# Self-isolation under uncertainty* 

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#### Abstract

We analyze an epidemiological model where forward-looking individuals trade off the costs and benefits of self-isolation while being uncertain about the dynamics of the epidemic. We characterize the interior symmetric equilibrium and we identify necessary conditions of the optimal solution. We calibrate our model to the COVID-19 pandemic and simulate the dynamics of the epidemic under various scenarios to illustrate the impact of uncertainty on self-isolation behaviors. We show that uncertainty may cause a second wave of infection and that the average level of social activity can decrease with uncertainty. Finally, uncertainty about the epidemic dynamics may be welfare improving, both in terms of fraction of deaths and average payoff.


Keywords: SIR model; Self-isolation; Uncertainty; COVID-19 epidemic.
JEL codes: C73; D84, I12.

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## 1 Introduction

It is now well-documented that the dynamics of an epidemic depend on the behavior of the population in terms of social distancing and application of prophylactic measures. ${ }^{1}$ Conversely, many papers have documented that individuals adapt their behaviors to epidemic variables (incidence rate, level of hospitalizations, etc). ${ }^{2}$ For example, Fardoobi et al. (2020) show that attendance in public places declined as soon as the WHO announced the existence of a pandemic in March 2020, thus before the implementation of lockdown and closure policies. The object of the growing strategic epi-economic literature is to analyze the two-sided interactions between the dynamics of epidemic and the population behavior. In this literature, individuals trade off the costs and benefits of preventive behaviors on the basis of their evaluation of the risk of contracting the disease, which depends on the prevalence rate, i.e., the fraction of infected individuals in the population. Typically, individuals are assumed to know the current prevalence rate when they make their decisions. In reality, the presence of asymptomatic individuals and the imperfect knowledge of the characteristics of the disease (contagiousness rate, starting time of the epidemic, etc), make it difficult for individuals, if not impossible, to infer the current prevalence rate from their private information. This is particularly true when the disease is caused by a new virus or resurfaces at random times. Recent examples of such situations abound: COVID-19 and its different mutants, influenza which returns every winter in temperate zones, Ebola which has reappeared several times in DRC, but also in Guinea in 2021, etc...

The contribution of this paper is to analyze an epidemiological model where forwardlooking individuals are uncertain about some characteristics of the epidemic and are therefore unable to infer the fraction of the population that is infected. Individuals form beliefs about

[^1]the epidemic, that they continuously update on the basis of how much they might have been exposed to the virus. Simultaneously, they decide their degree of exposure to the virus by trading off the costs and benefits of self-isolation on the basis of their subjective beliefs.

Precisely, we amend the classical Susceptible-Infected-Recovered (SIR hereafter) model of Kermack and McKendrick (1927). In its classical version, the SIR model divides an 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. As in Baril-Tremblay et al. (2021), we consider two possible types of individuals in the population: symptomatic and asymptomatic. Individuals of the symptomatic type experience the symptoms of the disease immediately after being infected. In contrast, individuals of the asymptomatic type do not have symptoms. Initially, individuals do not know their type. We depart from Baril-Tremblay et al. (2021) by assuming that individuals do not observe the prevalence rate and are uncertain about some parameters governing the dynamics of the prevalence rate.

Individuals influence the transition rate from \{susceptible\} to \{infected\} by self-isolating, i.e., strategically reducing social activity. How does an individual who never had symptoms in the past tradeoff the costs and benefits of self-isolation? On the benefits side, self-isolation prevents one from being infected by reducing the probability of being infected by a contagious person. The costs side is more subtle. Indeed, an individual who does not get symptoms while having social activity becomes more optimistic both on the prevalence rate and on being the asymptomatic type. The costs of self-isolation are thus twofold: there is the direct cost of confinement (boredom, opportunity cost of not working, or of working in poorer conditions, lack of physical activity, etc..) and the opportunity cost of not learning about one's type and about the prevalence rate.

We characterize the symmetric equilibrium in which individuals partially self-isolate at every time. At this equilibrium, the level of social activity is equal to the ratio between, on the one hand, the direct cost of self-isolation, and on the other hand, the expected net cost of the social activity, which is equal to the expected welfare loss in case of infection minus the informational benefit.

We calibrate our model to the COVID-19 and we simulate the dynamics of the epidemic when the population is aware of two possible epidemics with different initial penetration rates.

