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We analyze the spread of an infectious disease in a population when individuals strategically choose how much time to interact with others. Individuals are either of the severe type or of the asymptomatic type. Only severe types have symptoms when they are infected, and the asymptomatic types can be contagious without knowing it. In the absence of any symptoms, individuals do not know their type and continuously trade off the costs and benefits of self-isolation on the basis of their belief of being the severe type. We show that all equilibria of the game involve social interaction, and we characterize the unique equilibrium in which individuals partially self-isolate at each date. We calibrate our model to the COVID-19 pandemic and simulate the dynamics of the epidemic to illustrate the impact of some public policies.

1. Introduction

The herd immunity level of an infectious disease is defined as the fraction of the population that must become immune for the spread of the disease to decline and stop. Under the simplest model, it is equal to \((R_0 - 1)/R_0\), where \(R_0\) is the basic reproduction number of the disease, which is often estimated to be approximately 60% for COVID-19. The figure of 60% assumes that the population is homogeneous and passive, while it is well-documented that the herd immunity level varies between populations consisting of people with different behaviors. The object of the growing epi-economic literature is to analyze the two-sided interactions between the dynamics of epidemics and individual behaviors.

One of the many features of COVID-19 is the wide variety of responses to the infection in the population, with some individuals completely asymptomatic, and others developing fatal forms within a few days. As with symptomatic individuals, asymptomatic patients are a source of the spread of infection. Before being infected, there is no way of knowing whether one is of the asymptomatic type. Therefore, individuals form beliefs about their type, which they continuously update on the basis of how much they might have been exposed to the virus. At the same time, they decide how much to expose themselves to the virus in function of their updated belief. For instance, an individual who interacts with many people without developing COVID-19 symptoms becomes more optimistic about being the asymptomatic type. As a result, she may be tempted to meet even more people and forget about social distancing. The contribution of this paper to the epi-economic literature is to introduce learning into an epidemiological model, and to analyze the dynamics of an epidemic when individuals trade off the costs and benefits of self-isolation on the basis of their subjective beliefs.

To analyze this question, we amend the classical Susceptible–Infected–Recovered (SIR hereafter) model of Kermack and McKendrick (1927) in two ways. In its classical version, the SIR model divides a homogeneous population into three groups: susceptible, infected and recovered, with individuals transiting from one group to another one at given, exogenous rates that depend on the size of each group. We depart from the homogeneity assumption by considering two possible types of individuals in the population: severe and asymptomatic. Individuals of the severe type...
experience the symptoms of the disease immediately after being infected. In contrast, individuals of the asymptomatic type do not have symptoms. Individuals with symptoms immediately self-isolate, but asymptomatic individuals can be contagious without knowing it. Therefore, the disease is spread in the population by asymptomatic types. Moreover, we assume that individuals without symptoms can influence the transition rate from susceptible to infected by strategically reducing the fraction of time they spend outside. Staying home prevents one from being infected, but comes at a cost (boredom, opportunity cost of not working, or of working in poorer conditions, lack of physical activity, etc.). Being infected is also costly for individuals of the severe type. Therefore, individuals continuously tradeoff the cost of self-isolation and the expected benefit of not having the symptoms on the basis of their belief of being the severe type. Finally, we assume that a vaccine will arrive at known time $T$.

Why would individuals voluntary engage in costly confinement? More confinement decreases the probability of suffering from the symptoms and increases the probability of getting the continuation payoff of a healthy individual. Therefore, self-isolation may be worth the cost at some dates, but may not at some other dates. We prove that individuals never completely self-isolate in equilibrium: if the rest of the population stays at home, the chances of being infected are 0, thus each individual can spare the confinement cost without risking infection. Intuitively, when self-isolating is costlier than having symptoms, individuals do not self-isolate at all in equilibrium, and the dynamics of the epidemic are the same as in the SIR model. When the confinement cost is relatively small, however, the equilibrium strategy is non-stationary and may be interior, i.e. such that individuals partially self-isolate at each date. We prove that there can be only one interior equilibrium, and that it is symmetric.

We calibrate our model to the COVID-19 epidemic in order to illustrate the impact of self-isolation behaviors on the dynamics of the epidemic, and to highlight the policy lessons that can be drawn from our findings. We focus on the cases where individuals partially confine at each date at equilibrium. We simulate the dynamics of the epidemic in the unique interior equilibrium of the game. We find that the population reacts to the epidemic announcement by self-isolating drastically, which results in a drop in the percentage of infected. Then individuals gradually increase the time they spend outside, maintaining the effective reproduction number below the value that accelerates the epidemic. As a result, the epidemic curve is decreasing between the time of announcement of the epidemic and the arrival of the vaccine, contrary to the well-known bell-shaped curve of the SIR model. We find that a later announcement of the epidemic increases the number of deaths, in line with the results of Silverio et al. (2020), who find a positive correlation between the number of cases before lockdown and the mortality rate in Italy. We also analyze the impact of policies aiming at mitigating the transmission of the virus such as mask distributions, messaging about hygiene measures, etc. We find that individuals compensate the decrease in the risk of infection by reducing social distances, but not to the point of accelerating the epidemic. Overall, we find that these policies reduce the number of deaths. We also show that a more performing health system results of less self-isolation but overall decreases the number of deaths. Finally, policies subsidizing self-isolation flatten the economic curve, but we find no substantial difference when self-isolation is encouraged at the beginning or at the end of the epidemic.

Related literature Many papers have documented that individuals adapt their behavior when facing a risk of infection. For instance, Philipson and Posner (1993) show that the demand for measles, mumps and rubella vaccines increases when there is a large increase in measles cases in a community, and Ahituv et al. (1996) show that the demand for condoms increases in regions where HIV is prevalent. Some papers4 also prove that individual behaviors impact the spread of infectious diseases. In the case of COVID-19, Cowling et al. (2020) show that border restrictions and changes in individual behavior are partly responsible for reduced transmission in Hong Kong in February 2020.

In the theoretical literature, some models analyze the effect of social distance in SIR or SIS epidemiological models, either in a social optimum approach (e.g. Sethi (1978) and Chen et al. (2011)) or also with strategic individual decisions (Reluga, 2010; Chen, 2012; Fenichel et al., 2011; Fenichel, 2013; Toxvaerd, 2019; Rowthorn and Toxvaerd, 2020). This literature has grown considerably with the COVID-19 crisis. A strand of papers analyze the optimal control of the epidemics in the SIR model, either in the case of a homogeneous population (Kruse and Strack, 2020; Eichenbaum et al., 2020; Alvarez et al., 2020; Jones et al., 2020; Glover et al., 2020), or when older people are more likely to die from the disease (Acemoglu et al., 2020; Favero et al., 2020; Rampini, 2020; Bairoliya and Imrohoroglu, 2020). The problem of individuals who tradeoff the costs and benefits of self-isolation in the SIR model has been studied notably by Toxvaerd (2020), Farboodi et al. (2020) and Brotherhood et al. (2020). In an infinite horizon model, Toxvaerd (2020) characterizes the exposure level at the symmetric equilibrium and shows that self-isolation flattens the epidemic curve. Farboodi et al. (2020) prove that individuals do not self-isolate enough with respect to what would be socially optimal, and Brotherhood et al. (2020) analyze the effect of testing and age-specific policies in a heterogeneous population with observable characteristics. Our model is the only one in which the tradeoff faced by individuals also depends on their subjective beliefs of being the asymptomatic type.

The remainder of this paper is organized as follows. Section 2 sets up the model. In Section 3, we solve the best-response problem of a player, analyze some properties of the equilibrium and characterize equilibria in which there is no confinement at all, or always partial confinement. In Section 4, we calibrate our model to fit the COVID-19 pandemic, we simulate the dynamics of the epidemic in equilibrium and provide some policy analysis. Section 5 concludes and technical proofs are gathered in Appendix.

2. An epidemiological model with voluntary confinement

The population Time $t \in [0, +\infty)$ is continuous and discounted at a common rate $r > 0$. There is a rampant disease in the population, against which a vaccine will arrive at time $T > 0$. The population is a continuum of individuals who must continuously decide what fraction of their time they spend outside. An individual who stays home is protected from infection, while an individual who goes out may be infected by other individuals, with a probability that will be described later. For simplicity, we assume that an individual is contagious as long as she is infected. People know whether they have been infected only if they experience the symptoms of the disease. There are two types of individuals in the population. Individuals of type $\theta_s$, the severe type, who experience the symptoms of the disease immediately after being infected. In contrast, individuals of type $\theta_a$, the asymptomatic type, who do not have symptoms, thus never realize that they have been infected. The proportion $\mu \in (0, 1)$ of asymptomatic types in the population is common knowledge, but individuals do not know their own type, unless they are of type $\theta_s$ and become infected.

4 For instance, Cowling et al. (2009) and Aiello et al. (2010) show that masks and hand washing can reduce household transmission of respiratory infections in small areas.
We assume that an individual who gets symptoms self-isolates immediately until the end of the symptoms, either to protect others, or simply because she is too sick to go out. Therefore, a strategy for player \(i\) is a measurable function \(k_i: \mathbb{R}_+ \to [0, 1]\), with the interpretation that \(k_i(t)\) is the proportion of time spent outside at time \(t\), absent symptoms by time \(t\).

**Evolution of the epidemic** To model the spread of the disease, we use the SIR model from Kermack and McKendrick (1927). At each time \(t\), the population is divided into three groups: susceptible \(S(t)\), infected \(I(t)\) and recovered \(R(t)\), i.e., those who died from the disease or recovered and are now immune to it.

