - \mu u_{t^*}) (1 - i_{t^*}) - \delta) i_{t^*} < 0
\]
which implies that \(i_{t^*}\) cannot growth beyond \(i_{t^*}\) and this implies the thesis.

In order to demonstrate that, under the conditions stated in the proposition, \(i_t < 0\) it is enough to prove that:
\[
\dot{i}_t = (\alpha - \delta \omega \tau - \mu u_t) (1 - i_t) i_t - \delta i_t < 0
\]
Because \(i_t > 0\), this implies:
\[
\alpha - \delta \omega \tau - \mu u_t < \frac{\delta}{1 - i_t}
\]
If \(\alpha - \delta \omega \tau - \mu u_t \leq 0\), that is:
\[
u_t \geq \frac{\alpha - \delta \omega \tau}{\mu}
\]
then the above inequality is satisfied for any \(i_t\). Otherwise, using the fact that \(u_t > 0\), a sufficient condition that implies the above inequality is that \(\alpha - \delta \omega \tau < \frac{\delta}{1 - i_t}\), which is equivalent to:
\[
1 - i_t < \frac{\delta}{\alpha - \delta \omega \tau}
\]
and this implies the thesis. Moreover, note that because \(\theta = \alpha - \delta - \delta \omega \tau \geq 0\), then \(\alpha - \delta \omega \tau \geq 0\), which implies that \(1 - \frac{\delta}{\alpha - \delta \omega \tau} < 1\).

Appendix B. Numerical approach and algorithm

The first order optimality conditions for problems (27)–(29) give rise to a system of forward–backward ordinary differential equations in the state and costate variables, with the addition of an algebraic equation describing the maximum principle given by the third equation in (32). Specifically, the state variable has an initial condition while the costate variable has a final condition, and one of most widely used algorithm to deal with this forward–backward setting is the so-called sweep algorithm. A detailed presentation and implementation of the sweep algorithm for solving forward–backward optimal control problems over a finite time horizon has been presented in McAsey et al. (2012). By building on their approach, we implement the forward–backward sweep method for our system of first order optimality conditions as follows:

1. Focusing of the first equation of (32), we start by adopting an initial guess \(\lambda^0 = \lambda_T^0\).
Fig. 7. Comparison between the analytical (top panels) and the numerical (bottom panels) solutions in the early epidemic setting in the case of the seasonal flu.

2. Iteration for \( j \geq 0 \): by employing the spectral method, we solve:

\[
\frac{d\lambda_{i+1}}{dt} = (\alpha - \delta \omega_t)(1 - \lambda_{i+1}^{j+1}) - \delta \lambda_{i+1}^{j+1} - \mu \lambda_{i+1}^{j+1} e^{-\rho t}
\]

with the initial condition:

\[
i_{0}^{j+1} = i_0
\]

We reverse the second equation of (32) in time, via the change of variable \( \tilde{\tau} = T - t \), turning the problem into a forward problem, with initial conditions given by the fourth equation in (32). Notably the initial condition in the time-reversed equation depends on \( T \). Then, we solve:

\[
\frac{d\lambda_{i+1}^{j+1}}{d\tilde{\tau}} = -\lambda_{i+1}^{j+1}(\alpha - \delta \omega_{\tilde{\tau}})(1 - 2\lambda_{i+1}^{j+1}) - \lambda_{i+1}^{j+1} e^{-\rho \tilde{\tau}} + \mu \lambda_{i+1}^{j+1}
\]

with initial condition in \( \tilde{\tau} \) given by:

\[
\lambda_{0}^{j+1} = \frac{\phi}{T} e^{-\rho T}
\]

Finally we check for convergence by computing the difference between the values of \( \lambda_{i} \) and \( \lambda_{i} \) in two subsequent iterations (i.e. \( j + 1 \) and \( j \)). If the \( L^2 \)-norm of the difference is negligibly small, we display the current function as solution, otherwise we continue iterating.

3. Once we get a satisfactory numerical approximation of \( \lambda_{i} \) and \( \lambda_{i} \), and hence of \( u_{i} \), we evaluate the cost function (27) for different values of \( T \). We then select the cost-function minimizing value of \( T \), as shown in our figures.