The impact of uncertainty is ambiguous. For any prior belief, individuals self-isolate drastically after the epidemic announcement, which results in a drop in the fraction of infected; then, they gradually increase the level of social interactions. The rate at which social activity increases varies with the prior that the epidemic has a low initial penetration rate. When the prior increases, individuals self-isolate less at the beginning of the epidemic and more at the end. One reason for this reversal is that individuals believe they have been less exposed to the disease at the beginning of the epidemic when the initial penetration rate is low. As a result, they are less confident in being immune to the disease, and self-isolate more at the end of the outbreak. In the more aggressive epidemic, for any prior, individuals self-isolate enough to maintain the effective reproduction number below the value that accelerates the epidemic, thus the epidemic curve continuously decreases until the arrival of the vaccine. In the less aggressive epidemic, when the initial prior is small there is a second wave of infections with a second peak that is higher the more erroneous the beliefs are. This second wave arises because, for these priors, individuals choose a higher level of social activity from some date on. This leads to an inadequate reaction to the true level of the epidemic that may induce an effective rise of the cumulated fraction of infected individuals. Therefore, the social value of information depends on the initial state of the epidemic. We find that transparency is welfare improving only in the less aggressive epidemic, both in terms of fraction of deaths and payoffs. For all the priors we consider, the ex-ante fraction of deaths is smaller when individuals are uncertain about the state than without uncertainty, suggesting that opacity can prevent deaths. In terms of payoffs, the information value is negative when the population is relatively confident that the epidemic is initially aggressive.

Related literature Many papers in economics have documented that individuals adapt their behavior when facing a risk of infection. Before COVID-19, concern was mainly on AIDS, thus papers analyzed steady-states of Susceptible-Infected models (see for instance Kremer (1996), Philipson and Posner (1993), Toxvaerd (2019)). To analyze an infectious disease like COVID19, recent papers are based on SIR models in continuous time with either forward-looking or myopic individuals. Carnehl, Fukuda and Kos (2022.a) analyze a SIR model with infinite horizon and myopic agents. The infection risk is linear in the average level of social interaction and the isolation cost are quadratic. They show that there exists a unique symmetric equilibrium and that a second wave is impossible. Surprisingly, if the initial faction of infected individuals
is sufficiently small, an epidemic may not start if the virus is very contagious. In a companion paper (Carnehl, Fukuda \& Kos (2022.b)), they assume non stationary isolation cost to capture the lockdown fatigue phenomenon, and give sufficient conditions for the existence of a second wave. Dasaratha (2022) assumes that infected individuals do not observe their health status but know when they are recovered. The infection risk is a quadratic function of the average level of social activity. In the main part of the paper, agents are supposed to be myopic. The author shows that, because agents adapt their behavior, an exogenous marginal increase of the infected individuals can have a negative effect on the number of new cases. Phelan \& Toda (2022) analyze a model in which infected individuals can be asymptomatic with a certain probability. The population is finite and agents ignore the effect of their choice on the infection rate. A vaccine arrives at some random date. They show that there is a unique value function that satisfies the Bellman Equation and that there exists a Markovian equilibrium. They derive the optimal lockdown policy. Toxvaerd (2022) considers a model in which players can be of two types: asymptomatic and symptomatic. An agent's type determines whether he will develop symptoms or not when infected. When an individual is infected and develops symptoms, he chooses the maximal social interaction level. The author shows that when types are not observable, the total number of infection cases is higher but the number of infected with symptoms is lower. Consequently, the welfare is higher when types are not observable. A comparison between the effect of infection and the immunity tests on the dynamic of the epidemic reveals that, except in the early stages, the latter have a higher private value. Finally, our model generalizes Baril-Tremblay et al. (2021) by introducing uncertainty. In both papers, we assume that each agent can be of two types, as in Toxvaerd (2022), the infection risk is a quadratic function of the average level of social activity, the self-isolation costs are linear and the horizon is finite (the underlying assumption is that a vaccine is available after some date).

Several papers adopt a macroeconomics perspective and compare the competitive equilibrium with the confinement effectiveness. Farboodi, Jarosh \& Shimer (2021) assume imperfect observation of the health status. They compare the dynamics obtained at the decentralized equilibrium with the one obtained under the optimal policy and show that the competitive equilibrium is suboptimal. Brotherhood et al. (2020) assume that agents are heterogenous with respect to age and the older people are more likely to die. Rachel (2020) considers a
lockdown problem and provides analytical results.
Several papers put aside the question of individuals' responses to an epidemic risk and focus exclusively on optimal mitigation policies by assuming that a planner controls the transmission rate (see for instance Kruse \& Strack (2022), Alvarez et al (2020)...). Acemoglu et al assume that agents are heterogenous. Giannitsarou et al analyze the dynamics of epidemics under waning immunity.

It is well known that in SIR models without decentralized self-isolation choice and policy intervention, a second wave cannot emerge. In Carnehl, Fukuda \& Kos (2022.b) it arrises because the self-isolation costs are not stationary. Rachel (2020) shows that a second wave can occur after a lockdown. Giannitsarou et al (2021) show that waning immunity induces oscillations. In our paper, the second wave is not due to non stationary self-isolation costs, waning immunity or lockdown release but only to uncertainty.

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 the symmetric equilibrium. In Section 4, we calibrate our model to fit the COVID-19 pandemic, we simulate the dynamics of the epidemic in equilibrium and investigate the impact of uncertainty. In Section 5, we analyze the problem of a government who must decide upon the optimal isolation policy. Proofs are gathered in the Appendix.