Accordingly, \(S(t)\) is the fraction of the population that is healthy but susceptible to be infected at time \(t\), \(I(t)\) the fraction of the population that is infected at time \(t\), and \(R(t) = 1 - S(t) - I(t)\) the fraction of the population that has died or recovered from the disease at \(t\).

The disease is transmitted to a susceptible individual through contact with an infected individual at rate \(\beta \in (0, 1)\), which measures the contagiousness of the disease. Therefore, the mass of individuals who become infected between \(t\) and \(t+\Delta t\) depends on \(\beta\), but also on the size of groups \(S(t)\) and \(I(t)\) and on the behavior of the population in each. Given a strategy profile \(k := (k_j)\), this mass equals \(\beta \times \int_{S(t)} k_j(t) \, dt \times \int_{I(t)} k_j(t) \, dt\), thus the group of susceptible evolves according to the dynamics:

\[
\frac{dS(t)}{dt} = -\beta k_i(t)S(t)i(t), \quad \text{with} \quad S(0) = s_0 \in (0, 1),
\]

where \(\bar{k}(t) = \frac{1}{r} \int_{S(t)} k_j(t) \, dt\) and \(\bar{k}(t) = \frac{1}{r} \int_{I(t)} k_j(t) \, dt\). The average fraction of time spent outside at \(t\) by infected and susceptible individuals, respectively. Infected recover from the disease at rate \(\gamma \in (0, 1)\). We assume that asymptomatic individuals do not die from the disease, while individuals of the severe type die at rate \(\nu\), with \((\gamma + \nu) \in (0, 1)\). As the fraction of infected is also increased by \(-\bar{i}(t)\), the group of infected evolves according to the following dynamics:

\[
\frac{dI(t)}{dt} = \beta \bar{k}(t)i(t)\bar{i}(t) - (\gamma + (1 + \mu)\nu)i(t), \quad \text{with} \quad i(0) = i_0 = 1 - s_0.
\]

**Evolution of subjective beliefs** At time \(t\), individual \(i\) holds a subjective belief \(p_i(t)\) of being type \(\theta_i\), with a common prior belief \(p_i(0) = 1 - \mu\) for all individuals. In this model, no news is good news: the subjective belief of being the severe type decreases as time passes without the arrival of symptoms, and jumps to 1 the first time the symptoms occur. Let us now describe the law of motion of \(p_i(t)\) of a susceptible individual.

A susceptible individual \(i\) develops symptoms in \([t, t+\Delta t)\) with probability 0 when she is of type \(\theta_i\); when she is of type \(\theta_i\), she develops symptoms if she meets and is infected by some individual in \([t, t+\Delta t)\), which occurs with instantaneous probability \(k_i(t) \times \beta \bar{k}(t)i(t)\, dt\). By Bayes’ rule, the law of motion of the subjective belief of individual \(i\) is thus:

\[
p_i(t) = \frac{p_i(t)(1 - p_i(t))k_i(t)\bar{k}(t)i(t)}{p_i(t)(1 - p_i(t))k_i(t)\bar{k}(t)i(t) + (1 - p_i(t))}, \quad \text{with} \quad p_i(0) = 1 - \mu.
\]

**Payoffs** Staying home prevents one from being infected, but comes at a cost (boredom, opportunity cost of not working or working in poorer conditions, lack of physical activity, etc.). Being infected is also costly for individuals of the severe type because they suffer from the symptoms, and, in the worst case, because they die from the disease. Therefore, at each time \(t\), individuals tradeoff the cost of self-isolating and the expected benefit of not having the symptoms. We denote by \(c_i\) the flow cost per unit of time spent at home, by \(c_i\) the flow cost of having symptoms and by \(c_D\) the flow cost of being dead.

Fix some strategy profile \(k\) and let us describe the expected payoff to individual \(i\) at time \(t \leq T\) when she plays the same strategy \(k_i\), denoted by \(v_i(t; k_i)\). Uncertainty is solved for individual \(i\) the first time she has symptoms. In that event, she knows that she is the severe type, thus that she will stay at home until she recovers or passes away, thereby incurring a total cost of \(\int_{0}^{t} e^{-\tau} c_i k_i(t) \, dt\), with \(r_0\) and \(r_0\) standing for the random times of healing and death, respectively. If she recovers (i.e., if \(t_H < t_0\)), she becomes immune to the disease, plays \(k(t) = 1\) forever after, thus obtains the continuation payoff 0. If she dies (i.e., if \(t_0 < t_H\)), she incurs the flow cost \(c_D\) forever after, thus obtains the continuation payoff \(-c_D/r\). Therefore, the expected continuation payoff to individual \(i\) the first time she has symptoms is:

\[
v_i = -E \left[ \int_{0}^{\min(t_H, T)} e^{-\tau} (c_i + r_0) \, dt + \frac{c_D}{r} e^{-\tau} 0_{1 < t_H} \right]
\]

Conditionally on having no symptoms before \(s \in [t, T]\), the instantaneous payoff to player \(i\) at time \(s\) is \(v_i\) if she has symptoms, which occurs with subjective probability \(p_i(s) k_i(s) \beta \bar{k}(s)i(s)\), minus the cost \(c_i\) scaled with the proportion of time spent in isolation, \(1 - k_i(s)\). At time \(t\), the subjective probability of not having any symptoms before \(s = 1 - p_i(t) + p_i(t)e^{-\int_{t}^{s}(s) \beta \bar{k}(s)i(s)\, ds}\), which reduces to \(e^{-\int_{t}^{s}(s) \beta \bar{k}(s)i(s)\, ds}\) after standard simplifications.

Finally, individual \(i\) is vaccinated at time \(T\), thus plays \(k_i(t) = 1\) for every \(t \geq T\) and obtains the continuation payoff \(v_i(T) = 0\). Therefore,

\[
v_i(t; k_i) = \int_{t}^{T} e^{-\tau} e^{-\int_{t}^{s}(s) \beta \bar{k}(s)i(s)\, ds} p_i(s) k_i(s) \beta \bar{k}(s)i(s)\nu_i(s)
\]

where functions \(s(\cdot), i(\cdot)\) and \(p_i(\cdot)\) are defined by (1), (2) and (3).

**3. Equilibrium analysis**

Fix a strategy profile \(k\) and an individual \(i\). The best-response problem faced by \(i\) is the optimal control problem:

\[
\max_{k_i \in K} \quad v_i(0; k_i)
\]

such that \(p_i(t) = -p_i(t)(1 - p_i(t))k_i(t)\beta \bar{k}(t)i(t)\) \(\forall t\) and \(p_i(0) = 1 - \mu\).
which we solve in the Appendix using Pontryagin’s principle. Here, we explain the intuition of the solution with a heuristic dynamic programming argument. What is the tradeoff for player \( i \) between self-isolating “today” and self-isolating “tomorrow” for player \( t \)? The best response at time \( t \) maximizes the sum of her current expected payoff and of her discounted continuation payoff, should no symptoms occur in the interval \([t, t+dt)\). Therefore, her best-response payoff at time \( t \) satisfies the Bellman equation:

\[
V_i(t) = \max_{k_i(t) \in [0, 1]} \left\{ (1 - k_i(t))c_H + p_i(t)k_i(t)\dot{\bar{k}}_i(t)(V_i(t) - v_i)dt + \int_0^{t} (1 - p_i(t)\bar{k}_i(t))\dot{c}_H dt \right\}
\]

Using \((1 - rd_t)\) as an approximation for \(e^{-rd_t}\) and eliminating terms to the order \((dt)^2\), \(V_i(t+dt)\) is approximated by \(V_i(t)\) and the latter equation rewrites:

\[
V_i(t) = V_i(t + dt)(1 - rd_t) - c_H dt + \max_{k_i(t) \in [0, 1]} \left\{ k_i(t)(c_H - p_i(t)\bar{k}_i(t)(V_i(t) - v_i))dt \right\}.
\]  

(6)

It appears that the best response of player \( i \) at time \( t \) depends on the sign of the expression \( c_H - p_i(t)\bar{k}_i(t)(V_i(t) - v_i) \). To interpret this, note that two things can happen for individual \( i \) at time \( t \): either she develops symptoms, thus obtains the payoff \( v_i \) “today”, or she does not, thus obtains “tomorrow” the continuation payoff \( V_i(t + dt)\), whose value at time \( t \) can be approximated by \( V_i(t)\). More self-isolation thus affects the payoff in three ways: it decreases the probability of getting \( v_i \) at rate \( p_i(t)\bar{k}_i(t)(i(t)) \), increases the probability of getting \( V_i(t)\) at rate \( p_i(t)\bar{k}_i(t)(i(t)) \) and increases the incurred cost at rate \( c_H\). This is why \( p_i(t)\bar{k}_i(t)(i(t))(V_i(t) - v_i) \) can be interpreted as the marginal expected benefit of confinement for player \( i \) at time \( t \) and \( c_H \) as the marginal cost of confinement; accordingly, player \( i \)’s best response at time \( t \) depends on whether the marginal cost of confinement is larger or smaller than the marginal expected benefit. This is formally stated in the next proposition.