Fig. 7 validates our numerical algorithm in the early epidemic case by comparing the optimal share of infectives (left panels) and social distancing intensity (right panels) derived from the closed-form expressions given by Theorem 1 (top panels) and from our numerical approach (bottom panels). The employed parameter values are exactly the same and related to our seasonal flu calibration case with \( \phi = 1 \), ensuring thus that the parameter conditions in Proposition 1 are met. We can observe that the two solutions are almost identical, confirming thus that our numerical approach works well by replicating precisely the analytical solution outlined in Theorem 1. Therefore, we can claim that even in the absence of closed-form solutions, as in the case of the advanced epidemic setting, our numerical approach may provide us with reliable results, and it can thus be used as a benchmark to analyze the implications of different parameters on the optimal intensity and duration of the social distancing policy.

References

Acemoglu, D., Chernozhukov, V., Werning, I., Whinston, M.D., 2020. A multi-risk SIR model with optimally targeted lockdown. In: NBER Working Paper 27102.

Acemoglu, D., Johnson, S., 2007. Disease and development: the effect of life expectancy on economic growth. J. Polit. Econ. 115, 925–985.

Adda, J., 2016. Economic activity and the spread of viral diseases: evidence from high frequency data. Q. J. Econ. 131, 891–941.

Alvarez, F.E., Argente, D., Lippi, F., 2020. A simple planning problem for COVID-19 lockdown. Am. Econ. Rev.: Insights (forthcoming).

Anderson, S.T., Laxminarayan, R., Salant, S.W., 2010. Diversify or focus? Spending to combat infectious diseases when budgets are tight. J. Health Econ. 31, 658–675.

Atkeson, A., 2020. What will be the economic impact of COVID-19 in the US? Rough estimates of disease scenarios. In: NBER Working Paper 26867.

Bailey, N.T., 1975. The Mathematical Theory of Infectious Diseases. Hafner Press.

Barro, R.J., Sala-i Martin, X., 2004. Economic Growth. MIT Press, Cambridge, Massachusetts.

Bleakley, H., 2007. Disease and development: evidence from hookworm eradication in the American South. Q. J. Econ. 122, 73–117.

Bosi, S., Camacho, C., Desmarchelier, D., 2020. Optimal Lockdown in Altruistic Economies. halshs-02652165.

Boucknine, R., Desbordes, R., Latzer, H., 2009. How do epidemics induce behavioral changes? J. Econ. Growth 14, 233–264.

Buonanno, P., Galletta, S., Puca, M., 2020. Estimating the Severity of COVID-19: Evidence from the Italian Epicenter. ETH Center for Law & Economics WP 03/2020.

Cervellati, M., Sunde, U., Valmoni, S., 2017. Pathogens, weather shocks and civil conflict. Econom. J. 127, 2581–2616.

Chakraborty, S., Papageorgiou, C., Pérez Sebastián, F., 2010. Diseases, infection dynamics and development. J. Monetary Econ. 57, 859–872.
Cheng, C., Barceló, J., Hartnett, A.S., Kubinec, R., Messerschmidt, L., 2020. COVID-19 government response event dataset (CoronaNet v1.0). Nat. Hum. Behav. 4, 756–768.

Chowell, G., Satenspiel, L., Bansal, S., Viboud, C., 2016. Mathematical models to characterize early epidemic growth: a review. Phys. Life Rev. 18, 66–97.

Coburn, B.J., Wagner, B.G., Blower, S., 2009. Modeling influenza epidemics and pandemics: insights into the future of swine flu (H1N1). BMC Med. 7, article 30.

Di Nicola, F., Boschi, M., Mongelli, G., 2017. Effective marginal and average tax rates in the 2017 Italian tax-benefit system. Econ. Pubbl. 2017, 533–534.

Eichenbaum, M., Rebelo, S., Trabandt, M., 2020. The macroeconomics of epidemics. In: NBER Working Paper 26882.

European Commission, 2020. Spring 2020 economic forecast: a deep and uneven recession, an uncertain recovery. available at: https://ec.europa.eu/commission/presscorner/detail/en/ip_20_799.

Gersovitz, M., Hammer, J.S., 2003. Infectious diseases, public policy and the marriage of economics and epidemiology. World Bank Res. Obs. 18, 129–157.