## 2 An epidemiological model with uncertainty

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 $j \in[0,1]$ who must continuously choose a level of social activity, which can be interpreted as the fraction of their time they do not spend at home. An individual who stays home is protected from infection, while an individual who goes out may be contaminated by contact with an infected individual.

Infection may be totally asymptomatic. Whether an individual develops symptoms or not when she is infected is an idiosyncratic characteristic described by her type. There are two types of individuals in the population: Individuals of type $\theta_{s}$-the symptomatic type- who experience the symptoms of the disease immediately after being infected, and individuals of
type $\theta_{a}$-the asymptomatic type- who do not have symptoms in case of infection, thus never realize when they have been infected. Individuals do not know their type unless they are of type $\theta_{s}$ and catch the disease. There is a proportion $\alpha \in(0,1)$ of asymptomatic types in the population. The infection period ends by recovery at rate $\gamma_{a}$ for asymptomatic types, and by either recovery or death for symptomatic types, at rates $\gamma_{s}$ and $\nu$. However, infection stops for both types of individuals at the same rate, i.e., $\gamma_{a}=\nu+\gamma_{s}$. Therefore, at each time there is a proportion $\alpha$ of infected individuals who have no symptoms. Finally, individuals are contagious as long as they are infected, and are immune to the disease after recovering.

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 $j$ is a measurable function $k_{j}: \mathbb{R}_{+} \rightarrow[0,1]$, with the interpretation that $k_{j}(t)$ is the proportion of time spent outside at time $t$, absent symptoms by time $t .{ }^{3}$ Therefore, the disease is spread in the population by asymptomatic infected individuals.

The epidemic. An epidemic is characterized by its initial penetration level in the population $(\bar{s}, \bar{a}, \bar{r})$, where $\bar{s} \in[0,1]$ is the proportion of individuals who are not immune to the disease at time $0, \bar{a} \in[0,1]$ is the proportion of individuals infected without symptoms at time 0 and $\bar{r} \in[0,1]$ the proportion of individuals who already recovered from the disease at time 0 and are now immune to it. As the share of asymptomatic types in the general population is $\alpha$, the proportion of individuals infected with symptoms at time 0 is $\bar{i}=\frac{1-\alpha}{\alpha}$, and the proportion of dead individuals is $\bar{d}=1-\bar{s}-\bar{a}-\bar{r}-\bar{i} \in[0,1]$. ${ }^{4}$

We identify an epidemic $\omega$ with the tuple $\{\bar{s}, \bar{a}, \bar{r}\}$ and we denote by $\Omega$ the finite set of possible epidemics.

Dynamics of the epidemic. To model the spread of epidemic $\omega$, we use the classical Susceptible-Infected-Recovered (SIR) model by Kermack and McKendrick (1927), that we amend to introduce individual behaviors and uncertainty.

At each time $t$, the population is divided into five groups: the group of susceptible individuals who have never been infected by the disease, denoted by $S(t \mid \omega)$ and of size

[^2]$s(t \mid \omega):=\int_{j \in S(t \mid \omega)} d j$; the group of symptomatic infected individuals who are infected with symptoms at time $t$, denoted by $I(t \mid \omega)$ and of size $i(t \mid \omega):=\int_{j \in I(t \mid \omega)} d j$; the group of asymptomatic infected individuals who are infected without symptoms at time $t$, denoted by $A(t \mid \omega)$ and of size $a(t \mid \omega):=\int_{j \in A(t \mid \omega)} d j$; the group of recovered individuals who already healed from the disease (with or without symptoms), denoted by $R(t \mid \omega$ ) and of size $r(t \mid \omega):=\int_{j \in R(t \mid \omega)} d j$ and the group of dead individuals, denoted by $D(t \mid \omega)$ and of size $d(t \mid \omega)=1-s(t \mid \omega)-i(t \mid \omega)-a(t \mid \omega)-r(t \mid \omega)$.

The evolution of the epidemic penetration depends on the behavior of the population. Here we explain how by using a probabilistic argument. What is the mass of individuals who become infected in the interval $[t, t+d t)$, in expectation? Fix some date $t$, some strategy profile $\mathbf{k}:=\left(k_{j}\right)_{j \in[0,1]}$ and some small $d t>0$. The probability that a susceptible individual $s \in S(t \mid \omega)$ meets and is infected by some infected asymptomatic individual $a \in A(t \mid \omega)$ during the interval $[t, t+d t)$ is $k_{s}(t) k_{a}(t) \beta d t$. Therefore, the probability that $s$ becomes infected in $[t, t+d t)$ is $k_{s}(t)\left(\int_{j \in A(t \mid \omega)} k_{j}(t) d j\right) \beta d t$, and the expected mass of newly infected individuals is $\int_{s \in S(t \mid \omega)}\left(k_{s}(t) \int_{a \in A(t \mid \omega)} k_{a}(t) d a \beta d t\right) d s$. Therefore, the fraction of susceptible individuals evolves as follows: ${ }^{5}$