**Proposition 1 (Best Response).** Given a strategy profile \( \mathbf{k} \), the best-response problem of player \( i \) admits a solution \( k_i^* \), which is characterized by the pair of \( C^1 \) functions \( V_i^* : \mathbb{R}_+ \to \mathbb{R} \) and \( p_i^* : \mathbb{R}_+ \to [0, 1] \) such that, for all \( t \),

\[
k_i^*(t) = \begin{cases} 
1 & \text{if } p_i^*(t)\dot{\bar{k}}_i(t)(i(t)(V_i^*(t) - v_i)) - c_H < 0, \\
0 & \text{if } p_i^*(t)\dot{\bar{k}}_i(t)(i(t)(V_i^*(t) - v_i)) - c_H > 0. 
\end{cases}
\]

with

\[
V_i^*(t) - rV_i^*(t) = c_H + k_i^*(t)p_i^*(t)\dot{\bar{k}}_i(t)(i(t)(V_i^*(t) - v_i)) - c_H, \\
V_i^*(T) = 0, \\
p_i^*(T) = p_i^*(t)\hat{\bar{k}}_i(0) = 1 - \mu (\text{Belief dynamics}).
\]

(7)

**Proof.** See Appendix. □

An immediate corollary of Proposition 1 is that all equilibria feature social interaction, in the sense that, at every time, there is a mass of individuals who do not self-isolate. The reason is simple: if the rest of the population stays at home, each individual can spare the confinement cost \( c_H \) by going out without risking infection.

**Proposition 2.** Let \( (k_i^*) \) be an equilibrium. At every time \( t \), there is a non-empty set of individuals such that \( k_i^*(t) > 0 \) for every \( i \) in this set.

**Proof.** Fix time \( t \) and suppose that \( k_i^*(t) = 0 \) for all \( i \), i.e., \( \hat{k}_i(t) = 0 \). Yet, by condition (7) in Proposition 1, the best response of each individual \( i \) to \( k_i(t) = 0 \) is to play \( k_i(t) = 1 \). This contradicts \( k_i(t) = 0 \). □

Because the expected marginal benefit of confinement depends on \( t \) in expression (7), self-isolation may be worth the confinement cost at some dates, but may not at some other dates. Intuitively, when \( c_H \) is relatively large with respect to \( v_i \), it is a dominant strategy for individuals to go out at every date.

**Proposition 3 (The No-Confinement Equilibrium).** If \( (1 - \mu)\beta\mu v_i + c_H > 0 \), the game admits a unique equilibrium, in which all individuals play \( k(t) = 1 \) for every \( t \in [0, T] \). In this equilibrium, the players’ payoff at time \( t \) is

\[
V(t) = \frac{v_i(1 - \mu)\beta\mu}{T} e^{-\beta t(1-\mu)}\left(1 - e^{-\beta t(1-\mu)}\right) \int_0^T \left(1 - s(t)\right)e^{-\beta s(t)u}du - \mu(1 - \mu)e^{-\beta \mu u}du,
\]

where \( i(t) \) is the unique solution of the system

\[
\begin{cases} 
i(0) = 1 - s(0) = i_0 \text{ and } \forall t \in [0, T], \\
i(t) = \beta s(t)i(t) - (\gamma + (1 - \mu)v)\dot{i}(t), \\
\dot{s}(t) = -\beta s(t)i(t).
\end{cases}
\]

**Proof.** See Appendix. □

When \( c_H \) is so large that nobody self-isolates, the epidemic ends quickly but results in a large number of deaths. As a vaccine will arrive at time \( T \), the government may want to implement policies to reduce the cost of confinement, in order to create the conditions under which individuals might consider self-isolation. We thus investigate the existence of interior equilibria, in which individuals partially self-isolate at every date. We prove that there can exist only one interior equilibrium, and that it is symmetric.

**Proposition 4 (The Interior Equilibrium).** Let \( \hat{k} \) be the strategy defined by the system of equations for every \( t \in [0, T] \):

\[
\begin{align*}
\dot{k}_i(t) &= \frac{c_H}{\mu}(1 - e^{-\beta(1-\mu)} - v_i) \\
\dot{p}_i(t) &= -p_i(t)(1 - p_i(t))\beta\mu(i(t)^2) \\
\dot{s}(t) &= -\beta s(t)i(t)^2, \\
\dot{i}(t) &= \beta s(t)i(t)^2 - (\gamma + (1 - \mu)v)i(t), \\
p(0) &= 1 - \mu \text{ and } i(0) = 1 - s(0) = i_0.
\end{align*}
\]

If \( \hat{k}(t) \) is the unique equilibrium, in which all individuals play \( k \). In this equilibrium, the players’ payoff at time \( t \) is

\[
V(t) = -\frac{c_H}{r} \left(1 - e^{-\beta(1-\mu)}\right).
\]

**Proof.** See Appendix. □

4. Illustration

The purpose of this section is to illustrate the impact of self-isolation behaviors on the dynamics of the epidemic, and to highlight the policy lessons that can be drawn from our findings. To do so, we simulate the dynamics of the epidemic in the interior equilibrium described in Proposition 4, and compare it with the dynamics of the standard SIR model (referred to as the SIR model in the rest of the section), i.e., the model described by Eqs. (1) and (2) with \( \hat{k}(t) = k(t) = 1 \). We chose the behavioral parameters \( c_i, c_H \) and \( c_D \) arbitrarily in such a way that the interior equilibrium exists, and we calibrate the epidemiological parameters \( \beta, \gamma, \mu \) and \( v \) to the COVID-19 pandemic.

Throughout our simulations, we assume that individuals are not aware of the epidemic until some time \( \tau \in (0, T) \), which can be interpreted as the moment at which the government makes...
the epidemic common knowledge in the population via a public announcement. Before time $\tau$, individuals play $k(t) = 1$. After time $\tau$, they commonly know the epidemiological parameters as well as the current fraction of susceptible and infected, and adapt their behavior accordingly.

### 4.1. Calibration

We assume that initially $i_0 = 0.1\%$ of the population is infected, and that individuals discount time at rate $r = 0.014\%$, in line with in Fenichel et al. (2011).\footnote{Precisely, Fenichel et al. (2011) study a discrete-time model in which they set the discount rate to $\delta = 0.99986$, which corresponds to a 5% annual discount rate. The analog of $\delta$ in a continuous-time model is $r = -\ln(\delta)$, thus we set $r = -\ln(0.99986)$.} We also assume that the population learns on day $\tau = 30$ that a virus has been spreading since day 0, against which a vaccine will be available on day $T = 350$. Finally, we set the costs arbitrarily to $c_\mu = 0.0009$, $c_1 = 0.09$ and $c_\nu = 9$.

In the absence of exhaustive testing campaigns, it is very difficult to have a satisfactory estimate of the proportion of asymptomatic in the population. In a nationwide study of over 61,000 participants, Pollán et al. (2020) find that the proportion of asymptomatic individuals in the Spanish population who developed antibodies to the SARS-CoV-2 ranges from 21.9% to 35.8%. Therefore, we set $\mu$ to the middle of this interval: $\mu = 0.2885$.

To calibrate the contagion rate $\beta$ and the recovery rate $\gamma$, we use the estimates of Remuzzi and Remuzzi (2020)’s, i.e., a basic reproduction number of $R_0 = 2.76$ and an infection duration of 15 days. The infection duration directly yields: $\gamma = 1/15$. Some precautions must be taken to infer $\beta$ from $R_0$. By definition, the basic reproduction number is the average number of secondary infections produced by a typical case of an infection in a population where everyone is susceptible. It is affected by the rate of contacts in the population, the probability of infection being transmitted during contact and the duration of infectiousness. In our model, $R_0 = \beta \mu / \gamma$. Let us explain why. As infected self-isolate when they are the severe type, a randomly chosen infected individual contaminate a susceptible only if she is asymptomatic (which happens with probability $\mu$) and if the virus is transmitted during contact (which happens with probability $\beta$). As she is infectious during a period of expected length $1/\gamma$, the average number of new infections caused by an infected is $\beta \mu / \gamma$. Therefore, we set: $\beta = 2.76 \gamma / \mu$.

On March 3, 2020, the Director General of the WHO declared that approximately 3.4% of reported cases of COVID-19 died from the disease. As only patients with severe symptoms were tested at the beginning of the outbreak, we believe that the mortality rates measured in March 2020 are a valid estimate of the probability of death for an individual of the severe type infected by the disease,\footnote{In our model, an infected of the severe type dies if the event “Death” occurs for her before the event “Healing”. Therefore, the probability of death (conditional on being infected and the severe type) is $P(t_0 < t_H)$, with $t_0$ and $t_H$ denoting the random times of healing and death, respectively. Straightforwardly, $P(t_0 < t_H) = \int_0^{t_0} \int_0^{t_H} f_{t_0}(t) f_{t_H}(t) dt = v/(\gamma + v)$ since $f_{t_0}(t) = ve^{-\gamma t}$ and $f_{t_H}(t) = ve^{-\gamma t}$.} i.e., $v/(\gamma + v)$. Therefore, we set: $v/(\gamma + v) = 0.034$.

### 4.2. The dynamics of the epidemic in the interior equilibrium

We begin by analyzing the impact of strategic self-isolation on the dynamics of the epidemic, which is illustrated in Fig. 1.

Contrary to the now well-known bell-shaped curve of the SIR model, the epidemic curve (i.e., the graph of the percentage of infected plotted against time) continuously decreases on $[\tau, T]$. Therefore, the epidemic peak is reached before the population is informed about the epidemic, while in the SIR model, the fraction of infected continues to increase after time $\tau$, reaching later a higher peak. The reason is that the population reacts to the epidemic announcement by self-isolating drastically after time $\tau$, which results in a rapid decline in the percentage of infected. As the probability of being infected decreases, the marginal benefit of confinement decreases and individuals gradually increase the time they spend outside between time $\tau$ and $t = 190$ as a result. As $k(t)$ sort of stabilizes to 0.62 after $t = 190$, there is no rebound of the epidemic.