Gersovitz, M., Hammer, J.S., 2004. The economical control of infectious diseases. Econom. J. 114, 1–27.

Goenka, A., Liu, L., 2012. Infectious diseases and endogenous fluctuations. Econom. Theory 50, 125–149.

Goenka, A., Liu, L., 2020. Infectious diseases, human capital and economic growth. Econom. Theory 70, 1–47.

Goldman, S.M., Lightwood, J., 2002. Cost optimization in the SIS model of infectious disease with treatment. Top. Econ. Anal. Policy 2, Article 4.

Hethcote, H.W., 2000. The mathematics of infectious diseases. SIAM Rev. 42, 599–653.

Hethcote, H.W., 2008. The basic epidemiology models: models, expressions for $R_0$, parameter estimation, and applications. In: Ma, S., Xia, Y. (Eds.), Mathematical Understanding of Infectious Disease Dynamics. Institute for Mathematical Sciences at the National University of Singapore, Singapore, pp. 1–61.

Jung, E., Lenhart, S., Feng, Z., 2002. Optimal control of treatments in a two-strain tuberculosis model. Discrete Contin. Dyn. Syst. B 2, 473–482.

Kermack, W.O., McKendrick, A.G., 1927. A contribution to the mathematical theory of epidemics. Proc. R. Soc. A 115, 700–721.

Kling, M.J., Milionis, P., 2020. The international epidemiological transition and the education gender gap. J. Econ. Growth 25, 37–86.

La Torre, D., Liuzzi, D., Marsiglio, S., 2017. Pollution control under uncertainty and sustainability concern. Environ. Resour. Econ. 67, 885–903.

La Torre, D., Malik, T., Marsiglio, S., 2020. Optimal control of prevention and treatment in a basic macroeconomic-epidemiological model. Math. Social Sci. 100, 100–108.

Lazzerini, M., Putoto, G., 2020. COVID-19 in Italy: momentous decisions and many uncertainties. Lancet Glob. Health 8, e641–e642.

Lopez, A.D., Mathers, C.D., Ezzati, M., Jamison, D.T., Murray, C.J.L., 2006. Global Burden of Disease and Risk Factors. Oxford University Press, New York.

Ma, J., 2020. Estimating epidemic exponential growth rate and basic reproduction number. Infect. Dis. Model. 5, 129–141.

Maharaj, S., Kleczkowski, A., 2012. Controlling epidemic spread by social distancing: do it well or not at all. BMC Publ. Health 12 (679).

Manski, C.F., Molinari, F., 2020. Estimating the COVID-19 infection rate: anatomy of an inference problem. In: NBER Working Paper 27023.

McAsey, M., Mou, L., Han, W., 2012. Convergence of the forward-backward sweep method in optimal control. Comput. Optim. Appl. 53, 207–226.

Ng, W.L., 2020. To lockdown? When to peak? Will there be an end? A macroeconomic analysis on COVID-19 epidemic in the United States. J. Macroecon. 65, 102330.

Philipson, T., 2000. Economic epidemiology and infectious disease. In: Cuyler, A.J., Newhouse, J.P. (Eds.), Handbook of Health Economics, Vol. 1B. North Holland, Amsterdam, pp. 1761–1799.

Remuzzi, A., Remuzzi, G., 2020. COVID-19 in Italy: what next? Lancet 395, 1225–1228.

Rotman, H.A., Byrareddy, S.N., 2020. The epidemiology and pathogenesis of coronavirus disease (COVID-19) outbreak. J. Autoimmun. 102433.

Velenyi, E.V., Smitz, M.F., 2014. Cyclical patterns in government health expenditures between 1995 and 2010: are countries graduating from the procyclical trap or falling back? In: Health, Nutrition, and Population (HNP) Discussion Paper. World Bank Group, Washington, DC.

World Health Organization, 2000. World health statistics 2009. available at: http://www.who.int/gho/publications/world_health_statistics/EN_WHS09_Full.pdf.

World Health Organization, 2020a. Report of the WHO - China joint mission on coronavirus disease 2019 (COVID-19). available at: https://www.who.int/docs/default-source/coronaviruse/who-china-joint-mission-on-covid-19-final-report.pdf.

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.