$$
\begin{equation*}
\dot{s}(t \mid \omega)=-\beta \bar{k}_{S}(t \mid \omega) s(t \mid \omega) \bar{k}_{A}(t \mid \omega) a(t \mid \omega) \tag{1}
\end{equation*}
$$

where $\bar{k}_{S}(t \mid \omega)=\frac{1}{s(t \mid \omega)} \int_{j \in S(t \mid \omega)} k_{j}(t) d j$ and $\bar{k}_{A}(t \mid \omega)=\frac{1}{a(t \mid \omega)} \int_{j \in A(t \mid \omega)} k_{j}(t) d j$ denote the average behavior of susceptible and asymptomatic infected individuals, respectively. At each time $t$, the fraction of newly infected $-\dot{s}(t \mid \omega)$ is split between $A(t \mid \omega)$ and $I(t \mid \omega)$, in proportions $\alpha$ and $1-\alpha$, respectively. The other groups of the population thus evolve as follows:

$$
\begin{align*}
\dot{a}(t \mid \omega) & =-\alpha \dot{s}(t \mid \omega)-\gamma_{a} a(t \mid \omega)  \tag{2}\\
\dot{i}(t \mid \omega) & =-(1-\alpha) \dot{s}(t \mid \omega)-\left(\gamma_{s}+\nu\right) i(t \mid \omega)  \tag{3}\\
\dot{r}(t \mid \omega) & =\gamma_{a} a(t \mid \omega)+\gamma_{s} i(t \mid \omega)  \tag{4}\\
\dot{d}(t \mid \omega) & =\nu i(t \mid \omega) \tag{5}
\end{align*}
$$

with $s(0 \mid \omega)=\bar{s}, i(0 \mid \omega)=\bar{i}, a(0 \mid \omega)=\bar{a}, r(0 \mid \omega)=\bar{r}$ and $d(0 \mid \omega)=\bar{d}$. The assumptions $\gamma_{a}=\nu+\gamma_{s}$ and $\bar{i}=\frac{1-\alpha}{\alpha} \bar{a}$ guarantee that $i(t \mid \omega)=\frac{1-\alpha}{\alpha} a(t \mid \omega)$ for every $t$.

[^3]Uncertainty and beliefs. At time 0 , individuals learn the existence of an epidemic, but do not know their own type nor which epidemic they are facing. Moreover, they never observe the current epidemic penetration, hence the only additional information they have at time $t$ is whether they had or did not have symptoms before $t .{ }^{6}$

We denote by $p_{j}(t): \Omega \rightarrow[0,1]$ the subjective belief of individual $j$ at time $t$ that she is the symptomatic type, with the interpretation that $p_{j}(t \mid \omega)$ is the probability of player $j$ being type $\theta_{s}$ conditionally on the epidemic being $\omega$ and having experienced no symptom by time $t$. Individual $j$ continuously updates her belief on the basis of whether she is having symptoms, conditionally on the strategy profile of the population and the dynamic system (1) and (2). Precisely, the law of motion of the subjective belief of individual $j$ is ${ }^{7}$

$$
\begin{equation*}
\dot{p}_{j}(t \mid \omega)=-p_{j}(t \mid \omega)\left(1-p_{j}(t \mid \omega)\right) k_{j}(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega), \text { with } p_{j}(0 \mid \omega)=1-\alpha \tag{6}
\end{equation*}
$$

Moreover, we denote by $\mu_{j}(t): \Omega \rightarrow[0,1]$ the subjective belief of individual $j$ at time $t$ over $\Omega$, with the interpretation that $\mu_{j}(t, \omega)$ is the probability at time $t$ for individual $j$ that the epidemic is $\omega$, conditional on having experienced no symptom by time $t$. At time 0 , individuals hold the common belief $\mu^{0}: \Omega \rightarrow[0,1]$. The subjective belief that the epidemic is $\omega$ depends on the subjective belief $p_{j}(t)$ as follows: ${ }^{8}$

$$
\begin{equation*}
\mu_{j}(t, \omega)=\frac{\mu^{0}(\omega) /\left(1-p_{j}(t \mid \omega)\right)}{\sum_{\omega^{\prime} \in \Omega} \mu^{0}\left(\omega^{\prime}\right) /\left(1-p_{j}\left(t \mid \omega^{\prime}\right)\right)} \tag{7}
\end{equation*}
$$

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 symptomatic type because they suffer from the symptoms, and, in the worst case, die from the disease. Therefore, at each time $t$, individuals tradeoff the cost of self-isolating and the expected benefit of having no symptoms. We denote by $c_{S}$ the flow cost of self-isolation, by $c_{I}$ the flow cost of having symptoms and

[^4]by $c_{D}$ the flow cost of being dead. The flow payoff of having social activity and being healthy is normalized to 0 .