One of the parameters monitored by health authorities is the effective reproduction number, i.e., the average number of secondary cases per infectious case in a population made up of both susceptible and recovered individuals. In the SIR model, the effective reproduction number is $R_{\text{eff}}(t) = \beta \mu / \gamma$. As the fraction of susceptible continuously decreases on $[0, T]$ (see Eq. (1)), the effective reproduction number in the SIR model decreases on $[0, T]$ and stabilizes to $R_{\text{eff}}(350) = 0.23$. In the interior equilibrium, an infected of the asymptomatic type has a “probability” $\tilde{k}(t)$ of going out, and when she is out, a “probability” $\tilde{k}(t)s(t)$ of meeting a susceptible. Therefore, the effective reproduction number in the interior equilibrium equals $R_{\text{eff}}(t) = \mu \beta \tilde{k}(t)s(t)/\gamma$. This explains why, contrary to the SIR model, the effective reproduction number in equilibrium falls after the epidemic announcement, then gradually increases and stabilizes approximately\footnote{Precisely, $R_{\text{eff}}(220) = 1.00007$.} to 1 once people start deconfining, thus after $t = 220$.

Finally, one can see in Fig. 2 that the SIR model overestimates the number of deaths compared to a model with strategic self-isolation. This should not be interpreted in favor of strategies
pursuing herd immunity, however, because even the smaller number of cases predicted by our model may well overwhelm hospital capacity.

4.3. Policy analysis

Experts are still debating which public interventions should be imposed to manage the COVID-19 crisis. When it comes to public policies, not taking into account the fact that people react strategically to their environment might be counter productive. In this section, we illustrate how our results can inform some of the current debates over policies to control COVID-19.

4.3.1. The timing of announcement

We begin by analyzing the impact of the announcement time on the epidemic course. As the epidemic peak is reached in $\tau$, a later announcement mechanically increases the proportion of infected when the population learns about the epidemic. This has two opposite effects on the marginal benefit of confinement for an individual. On the positive side, it increases the risk of being infected, which gives her more incentives to self-isolate. On the negative side, it increases her confidence in being the asymptomatic type after she realizes that she has developed no symptoms during $[0, \tau]$, which gives her more incentives to go out.

These opposite effects explain the form of the graphs in Fig. 3: when the epidemic announcement is delayed, the population starts deconfining later (the risk effect) but the percentage of time spent outside stabilizes at a higher level (the confidence effect).

However, we find that a later announcement increases the number of deaths, as illustrated in Fig. 4. This is consistent with the findings of Silverio et al. (2020), who analyze the relationship between the penetration of COVID-19 at the time of lockdown and the mortality in the different Italian regions. They find a significant, positive correlation between the number of cases before lockdown and the mortality up to sixty days, and show that every day of delay in containment was associated with increased mortality.

4.3.2. Mitigating the transmission of the virus

The aim of lockdown policies is to flatten the economic curve by reducing the frequency of contacts in the population. Another way to do so is to mitigate the probability of transmission of the virus per contact, i.e., $\beta$. Governments can reduce $\beta$ by subsidizing face masks, installing hand sanitizers in public spaces, messaging about hygiene measures such as hand washing, use of disposable tissues, etc. Governments can also organize screening campaigns to detect infected but asymptomatic people. Formally, if a fraction $x$ of the population is tested for COVID-19 (and if asymptomatic people who test positive self-isolate), then only a fraction $(1-x)\mu$ of the infected people can infect susceptible individuals. In terms of the dynamics of the epidemic, this

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14 When an individual with no symptoms learns at time $\tau$ that the epidemic has been spreading since time 0, she updates her belief of being type $\delta_i$ to $p_i(\tau) = (1-\mu)/(1-\mu + \mu e^{\mu \int_0^\tau i(t)dt})$. Therefore, the larger $\tau$, the smaller $p_i(\tau)$. 

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---

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Fig. 4. Impact of $\tau$ on the cumulative number of deaths.

Fig. 5. Impact of $\beta\mu$ on the percentage of time spent outside and the subjective beliefs.

is equivalent to assuming that the marginal rate of infection decreases to $\beta(1-x)\mu$. Let us refer to the policies reducing $\beta\mu$ as mitigating policies.

In the SIR model, mitigating policies unambiguously flatten the epidemic curve. In our model, however, the effect of these policies is more subtle. First, mitigating policies have two opposite effects on the marginal benefit of confinement for an individual. On the negative side, they decrease the risk of infection per contact, which gives her less incentives to self-isolate. On the positive side, they slow down the learning process at work in the model, hence decrease her confidence in being the asymptomatic type, which gives her less incentives to go out. The risk compensating behavior can be observed in Fig. 5 for the three smallest values of $\beta\mu$. Interestingly, a decrease from $\beta\mu = 0.276$ to $\beta\mu = 0.230$ make individuals deconfining earlier (the risk effect) but the percentage of time spent outside stabilizes at a smaller level (the confidence effect).

This is in line with the empirical findings of Yan et al. (2020a), who use SafeGraph smart device location data to investigate the consequences of wearing face masks on self-isolation behavior in the American population. They find evidence that masks enable disinhibition behavior and that Americans spend 20-30 min less time at home and more time in moderate to high-risk locations following orders to wear masks.

However, we find that mitigating policies are efficient in reducing the number of deaths, as illustrated in Fig. 6.

4.3.3. Improving health system performance

The COVID-19 crisis has highlighted important differences between countries in terms of health system performance, even within the OECD group. It is reasonable to assume that the state of the health system (intensive care beds capacity, possibility of inter-hospital patient transfers or of setting up field hospitals, etc.) influences the recovery rate $\gamma$ and the mortality rate $\nu$.

We analyze the impact of the health system performance on the dynamics of the epidemic by comparing our baseline system ($\gamma = 1/15$ and $\nu = 0.00094$) with a more performing system where $\gamma = 0.07334$ and $\nu = 0.00187367$.) influences the recovery rate $\gamma$ and the mortality rate $\nu$.

Given a fixed percentage of time spent out by individuals, a better health system directly flattens the epidemic curve and decreases the number of deaths. However, with strategic self-isolation a better health system has also a perverse effect via the increase in the continuation payoff\(^1\) in case of infection. Since the expected cost of having symptoms decreases with the quality of the health system, the marginal benefit of confinement decreases and individuals have less incentives to self-isolate. One can observe this risk compensating behavior in Fig. 7: in a more performing health system, individuals deconfine earlier and the percentage of time spent outside stabilizes at a higher level.

However, we find that the performance of the health system decreases the number of deaths, as illustrated in Fig. 8.

\(^1\) The law of motion of an individual’s belief is $p(t) = -p(t)(1 - p(t))\beta\mu t\hat{k}(t)^2$, hence decreasing $\beta\mu$ decreases the growth rate of $p(t)$ in absolute value.

\(^{17}\) One can see in expression (4) that $v_1$ increases when $\gamma$ increases or when $\nu$ decreases.
4.3.4. Subsidizing self-isolation

The policy insight of Proposition 3 is that reducing the cost of confinement is also a means to be considered in controlling the spread of an infectious disease. To reduce $c_H$, governments can e.g. set up partial unemployment compensation schemes, subsidize the purchase of computer equipment, etc. The impact of a decrease in $c_H$ on the dynamics of the epidemic is immediate: it decreases the percentage of time spent outside, thus the number of deaths.

Since measures encouraging self-isolation are costly, it is conceivable that the government may not be able to cut back $c_H$ until the vaccine arrives, but only for a certain period of time, which raises the question of the optimal timing of subsidizing policies. To address this question, we simulate two different scenarios that we compare to the baseline simulation. In the first scenario (Public Policy 1), the confinement cost is reduced to $c_H = 0.000675$ only between $t = 30$ and $t = 180$. In the second scenario (Public Policy 2), the confinement cost is reduced to $c_H = 0.000675$ only between $t = 180$ and $t = 350$. As one can see in Fig. 9, Policy 1 induces more self-isolation until $t = 180$. At $t = 180$, the time individuals spend outside skyrockets then rapidly decreases and returns to the equilibrium level without public intervention.
after the implementation of Policy 2, individuals self-isolate drastically, then the time they spend outside increases gradually until it returns to the equilibrium level without public intervention.

Finally, these two policies only reduce the number of deaths at the margin (see Fig. 10), because individuals already partially self-isolate in equilibrium. This should not be interpreted as evidence of inefficiency of policies subsidizing self-isolation. On the contrary, these policies allow for a shift from equilibria in dominant strategies without self-isolation to equilibria with partial self-isolation.

5. Concluding remarks

This paper is a first attempt to analyze the spread of an infectious disease in a population when individuals strategically choose how much time to interact with others. In the absence of any symptoms, individuals are not sure whether they are susceptible or infected but asymptomatic, and they continuously tradeoff the costs and benefits of self-isolation on the basis of their belief of being the asymptomatic type. We prove that when the cost of confinement is small enough, there exists an equilibrium in which the population partially self-isolates at every date. This is consistent with the findings of Yan et al. (2020b), who use smart device location data to show that people adopted avoidance behaviors following the announcement of the pandemic.

We calibrate the parameters of our model to the COVID-19 pandemic and simulate the impact of some public interventions on the dynamics of the epidemic. Calibration is very delicate in the case of an emerging disease for which knowledge is increasing daily. In particular, the choices of the proportion of asymptomatic and the basic reproduction number are questionable. To verify the robustness of our results, we perform a sensitivity analysis in Appendix. To do so, we simulate our model with other reasonable values of the parameters and obtain qualitatively the same results. In particular, we show that the number of deaths increases when the population is informed later of the epidemic, and decreases under mitigating policies.

Two subjects have been particularly debated since the beginning of the COVID-19 crisis: the application, timing and duration of lockdowns and the use of face masks in the general population.

Most countries have implemented lockdown policies to slow down the epidemic. As lockdowns cause huge collateral damage in terms of economic activity, education and access to care, some governments were tempted by strategies pursuing herd immunity at first. The optimal timing of lockdowns is a crucial question. In a theoretical model where individuals do not chose how much time they interact with others, Kruse and Strack (2020) show that if the government has the possibility to lockdown the population during only 100 days, delaying the moment to start the lockdown might actually decrease the total number of deaths. The reason is that delaying the lockdown increases the level of herd immunity in the population, which works as a protection for those individuals who remain susceptible after the lockdown. In contrast, we find that the number of deaths increases with the announcement time. The reason is that, when individuals behave strategically, they flatten the epidemic curve by self-isolating more when...
the epidemic is too fast. Therefore, delaying the announcement time postpones the moment at which the population strategically controls the epidemic course.

At present, many governments have mandated the use of face masks in public spaces, arguing that face masks are low cost and might help prevent some transmission. At the beginning of the outbreak, however, WHO officials did not recommend mask wearing in the general population, stressing that (1) masks are commonly misused, and as a result, do not offer the intended protections, and (2) wearing a mask can provide a false sense of security, leading some to become less vigilant in more important hygiene measures, such as hand washing and self-isolation. Our results confirm that making masks mandatory leads individuals to reduce social distance, which can accelerate the epidemic. However, as individuals adapt to the level of the epidemic, this reduction is moderate, and the negative effect of a higher level of social interaction is more than offset by the positive effect on virus transmission, so that mandatory masks lead to a reduction in the number of deaths.

In our model we assume a homogeneous population, full immunity after healing and no incubation period. These are strong assumptions in the case of the COVID-19 pandemic, which could be relaxed for future research.

**Heterogeneous population.** We assume that individuals have all the same prior belief of being the severe type, thus of being asymptomatic. However, they know that, if they catch the disease, they are unclear whether individuals with co-morbidities are less likely to be asymptomatic. In the Appendix, we prove that any assumption in the case of the COVID-19 pandemic, which could be relaxed for future research.

**Waning immunity.** In our model, individuals who recover from the disease become perfectly immune to the virus. This is true for many infectious diseases, but likely not for COVID-19. Antibodies to other coronaviruses are known to wane over time (12 to 52 weeks from the onset of symptoms) and homologous re-infections have been observed (see e.g. Kellam and Barclay (2020)). SARS-CoV-2 IgM and IgG antibody levels may remain over the course of seven weeks or at least in 80% of the cases until day 49 (see Xiao et al. (2020) and Zeng et al. (2020)). Therefore, it is reasonable to assume that individuals are immune immediately after recovery, but may lose their immunity after a random period of time, probably before the vaccine arrives. One possible way to introduce waning immunity into our model would be to assume that healed individuals become susceptible again at some rate \( \eta \). This would change the dynamics of the epidemic and also the continuation payoff in case of symptoms. Like in our model, an individual would learn that she is the severe type the first time she has symptoms. The difference is that she would face a new type of uncertainty after healing, as she would not know whether she is still immune to the virus. As a result, playing \( k(t) = 1 \) forever after healing would not be a dominant strategy, and she would face a non trivial dynamic optimization problem after healing. The technical analysis of this augmented model is challenging and left for future research.

**Incubation period.** The most critical assumption of our model is the absence of incubation period, which implies that the virus is spread in the population only by asymptomatic individuals. In the case of COVID-19, the time from exposure to the development of symptoms is estimated to be 5.2 days on average (see Qun et al. (2020)), hence severe types can also spread the virus during the incubation period. A simple way to introduce an incubation period in our model would be to assume that an individual infected at time \( t \) develops symptoms only at time \( t + \Delta \), with \( \Delta \) standing for the incubation period. The dynamics of the epidemic would be the same, except for the law of motion of beliefs. Indeed, in this variant of the model the probability of developing symptoms between \( t \) and \( t + dt \) conditional on being type \( \theta \) would be the probability of having been infected in \( t - \Delta \). Therefore, the law of motion of player \( i \)'s belief would be \( \bar{p}_i(t) = -\bar{p}_i(t)\{1 - \bar{p}_i(t)\}k(t)\{t - \Delta\}k(t - \Delta)\{t - \Delta\}j(t - \Delta)j(t - \Delta)\). The best-response problem of individuals would be an optimal control problem with time lag in the control variable, which is very difficult to solve. However, we believe that the equilibria of the game would be similar, as the uncertainty that people might be infected and contagious but asymptomatic is already present in our model with the uncertainty about the type.

**Appendix**

### A.1. Proofs for Sections 2 and 3

**Lemma 1.** Let \( \tau_H \) and \( \tau_D \) be independent random variables distributed according to \( f(t) = ye^{-r + \gamma}t \) and \( f(t) = ve^{-r - \gamma}t \), respectively. The following equality holds:

\[
E \left[ \int_0^{\min(\tau_H, \tau_D)} e^{-r} (c_H + c_I)dt + \frac{C_D}{r} e^{-r\tau_D} 1_{\tau_D < \tau_H} \right] = \frac{1}{r + \gamma + \nu} (c_H + c_I + \nu \frac{C_D}{r}).
\]

**Proof.** Let \( g(\tau_H, \tau_D) := \int_0^{\min(\tau_H, \tau_D)} e^{-r} (c_H + c_I)dt + \frac{C_D}{r} e^{-r\tau_D} 1_{\tau_D < \tau_H} \).

Straightforwardly,

\[
g(\tau_H, \tau_D) = \frac{c_H + c_I}{r} + (1 - e^{-r\min(\tau_H, \tau_D)}) + \frac{C_D}{r} e^{-r\tau_D} 1_{\tau_D < \tau_H}.
\]

The random variable \( \min(\tau_H, \tau_D) \) is distributed according to \( f(t) = (\gamma + v) e^{-(\gamma + v)t} \). Therefore,

\[
E [e^{-r\min(\tau_H, \tau_D)}] = \frac{\gamma + v}{r + \gamma + v}.
\]

### End of Proof.
Moreover,
\[ E[e^{-rT}1_{T<\tau_D}] = \int_0^{\infty} \left( \int_0^T e^{-(r+\nu)v} \nu \, dt_2 \right) ye^{-rY} \, dt = \frac{c_1}{r + y + \nu}. \]
Therefore,
\[ E[g(\tau_D, \tau_0)] = (c_3 + c_1) \frac{1}{r + y + \nu} + \frac{c_1}{r + y + \nu}. \]

**Proof of Proposition 1.** The best-response problem of player \( i \) is to determine the strategy \( k_i \) that maximizes her expected discounted payoff \( u_i(0; k_i) \), the pair of functions \( s(\cdot) \) and \( \nu(\cdot) \) being fixed and defined by the dynamics:

\[
\begin{align*}
\{0\} &= 1 - i(0) = 0 \quad \text{and, } \forall \ t \in [0, T], \\
\{s(t) = -a\bar{k}_0(1 - k(t)) \} dt, \\
\nu(t) &= 0. \quad \therefore \text{Formally, it is the solution of the optimal control problem:}
\end{align*}
\]

\[
\begin{align*}
\max_{k \in K} \int_0^T e^{-rT} s_i(t) e^0 T \mu_i(1-j(t)) \nu_k(t) V_i(t) \, dt \\
\text{w.r.t.} \quad \nu_k(t) = 0. \quad \therefore \text{Hence, the maximization problem } P(k) \text{ is rewritten as:}
\end{align*}
\]

\[
\begin{align*}
P(k) \quad \max_{k \in K} \int_0^T e^{-rT} f(t, x(t), k(t)) \, dt \\
\text{w.r.t.} \quad x(t) = f(t, x(t), k(t)) \quad \text{and} \quad x(0) = 1.
\end{align*}
\]

The latter condition can be more conveniently rewritten as:

\[
(i) \quad \{k^*(t) - k(t)\} [c_4(1 - p^*(t)\beta \bar{k}_0(t)|V(t) - v_i)] \geq 0,
\]

with \( p^*(t) = 1 - \frac{m}{t+1} \). Accordingly, if \( c_4(1 - p^*(t)\beta \bar{k}_0(t)|V(t) - v_i) > 0 \), then \( k^*(t) \) must be larger than every admissible control, which is true only if \( k^*(t) = 1 \). If, on the contrary, \( c_4(1 - p^*(t)\beta \bar{k}_0(t)|V(t) - v_i) < 0 \), \( k^*(t) \) must be smaller than every admissible control, which is true only if \( k^*(t) = 0 \). Therefore, condition (ii) reduces to

\[
(ii) \quad k^*(t) = \begin{cases} 1, & \text{if } c_4(1 - p^*(t)\beta \bar{k}_0(t)|V(t) - v_i) > 0, \\
0, & \text{if } c_4(1 - p^*(t)\beta \bar{k}_0(t)|V(t) - v_i) < 0. \end{cases}
\]

Let us now prove that necessary conditions are also sufficient.