Uncertainty about her type is solved for an individual the first time she has symptoms. In that event, she knows that she is the symptomatic type, thus that she will stay at home until she heals or passes away, thereby incurring a total cost of $\int_{0}^{\min \left\{\tau_{H}, \tau_{D}\right\}} e^{-r t}\left(c_{S}+c_{I}\right) d t$, with $\tau_{H}$ and $\tau_{D}$ standing for the random times of healing and death, respectively. If she heals from the disease (i.e., if $\tau_{H}<\tau_{D}$ ), she becomes immune to it, plays $k(t)=1$ forever after, thus obtains the continuation payoff 0 . If she dies (i.e., if $\tau_{D}<\tau_{H}$ ), she incurs the flow cost $c_{D}$ forever after, thus obtains the continuation payoff $-c_{D} / r$. Therefore, the expected continuation payoff to an individual the first time she has symptoms is: ${ }^{9}$

$$
\begin{equation*}
v_{I}=-E\left[\int_{0}^{\min \left\{\tau_{H}, \tau_{D}\right\}} e^{-r t}\left(c_{S}+c_{I}\right) d t+\frac{c_{D}}{r} e^{-r \tau_{D}} \mathbb{1}_{\tau_{D}<\tau_{H}}\right]=-\frac{1}{r+\gamma^{s}+\nu}\left(c_{S}+c_{I}+\nu \frac{c_{D}}{r}\right) \tag{8}
\end{equation*}
$$

Let us express the payoff increment at time $t \in(0, T)$ in epidemic $\omega$. Conditionally on having no symptoms before $t$, player $j$ obtains the continuation payoff $v_{I}$ if she has symptoms, which occurs if she is the symptomatic type and gets infected, thus with probability $p_{j}(t \mid$ $\omega) k_{j}(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)$. She also bares the confinement cost per unit of time in selfisolation, thus $c_{S}\left(1-k_{j}(t)\right)$. After getting vaccinated at time $T$, she has a probability 0 of developing symptoms and plays $k_{j}(t)=1$ for every $t \geq T$, which yields the continuation payoff 0 . Finally, the subjective probability of having no symptom before $t \in[0, T]$ in epidemic $\omega$ is $1-p_{j}(0 \mid \omega)+p_{j}(0 \mid \omega) e^{\left.-\int_{0}^{t} k_{j}(s) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega)\right] d s}$, which reduces to $e^{-\int_{0}^{t} p_{j}(s \mid \omega) k_{j}(s) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s}$ by integrating (6). After simplifications, the discounted expected payoff conditional on the epidemic being $\omega$ is: ${ }^{10}$
$v\left(k_{j} \mid \omega\right)=\int_{0}^{T} e^{-r t} \underbrace{\begin{array}{c}\text { Player } j \text { 's expected payoff increment at } t \text { con- } \\ \text { ditional on having no symptom before } t .\end{array}}_{\begin{array}{c}\text { Player } j \text { 's probability to } \\ \text { have no symptom before } t .\end{array}}$

[^5]
## 3 Equilibrium analysis

Fix a strategy profile $\mathbf{k}$. Player $j$ 's best-response problem is to maximize $E_{\mu^{0}}\left[v\left(k_{j} ;,.\right)\right]$, where the fraction of asymptomatic infected at time $t$ is given for each $\omega$ by the system of o.d.e. $\{(1),(2)\}$. Formally, it is the solution of the optimal control problem:

$$
\begin{cases}\max _{k_{j} \in \mathcal{K}} & E_{\mu^{0}}\left[v\left(k_{j} \mid .\right)\right] \\ \text { s.t. } & \forall \omega \in \Omega, p_{j}(0 \mid \omega)=1-\alpha \text { and, } \forall t \in[0, T], \\ & \dot{p}_{j}(t \mid \omega)=-p_{j}(t \mid \omega)\left(1-p_{j}(t \mid \omega)\right) k_{j}(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega),\end{cases}
$$

which we solve in the Appendix using Pontryagin's principle. Here, we explain the intuition of the solution with a heuristic dynamic programming argument, using the time and the player's current belief of being the symptomatic type $p$ as the state variable. At time $t$, the optimal social activity level of an individual maximizes the sum of her current expected payoff increment and of her discounted continuation payoff, should no symptoms occur in the interval $[t, t+d t)$. Given the strategy profile $\mathbf{k}$, the best-response payoff to a player at time $t$ and belief $p$ satisfies the Bellman equation
$V(t, p)=\max _{k \in[0,1]}\left\{\left(-(1-k) c_{S}+v_{I} P(S(t) \mid p)\right) d t+(1-P(S(t) \mid p) d t) e^{-r d t} V(t+d t, p+d p \mid \bar{S}(t))\right\}$,
where $S(t)$ stands for the event "having symptoms between $t$ and $t+d t$ " and $\bar{S}(t)$ for the complementary event. By Bayes' rule, the probability of developing symptoms between $t$ and $t+d t$ is linear in the individual's action $k$, with

$$
P(S(t) \mid p)=k \sum_{\omega} \mu(t, \omega) p(t \mid \omega) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega) .
$$

Moreover,

$$
V(t+d t, p+d p \mid \bar{S}(t))=V(t, p)+V_{t}(t, p) d t+\sum_{\omega} V_{p(t \mid \omega)}(t, p) \dot{p}(t \mid \omega) d t
$$

Using (6), eliminating terms to the order $(d t)^{2}$ and simplifying, we can rewrite the Bellman equation (10) as follows:

$$
\begin{aligned}
& r V(t, p)=V_{t}(t, p)-c_{S} \\
& \quad+\max _{k \in[0,1]} k(c_{S}-\beta \underbrace{\sum_{\omega} \mu(t, \omega) p(t \mid \omega) \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(V(t, p)-v_{I}+\frac{1-p(t \mid \omega)}{\mu(t, \omega)} V_{p(t \mid \omega)}(t, p)\right.}_{\text {expected net cost of social activity }}) .
\end{aligned}
$$

To interpret this expression, note that two things might happen for the individual at time $t$ : either she gets symptoms, or she does not. In the first case, she incurs a payoff loss of $V(t, p)-v_{I}$; in the second case, she becomes more confident in being the asymptomatic type, which increases her continuation payoff by $-V_{p(t \mid \omega)}(t, p)$. Therefore, the marginal benefit of more social activities is the difference between the direct cost of self-isolation, $c_{S}$, and the expected net cost of social activity, composed of

- the expected cost of the jump to $v_{I}$ in case of symptoms: $\beta \sum_{\omega} \mu(t, \omega) p(t \mid \omega) \bar{k}_{A}(t \mid$ $\omega) a(t \mid \omega)\left(V(t, p)-v_{I}\right)$;
- the opportunity cost in terms of payoff of not becoming more optimistic about being the asymptomatic type in the absence of symptoms: $\beta \sum_{\omega} p(t \mid \omega)(1-p(t \mid \omega)) \bar{k}_{A}(t \mid$ $\omega) a(t \mid \omega) V_{p(t \mid \omega)(t, p)}$.

The next proposition gives the necessary conditions for a strategy of player $j$ to be a best response against a strategy profile $\left(k_{j^{\prime}}\right)_{j^{\prime} \neq j}:=\mathbf{k}_{-j}$ :

Proposition 1 (Best response). If $k_{j}^{*}$ the best-response of player $j$ against the strategy profile $\mathbf{k}_{-j}$, then there exists functions $\psi_{j}: \mathbb{R}_{+} \times \Omega \rightarrow \mathbb{R}$ and $p_{j}: \mathbb{R}_{+} \times \Omega \rightarrow[0,1], C^{1}$ in the first argument and such that, such that, for all $t \leq T$ :

$$
k_{j}^{*}(t) \begin{cases}=1 & \text { if } c_{S}>\beta \sum_{\omega} \mu_{j}(t, \omega) p_{j}(t \mid \omega) \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi_{j}(t \mid \omega)-v_{I}\right),  \tag{11}\\ \in[0,1] & \text { if } c_{S}=\beta \sum_{\omega} \mu_{j}(t, \omega) p_{j}(t \mid \omega) \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi_{j}(t \mid \omega)-v_{I}\right), \\ =0 & \text { if } c_{S}<\beta \sum_{\omega} \mu_{j}(t, \omega) p_{j}(t \mid \omega) \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi_{j}(t \mid \omega)-v_{I}\right),\end{cases}
$$

where, for all $\omega \in \Omega$,
$\dot{\psi}_{j}(t \mid \omega)-r \psi_{j}(t \mid \omega)=k_{j}^{*}(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi_{j}(t \mid \omega)-v_{I}\right)+\left(1-k_{j}^{*}(t)\right) c_{S}, \psi_{j}(T \mid \omega)=0$, $\dot{p}_{j}(t \mid \omega)=-p_{j}(t \mid \omega)\left(1-p_{j}(t \mid \omega)\right) k_{j}^{*}(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega), p_{j}(0 \mid \omega)=1-\alpha$,
and $\mu_{j}(t, \omega)$ is defined by (7).
Proof. See the Appendix.

An immediate corollary of Proposition 1 is that all equilibria feature social interaction, in the sense that, at every period, 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 self-isolation cost $c_{S}$ by going out without risking infection. Therefore, in the symmetric equilibrium, at each date individuals either partially self-isolate or do not self-isolate at all. The strategy profile $\mathbf{k}$ is said to be symmetric interior if $k_{j}(t)=k_{j^{\prime}}(t)$ for all players $j, j^{\prime}$ and $k_{j}(t) \in(0,1)$ for all $t$. The following lemma gives necessary and sufficient conditions for a symmetric interior strategy profile to be an equilibrium.