**Lemma 3 (Sufficient Conditions).** Consider a continuous, piecewise continuously differentiable function \( \nu: \mathbb{R}^+ \to \mathbb{R} \) and a pair \((x^*, k^*)\) satisfying conditions (i) and (ii). For any admissible pair \((x, k), I_0^\infty e^{-rt} f(t, x(t), k(t)) \leq I_0^\infty e^{-rt} f(t, x(t), k(t)).
\]

**Proof.** Using the change of variable \( y(t) = \int_0^T e^{-rt} f(t, x(t), k(t)) dt \), the maximization problem \( \mathcal{P}(k) \) is rewritten as:

\[
\mathcal{P}(k) \quad \max_{k \in K} \int_0^T e^{-rt} f(t, x(t), k(t)) \, dt \\
\text{w.r.t.} \quad y(t) = k(t) \beta \bar{k}_0(t) |V(t) - v_i| \quad \text{and} \quad y(0) = \int_0^T \mu(t). \]

Therefore, the Hamiltonian of the problem can be written as:

\[
H(t, y, k, V) = \mu e^{-yt} (k(t) \beta \bar{k}_0(t) |V(t) - v_i| - \mu(1 - e^{-yt}) c_4(1 - k(t)) + V(t) \beta \bar{k}_0(t) |V(t)|)
\]

Let us define the function \( \hat{H}(y) = \lim_{t \to \infty} H(t, y, k, V). \) Straightforwardly,

\[
\hat{H}(y) = \begin{cases} 
\mu e^{-yt} \beta \bar{k}_0(t) |v_i - v_i - c_4(1 - \beta \mu v_i + V(t) \beta \bar{k}_0(t) |V(t) - v_i| > 0, \\
+\mu(1 - e^{-yt}) \mu(1 - \beta \mu V_i) + V(t) \beta \bar{k}_0(t) |V(t) - v_i| \\
+\mu(1 - e^{-yt}) \mu(1 - \beta \mu V_i) + V(t) \beta \bar{k}_0(t) |V(t) - v_i| \end{cases}
\]

Since \( v_i < 0, \) \( \hat{H}(y) \) is concave in \( y. \) Therefore, necessary conditions are also sufficient by the Arrow–Kurz theorem (see e.g. (Arrow and Kurz, 1970)).

**Proof of Proposition 3.** Fix a player \( i \), a date \( t \) and a value \( \bar{k}_0(t). \) As infected individuals of the severe type completely self-isolate, \( \bar{k}_0(t) \leq \mu. \) Moreover, \( p^*(t) \) is non increasing in \( t, \) thus \( p^*(t) \leq 1 - 1 - \mu \) for all \( t. \) Finally, \( \bar{i}(t) < 1 \) and \( \bar{i}(t) \leq V_i(t) \leq 0. \) Therefore, the following chain of inequalities holds:

\[
p_i^*(t) \beta \bar{i}(t) |V_i^*(t) - v_i| - c_4(1 - \mu \beta \bar{i}(t) |V_i^*(t) - v_i|) \leq 0.
\]

As a consequence, if \( (1 - \mu) \beta \bar{i}(t) |V_i^*(t) - v_i| + c_4(1 - \mu) \beta \bar{i}(t) |V_i^*(t) - v_i| > 0, \) \( p_i^*(t) \beta \bar{i}(t) |V_i^*(t) - v_i| - c_4(1 - \mu) \beta \bar{i}(t) |V_i^*(t) - v_i| < 0, \) which implies that \( k^*(t) = 1 \) is the unique best response for player \( i \) to every \( \bar{k}_0(t) \) by condition (7) in Proposition 1. Therefore, the condition \( (1 - \mu) \beta \bar{i}(t) |V_i^*(t) - v_i| + c_4(1 - \mu) \beta \bar{i}(t) |V_i^*(t) - v_i| > 0, \) guarantees that the game has a unique equilibrium in dominant strategies, in which \( k^*(t) = 1 \) for every \( t. \)

Let us now determine the players’ payoff in this equilibrium. As a first step, let us compute the players’ belief at time \( t. \) Plugging \( \bar{k}_0(t) = 1 \) and \( \bar{k}_0(t) = \mu \) into the belief dynamics (3), we obtain that player’s belief is the solution of the ODE:

\[
\bar{p}(t) = -p(t) \frac{1 - p(t)}{\bar{p}(t)} \mu(t) V_i(t)
\]

with initial condition \( p(0) = 1 - \mu. \) Integrating between 0 and \( t, \) we obtain

\[
\int_0^t \frac{1 - p(t)}{\bar{p}(t)} dt = \int_0^t \mu(t) \bar{u}(t) dt.
\]
which, after straightforward simplifications, yields:
\[
p(t) = \frac{1 - \mu}{1 - \mu + \mu z(t)},
\]
with \(z(t) = e^{\mu t} \beta k(t) \mu^\delta du\). Now, plugging the latter value of \(p(t)\), \(k_i(t) = 1\) and \(\hat{k}_i(t) = \mu\) into the Euler condition in Proposition 1, we obtain that the players’ payoff is the solution of the ODE:
\[
\dot{V}(t) - rV(t) - \frac{1 - \mu}{1 - \mu + \mu z(t)} \beta \mu \mu i(t)V(t) = -\frac{1 - \mu}{1 - \mu + \mu z(t)} \beta \mu \mu i(t) v_i,
\]
with initial condition \(V(0) = 0\). Multiplying both hands of the latter ODE by \(a(t) := e^{-rt} \frac{1 - \mu + \mu z(t)}{z(t)}\), we obtain:
\[
\dot{V}(t) a(t) + V(t) \dot{a}(t) = -e^{-rt}(1 - \mu) \beta \mu \mu i(t) \frac{v_i}{z(t)} v_i.
\]
Integrating (8) between \(t\) and \(T\) and using that \(V(T) = 0\), we obtain:
\[
V(t) = \psi(1 - \mu) \beta \mu \mu i(t) \int_t^T e^{-r(u-t)} \frac{\mu}{z(u)} du,
\]
which, after simplifications, reduces to the expression in Proposition 3. □

**Proof of Proposition 4.** Consider an interior strategy profile \(k^* = (k_i^*)\), i.e., such that \(k_i^* \in (0, 1) \forall i, t\).

- Let us first prove that, if \(k^*\) is an equilibrium, then it is symmetric, i.e., \(k_i^* = k_j^* \forall i, j\). Fix player \(i\) by condition (7) in Proposition 1, an interior strategy \(k^*_i\) is a best response to \(k_i^*\) if and only if, for every \(t \in [0, T]\),
\[
p_i^*(t) \beta k_i^*(t) i(t) V_i^*(t) - v_i) - c_i = 0.
\]
Plugging (9) into the Euler condition, player \(i\)'s equilibrium payoff satisfies in this case:
\[
\dot{V}_i^*(t) - rV_i^*(t) = c_i \forall t \in [0, T].
\]
Integrating (10) between \(t\) and \(T\) and using the terminal condition \(V(T) = 0\), we obtain player \(i\)'s payoff at time \(t\) in equilibrium \(k^*\):
\[
V_i^*(t) = -\frac{c_i}{r} \left(1 - e^{-r(T-t)}\right) := v_i(t).
\]
Plugging \(V_i^*(t) = v_i(t)\) into (9), we obtain player \(i\)'s belief at time \(t\) in equilibrium \(k^*\):
\[
p_i^*(t) = \frac{c_i}{\beta k_i^*(t) i(t)(v_i(t) - v_i)}.
\]
It follows that all individuals playing an interior strategy have the same belief at each date. By the belief dynamics in Proposition 1, \(p_i^* = p_i^*\) implies that \(k_i^* = k_i^*\), thus that \(k^*\) is symmetric.

- Let us now characterize the strategy \(\hat{k}\) of the interior (symmetric) equilibrium whenever it exists. As a preliminary, let us describe the dynamics of the population in a symmetric equilibrium in which all individuals play \(\hat{k}\). Since individuals completely self-isolate when they have symptoms, every susceptible individual as well as every infected individual of the asymptomatic type plays \(\hat{k}\) at time \(t\). As a result, \(\hat{k}(t) = \hat{k}(t)\) and \(\hat{k}(t) = \mu \hat{k}(t)\), which, once plugged into Eqs. (1) and (2), yields:
\[
\dot{\hat{s}(t)} = -\beta \mu s(t) \hat{k}(t) \hat{k}(t)^2,
\]
\[
\dot{\hat{i}(t)} = \beta \mu s(t) i(t) \hat{k}(t)^2 - (\gamma + (1 - \mu) \nu) i(t).
\]
Moreover, individuals have all the same law of motion of beliefs:
\[
\dot{\hat{p}(t)} = -p(t)(1 - p(t)) \beta \mu \mu i(t) \hat{k}(t)^2.
\]
The expression of \(\hat{k}(t)\) in Proposition 4 is obtained by plugging \(V_i^*(t) = v_i(t), p_i^*(t) = p(t)\) and \(k_i(t) = \mu \hat{k}(t)\) into (9).

- Finally, \(\hat{k}\) is well defined as an interior strategy if and only if \(\hat{k}(t) \in (0, 1)\) for every \(t \in [0, T]\). □

**A.2. An extension of the model with co-morbidities**

In this section we augment the model by assuming that each individual \(\epsilon_i \in [c, \bar{c}]\), with \(c\) standing for “presence of co-morbidities” and \(\bar{c}\) for “absence of co-morbidities”. An individual knows her health condition but not her type (asymptomatic or severe). For simplicity, we assume that the probability of being the asymptomatic type is independent of the health condition. Assuming that individuals with co-morbidities are more likely to be the severe type, though more realistic, would not change the form of the best-responses and the nature of our results.

All individuals of the asymptomatic type recover at rate \(\gamma\) when they are infected. Individuals of the severe type with health condition \(c \in [c, \bar{c}]\) recover at rate \(\gamma^c\) and die at rate \(\nu^c\), with \(\gamma^c + \nu^c \in (0, 1)\). To capture the effect of co-morbidities on the course of the disease, we assume that individuals of the severe type without co-morbidities recovery faster: \(\gamma^c \geq \gamma^s\), are less likely to die: \(\nu^c \leq \nu^s\), and suffer less from the disease: \(\bar{c}^c \leq \bar{c}^s\).

At each time \(t\) the population is divided into five groups: susceptible with and without co-morbidities: \(S^c(t) \) and \(S^s(t)\), infected with and without co-morbidities: \(I^c(t) \) and \(I^s(t)\), and recovered: \(R(t)\). Using the same notation as in the main model, the dynamics of the epidemic are governed by the following equations for each \(\epsilon \in [c, \bar{c}]\):
\[
s^{\epsilon}(0) = 1 - r(0) = s_0^\epsilon \in (0, 1) \land \forall t \in [0, T],
\]
\[
\dot{s}^{\epsilon}(t) = -\beta k^{\epsilon}(t) s^\epsilon(t) \bar{k}(t) i(t),
\]
\[
\dot{i}^{\epsilon}(t) = \beta k^{\epsilon}(t) s^\epsilon(t) \bar{k}(t) i(t) - (\gamma(1 - \mu) + (1 - \mu) \nu^\epsilon) i(t)\]
where \(\bar{k}(t) = \bar{k}(t) + \bar{k}(t)\), \(\bar{k}(t) := 1 - \int_{\epsilon_{\delta} \in [\bar{c}, \bar{c})} k(t) d\delta\) and \(\bar{k}(t) := 1 - \int_{\epsilon_{\delta} \in [\bar{c}, \bar{c})} k(t) d\delta\) denote the average fraction of time spent outside at time \(t\) by infected and susceptible people with condition \(\epsilon\), respectively. The continuation payoff of an individual with health condition \(\epsilon\) the first time she has symptoms equals:
\[
v^\epsilon_i = -\frac{1}{r + \gamma^c + \nu^c} (c_i + c_i^c + v^c)\]
As \(v^\epsilon_i\) increases with \(\gamma^c\) and decreases with \(c_i^c\) or \(v^c\), it is smaller for an individual with co-morbidities, thus \(v^c_i < v^\epsilon_i\). Like in the case of a homogeneous population, individual \(i\) decides whether to go out after comparing the confinement cost with her expected marginal benefit of confinement, thus the best-response of individual \(i\) at time \(t\) is:
\[
k_i^c(t) \begin{cases} 
0 & \text{if } p_i(t) \beta k_i(t) i(t) (V_i^c(t) - v_i^c) - c_i < 0, \\
\in [0, 1) & \text{if } p_i(t) \beta k_i(t) i(t) (V_i^c(t) - v_i^c) - c_i = 0, \\
1 & \text{if } p_i(t) \beta k_i(t) i(t) (V_i^c(t) - v_i^c) - c_i > 0.
\end{cases}
\]
Therefore, Proposition 2 and (an adapted version of) Proposition 3 remain true in the augmented model: any equilibrium features social interaction, the game admits a unique equilibrium in which no individual self-isolates provided that the confinement cost is large enough, i.e., when \(c_i + (1 - \mu) \bar{c}_i \geq 0\). However, the existence of observable types of individuals with different continuation payoffs in case of infection precludes the existence of interior equilibria, as stated in the next proposition.

**Proposition 5.** If all individuals with health condition \(\epsilon\) play an interior strategy in equilibrium, then they all play the same interior strategy, and no individual with health condition \(\epsilon' \neq \epsilon\) plays an interior strategy.
Proof. The proof is a direct adaptation of the proof of Proposition 3, in which we establish that players cannot play different interior strategies in equilibrium, and that an interior strategy yields the payoff \( v(t) = -\frac{\gamma}{2}(1 - e^{-\gamma(T-t)}) \).

Fix a health condition \( \epsilon \in [c, \bar{c}] \), and suppose that all individuals with condition \( \epsilon \) play an interior strategy. By Proposition 3, they all play the same strategy and hold the same belief \( p(t) \), which satisfies:

\[
p(t)\beta\hat{k}(t)\hat{v}(t)\hat{v}(t)\hat{v}(t) = \epsilon t - c_H = 0.
\]

The latter condition cannot be satisfied for \( v' \neq v' \), thus no player with condition \( \epsilon' \neq \epsilon \) can play an interior strategy. \( \Box \)

The next proposition states that the outcome in which individuals with co-morbidities self-isolate while those without comorbidities partially self-isolate exist in equilibrium.

**Proposition 6.** Let \( \hat{k}(t) := \frac{c_H}{p(t)\beta\mu\hat{k}(t) - \frac{\gamma}{2}(1 - e^{-\gamma(T-t)}) - v(t)^2} \), where \( p(t) \) and \( \hat{f}(.) \) are determined by:

\[
\begin{align*}
p(t)(1 - \mu), \ s(t)(1 - \hat{f}(0)) &= \bar{s}_0 \text{ and } \forall t \in [0, T], \\
p(t) &= -p(t)(1 - p(t))\beta\mu\hat{k}(t)\hat{v}(t)^3, \\
\hat{f}(t) &= -\beta\mu\hat{k}(t)\hat{v}(t)^2, \\
\hat{f}(t) &= -s(t) - (\mu\gamma + (1 - \mu)(\gamma^2 + v^2))\hat{f}(t).
\end{align*}
\]

If \( \hat{k}(t) \leq 1 \) for all \( t \leq T \), then \( k^*_c(t) = 0 \) and \( k^*_c(t) = \hat{k}(t) \) for every \( t \).

**Proof of Proposition 6.** Let us determine the conditions under which \( k^*_c(t) = 0 \) and \( k^*_c(t) \notin [0, 1] \) for every \( t \in [0, T] \). Individuals with condition \( c \) have a constant belief and do not get infected, thus \( \forall t \),

\[
p(t) = 1 - \mu, \ s(t) = \bar{s}_0 \text{ and } i(t) = \bar{i}_0e^{-\gamma(1 - \mu)(\gamma^2 + v^2)t}.
\]

Individuals with condition \( \bar{c} \) have a nondecreasing belief \( p(t) \) defined by

\[
\hat{f}(t) = -p(t)(1 - \hat{f}(t))\beta\mu\hat{k}(t)\hat{v}(t)^3, \quad \hat{f}(0) = 1 - \mu
\]

and the dynamics of the population of individuals without comorbidities is given by:

\[
\begin{align*}
\dot{k}(t) &= -\beta\hat{k}(t)\hat{v}(t)^2, \\
\hat{f}(t) &= -\hat{k}(t) - (\mu\gamma + (1 - \mu)(\gamma^2 + v^2))\hat{f}(t), \quad \hat{f}(0) = \bar{i}_0.
\end{align*}
\]

By Proposition 1, \( k^*_c(t) \notin [0, 1] \) if and only if

\[
c_H - p(t)\beta\mu k^*_c(t)\hat{i}(t)(V^*_c(t) - v^*_f) = 0.
\]

Plugging the latter indifference condition into the Euler condition, we obtain

\[
V^*_c(t) - rV^*_c(t) = c_H.
\]

Integrating the latter expression between \( t \) and \( T \), and using the terminal condition \( V^*_c(T) = 0 \), we obtain

\[
V^*_c(t) = -\frac{c_H}{r}(1 - e^{-r(T-t)}),
\]

and

\[
k^*_c(t) = \frac{-\frac{c_H}{r}(1 - e^{-r(T-t)} - v^*_f)}{p(t)\beta\mu k^*_c(t)\hat{i}(t)} := \hat{k}(t).
\]

If \( k^*_c(t) = 0 \) for every \( t \), then \( V^*_c(t) = -\frac{c_H}{r}(1 - e^{-r(T-t)}) \). Therefore, playing \( k^*_c(t) = 0 \) for every \( t \) is a best response for individuals with condition \( c \) if and only if

\[
c_H - p(t)\beta\mu k^*_c(t)\hat{i}(t)(V^*_c(t) - v^*_f) = \frac{c_H}{r}(1 - e^{-r(T-t)} - v^*_f) < 0.
\]

As \( p(t) = 1 - \mu \) and \( k^*_c(t) = \hat{k}(t) \),

\[
c_H - p(t)\beta\mu k^*_c(t)\hat{i}(t)(V^*_c(t) - v^*_f) = \frac{c_H}{r}(1 - e^{-r(T-t)} - v^*_f) \times (p(t)(V^*_c(t) - v^*_f) - (1 - \mu)(V^*_c(t) - v^*_f))
\]

As \( p(t) \leq 1 - \mu, v^*_f < v^*_c \) and \( V^*_c(t) = V^*_c(t) \), the above expression is negative. Therefore, the best response of players with condition \( c \) to \( k \) is to play \( k^*_c(t) = 0 \) for every \( t \). The only equilibrium condition is \( k(t) < 1 \) for every \( t \). \( \Box \)

### A.3. Sensitivity analysis

Because epidemiological parameter measurement (\( \beta, \gamma, R_0 \) and \( \mu \)) is sensitive to the context, it is natural to ask whether the main insights of the baseline simulation are sensitive to these measures. In this section, we conduct a sensitivity analysis in which we consider other couples \( (R_0, \mu) \). The first one, our upper bound, is the one of Acemoglu et al. (2020): (2.14, 40%). The second one, our lower bound, are the values observed by Mizumoto et al. (2020) and Zhang et al. (2020) on the Diamond Princess (2.28, 17.9%). In the rest of the section, we refer to the set of values \( A \) for the values used in Acemoglu et al. (2020) and to the set of values \( DP \) for the values observed on the Diamond Princess. Below we explain the context of study for each set of values.