Proposition 2 (The symmetric equilibrium). Let $\hat{\mathbf{k}}$ be the symmetric strategy profile where all individuals play $\hat{k}$ defined by

$$
\hat{k}(t)=\frac{c_{S}}{\beta \sum_{\omega} \mu(t, \omega) p(t \mid \omega) a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)}
$$

where

$$
\left\{\begin{array}{l}
\forall t \in[0, T], \forall \omega \in \Omega  \tag{12}\\
\dot{\psi}(t \mid \omega)-r \psi(t \mid \omega)=\hat{k}^{2}(t) \beta a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)+(1-\hat{k}(t)) c_{S} \\
\dot{p}(t \mid \omega)=-p(t \mid \omega)(1-p(t \mid \omega)) \beta \hat{k}^{2}(t) a(t \mid \omega) \\
\dot{s}(t \mid \omega)=-\beta \hat{k}^{2}(t) s(t \mid \omega) a(t \mid \omega) \\
\dot{a}(t \mid \omega)=-\alpha \dot{s}(t \mid \omega)-\gamma_{a} a(t \mid \omega) \\
\mu(t, \omega)=\frac{\mu^{0}(\omega) /(1-p(t \mid \omega))}{\sum_{\omega^{\prime}} \mu^{0}\left(\omega^{\prime}\right) /\left(1-p\left(t \mid \omega^{\prime}\right)\right)}
\end{array}\right.
$$

and $\psi(T \mid \omega)=0$.
The strategy profile $\hat{\mathbf{k}}$ is a symmetric interior equilibrium if and only if $\hat{k}(t) \in(0,1)$ for all $t$.

Proof. See the Appendix.

When the confinement cost $c_{S}$ is large relatively to the continuation payoff in case of infection, individuals have less incentives to self-isolate. The next proposition gives a sufficient condition such that there is no self-isolation at all in equilibrium.

Proposition 3. [The no self-isolation equilibrium/ If $(1-\alpha) \alpha \beta v_{I}+c_{S}>0$, the game admits a unique equilibrium in dominant strategy, where all individuals play $\hat{k}(t)=1$ for every $t$. In this equilibrium, the players' payoff is

$$
E_{\mu^{0}}[v(\hat{k} \mid .)]=v_{I}(1-\alpha) \beta \int_{0}^{T} e^{-r t} \sum_{\omega} \mu^{0}(\omega) a(t \mid \omega) e^{-\int_{0}^{t} \beta a(s \mid \omega) d s} d t
$$

where, for each $\omega, a(. \mid \omega)$ is the unique solution of the system

$$
\left\{\begin{array}{l}
\forall t \in[0, T] \\
\dot{s}(t \mid \omega)=-\beta s(t \mid \omega) a(t \mid \omega), s(0 \mid \omega)=\bar{s} \\
\dot{a}(t \mid \omega)=-\alpha \dot{s}(t \mid \omega)-\gamma_{a} a(t \mid \omega), a(0 \mid \omega)=\bar{a}
\end{array}\right.
$$

Proof. See the Appendix.

## 4 The effect of uncertainty

In this section we explore the role of uncertainty on the epidemic dynamics in the simplest possible setting where the population is aware of two possible epidemics: $\Omega=\left\{\omega_{L}, \omega_{H}\right\}$, and has prior belief $\mu^{0}\left(\omega_{L}\right)=1-\mu^{0}\left(\omega_{H}\right)=\bar{\mu}$. To do so, we simulate the dynamics of each epidemic $\omega$ for several values of $\bar{\mu}$ when the population plays the symmetric equilibrium described in Proposition 2. We denote by $\hat{k}_{\bar{\mu}}$ the symmetric interior equilibrium strategy when the prior is $\bar{\mu}$. We compare them with the dynamics of each epidemic without uncertainty, i.e., when $\bar{\mu}=1$ in epidemic $\omega_{L}$, and $\bar{\mu}=0$ in epidemic $\omega_{H}$. We calibrate the epidemiological parameters $\beta, \gamma_{a}, \gamma_{s}$ and $\nu$ to the COVID-19 pandemic and we chose the behavioral parameters $c_{I}, c_{S}$ and $c_{D}$ arbitrarily.

### 4.1 Simulation strategy and calibration

In any epidemic, the first cases go unnoticed. For SARS-COV2, the first case was reported on December 11, 2019, whereas several phylodynamics studies date the onset of the epidemic between late August and early December. ${ }^{11}$ Therefore, 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 form beliefs about the epidemic state and adapt their behavior accordingly.