**Set of values \( A \):** Acemoglu et al. (2020) have proposed a set of parameters to describe the Covid-19 infection based on the
report of Ferguson et al. (2020). These results, obtained in mid-March 2020 with very preliminary data, have since been strongly criticized for the pessimistic nature of their estimates. However, several governments (including U.K. and Canada) have based their lockdown decisions on these estimates, hence we have chosen to use them as an upper bound.

**Set of values DP**: In February 2020, at the very start of the pandemic, the Diamond Princess cruise ship, following the diagnosis of 10 of its passengers with COVID-19, was quarantined. The 3711 passengers and crew were tested and at least 712 of them were diagnosed positive for Covid-19 – of this number 14 have died. This natural experiment in a small environment where the population density is high, offered a unique opportunity for researchers to study the dynamics of the infection and to determine its key values. Mizumoto et al. (2020) determined, using statistical analysis adjusted for infection delay, that the proportion of asymptomatic individuals aboard the Diamond Princess as of February 20, 2020 was 17.9%. However, they did not estimated the epidemiological parameters. We therefore turned to Zhang et al. (2020). They estimated the value of $R_0$ for the Diamond Princess’ crew members and passengers to be $R_0 = 2.28$, using the maximum likelihood method and assuming $\gamma = 7.5$.

As a preliminary, we have checked that the interior strategy profile is an equilibrium with the set of values $A$ and $DP$. We first simulate the dynamics of the epidemic with each set of values. We observe that the fraction of infected, the fraction of time spent outside, the effective reproduction number and the fraction
Fig. 15. Sensitivity analysis: effect of a variation of $\tau$ on the fraction of infected.

Fig. 16. Sensitivity analysis: effect of a variation of $\tau$ on behaviors and beliefs.

Fig. 17. Sensitivity analysis: effect of a variation of $\tau$ on deaths.

Fig. 18. Sensitivity analysis: effect of a variation of $\beta \mu$ on the fraction of infected.
of deaths of the baseline simulation is framed between the two alternative set of values, and that the curves have the same shape (see Figs. 11–14). As $R_0$ is greater with the set $A$ than with the set $DP$, individuals self-isolate more with the former set than in the later. However, in all cases, individuals drastically reduce their contacts after the announcement; when the spread of the epidemic is under control, they gradually increase the time spent outside to a plateau, which maintains the effective reproduction number close to 1, thus containing the spread of infection.

Next, we simulate our policy analysis with the two alternative set of values and obtain the same qualitative results:

- Delaying the epidemic announcement accelerates the epidemic (see Fig. 15) and increases the number of deaths (see Fig. 17). This is because individuals drastically self-isolate after the announcement, then gradually reduce their level of self-isolation to a level maintaining the effective reproduction number approximately equal to 1 (see Fig. 16).

- We multiply the value of $\beta\mu$ by 0.75, 1, 1.25 and 1.5 for each set of values (which yields $\beta\mu \in \{0.1005, 0.134, 0.1675, 0.201\}$ in set $A$ and $\beta\mu \in \{0.228, 0.304, 0.38, 0.456\}$ in set $DP$). We find also find that decreasing $\beta\mu$ induces less self-isolation and reduces the number of deaths (see Figs. 18–20).

- We multiply the value of $\gamma$ by 1.1 for each set of values and the value of $\nu$ by 0.5 in each set of values. While the dynamics of the infection follow a very similar trajectory in the two scenarios, the infection is stronger in set $A$ than in
Fig. 22. Sensitivity analysis: effect of a variation of $\gamma$ on beliefs and behaviors.

Fig. 23. Sensitivity analysis: effect of a variation of $\gamma$ on deaths.

set DP (see Fig. 21). We also find that a better health system induces less self-isolation (Fig. 22) but reduces the number of deaths (see Fig. 23).

References

Acemoglu, D., Chernozhukov, V., Werning, I., Whinston, M.D., 2020. Optimal Targeted Lockdowns in a Multi-Group SIR Model. NBER Working Paper, p. 27102.

Ahituv, A., Hotz, V.J., Philipson, T., 1996. The responsiveness of the demand for condoms to the local prevalence of AIDS. J. Hum. Resour. 36, 897–910.

Aiello, A.E., Murray, G.F., Perez, V., Coulborn, R.M., Davis, R.M., Uddin, M., Monto, A.S., 2010. Mask use, hand hygiene, and seasonal influenza-like illness among young adults: a randomized intervention trial. J. Infec. Dis. 201 (4), 491–498.

Alvarez, F.E., Argente, D., Lippi, F., 2020. A Simple Planning Problem for Covid-19 Lockdown (No. W26981). National Bureau of Economic Research.

Arrow, K., Kurz, M., 1970. Optimal growth with irreversible investment in a ramsey model. Econometrica 38, 331–344.

Bairoliya, N., Imrohoroglu, A., 2020. Macroeconomic consequences of stay-at-home policies during the COVID-19 pandemic. Covid Econ. 13, 71-90.

Britton, T., Ball, F., Trapman, P., 2020. A mathematical model reveals the influence of population heterogeneity on herd immunity to SARS-CoV-2. Science.

Brotherhood, L., Kircher, P., Santos, C., Tertilt, M., 2020. An economic model of the Covid-19 epidemic: The importance of testing and age-specific policies. Chen, F. 2012. A mathematical analysis of public avoidance behavior during epidemics using game theory. J. Theoret. Biol. 302, 18-28.

Chen, F., Jiang, M., Rabidoux, S., Robinson, S., 2011. Public avoidance and epidemics: insights from an economic model. J. Theoret. Biol. 278 (1), 107–119.

Cowling, B.J., Ali, S.T., Ng, T.W., Tsang, T.K., Li, J.C., Fong, M.W., ..., Wu, J.T., 2020. Impact Assessment of Non-Pharmaceutical Interventions Against Coronavirus Disease 2019 and Influenza in Hong Kong: An Observational Study. The Lancet Public Health.

Cowling, B.J., Chan, K.H., Fang, V.J., Cheng, C.K., Fung, R.O., Wai, W., Chiu, B.C., 2009. Facemasks and hand hygiene to prevent influenza transmission in households: a cluster randomized trial. Ann Intern. Med. 151 (7), 437–446. Delamater, P.L., Street, E.J., Leslie, T.F., Yang, Y.T., Jacobsen, K.H., 2019. Complexity of the basic reproduction number (R0). Emerg. Infect. Diseases 25 (1), 1.

Eichenbaum, M.S., Rebelo, S., Trabandt, M., 2020. The Macroeconomics of Testing and Quarantining (No. W27104). National Bureau of Economic Research.

Farooni, M., Jarosch, G., Shimer, R., 2020. Internal and External Effects of Social Distancing in a Pandemic (No. W27059). National Bureau of Economic Research.

Favoro, C.A., Ichino, A., Rustichini, A., 2020. Restarting the Economy While Saving Lives under Covid-19. mimeo.

Fenichel, E.P., 2013. Economic considerations for social distancing and behavioral based policies during an epidemic. J. Health Econ. 32 (2), 440–451.

Fenichel, E.P., Castillo-Chavez, C., Ceddia, M.G., Chowell, G., Parra, P.A.G., Hickling, G.J., Springborn, M., 2011. Adaptive human behavior in epidemiological models. Proc. Natl. Acad. Sci. 108 (15), 6306–6311.

Ferguson, N., Laydon, D., Nedjati-Gilani, G., Imai, N., Ainslie, K., Baguelin, M., Dighe, A., 2020. Report 9: Impact of non-pharmaceutical interventions (NPIs) to reduce COVID19 mortality and healthcare demand. Imperial College London 10 (77482).

Glover, A., Heathcote, J., Krueger, D., Rios-Rull, J.V., 2020. Health Versus Wealth: On the Distributional Effects of Controlling a Pandemic (No. W27046). National Bureau of Economic Research.

Han, D., Li, R., Han, Y., Zhang, R., Li, J., 2020. COVID-19: Insight into the asymptomatic SARS-COV-2 infection and transmission. Int. J. Biol. Sci. 16 (15), 2803.

Jones, C.J., Philippon, T., Venkateswaran, V., 2020. Optimal Mitigation Policies in a Pandemic: Social Distancing and Working from Home (No. W26984). National Bureau of Economic Research.

Kellam, P., Barclay, W., 2020. The dynamics of humoral immune responses following SARS-CoV-2 infection and the potential for reinfection. J. Gen. Virol. 001439.

Kermack, W.O., McKendrick, A.G., 1927. A contribution to the mathematical theory of epidemics. Proc. R. Soc. Lon. Series A 115 (772), 700–721.

Kruse, T., Strack, P., 2020. Optimal Control of an Epidemic Through Social Distancing. mimeo.