The system (12) is well defined for each $\omega$ by initial values $p(0 \mid \omega), a(0 \mid \omega)$ and $s(0 \mid \omega)$. However, the algorithm we construct to simulate (12) also requires the specification of $\psi(0 \mid \omega)$, which cannot be taken arbitrarily since $\psi$ is determined by the terminal condition $\psi(T \mid \omega)=0$. To determine $\psi(0 \mid \omega)$, we use an adaptation of the Simulated Annealing algorithm, a stochastic

[^6]search-based algorithm described by Lewis (2007), whose principle is to solve the system (12) for several possible values of $\psi(0 \mid \omega)$ and to select the one that minimizes the distance between $\psi(T \mid \omega)$ and 0 for each $\omega .^{12}$

In line with Fenichel et al. (2011), we set the discount rate to $\boldsymbol{r}=\mathbf{0 . 0 1 4 \%} \%{ }^{13}$. The epidemiological parameters are calibrated to the SARS-COV-2:

- Absent an exhaustive testing campaign, the proportion of asymptomatic types in the population is rather difficult to estimate. In a nationwide study of over 61000 participants, Pollan 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 $\boldsymbol{\alpha}=\mathbf{0 . 3}$.
- The recovery rate is usually estimated to two weeks, which implies $\gamma_{a}=\mathbf{1} / \mathbf{1 5} .{ }^{14}$
- To calibrate the contagiousness rate of the disease $\beta$, we use the value of the basic reproduction number $\mathcal{R}_{0}$, i.e., the average number of secondary infections produced by a typical infected individual in a population where everyone is susceptible. In our model, $\mathcal{R}_{0}=\beta \alpha / \gamma_{a}$. Indeed, as symptomatic individuals self-isolate, a randomly chosen infected individual contaminates a susceptible individual only if she is the asymptomatic type and if the virus is transmitted during contact, hence with probability $\alpha \beta$. As the individual is contagious during a period of expected length $1 / \gamma_{a}$, the average number of infections caused by an infected is $\beta \alpha / \gamma_{a}$. For SARS-COV-2, the estimation of $\mathcal{R}_{0}$ ranges between 2.5 and 3.5, thus we set $\mathcal{R}_{0}=3.2$, and therefore set $\boldsymbol{\beta}=\mathbf{3 . 2} \boldsymbol{\gamma}_{a} / \boldsymbol{\alpha}$.
- There are various estimates of the Fatality-Infected ratio in the epidemiological literature. For instance, Verity et al. (2020) estimate this ratio to $0.7 \%$ percent and

[^7]Gudbjartsson et al. (2020) to $0.3 \%$ We set a fatality rate $\boldsymbol{\nu} /\left(\boldsymbol{\nu}+\boldsymbol{\gamma}^{\boldsymbol{s}}\right)=\mathbf{0 . 5 \%} .{ }^{15}$
Finally, we arbitrarily set the costs to $\boldsymbol{c}_{S}=\mathbf{1}, \boldsymbol{c}_{\boldsymbol{I}}=\mathbf{1 0}$ and $\boldsymbol{c}_{D}=\mathbf{1 0 0}$.
On day $\boldsymbol{\tau}=\mathbf{2 0}$, the population is informed that a virus has been spreading since day 0 , and that a vaccine will be available on day $\boldsymbol{T}=\mathbf{3 5 0}$. Individuals do not know whether the initial penetration of the disease is low or high, that is if the epidemic is $\omega_{L}$ or $\omega_{H}$ with $a\left(0 \mid \omega_{L}\right)=0.1 \%$ and $a\left(0 \mid \omega_{H}\right)=0.5 \%$. They know that nobody has died or recovered from the disease yet, thus that $r(0 \mid \omega)=d(0 \mid \omega)=0$ and $s(0 \mid \omega)=1-\frac{1}{\alpha} a(0 \mid \omega)$ for each $\omega \in\left\{\omega_{L}, \omega_{H}\right\}$. We shall focus on the following epidemic indicators:

Total deaths. The total fraction of deaths in epidemic $\omega$ is $T D_{\bar{\mu}}(\omega):=\lim _{t \rightarrow \infty} d(t \mid \omega)$. By equation (5), $\int_{0}^{\infty} \dot{d}(t \mid \omega) d t+\bar{d}=\bar{d}+\nu \frac{1-\alpha}{\alpha} \int_{0}^{\infty} a(t \mid \omega) d t$. For all $t \geq T, \dot{s}(t \mid \omega)=0$, hence $\dot{a}(t \mid \omega)=-\gamma_{a} a(t \mid \omega)$ and $a(t \mid \omega)=a(T \mid \omega) e^{-\gamma_{a}(t-T)}$. It follows that $\int_{0}^{\infty} a(t \mid \omega) d t=\int_{0}^{T} a(t \mid \omega) d t+\frac{1}{\gamma_{a}} a(T \mid \omega)$, thus the total fraction of deaths in epidemic $\omega$ is

$$
T D_{\bar{\mu}}(\omega)=\bar{d}+\nu \frac{1-\alpha}{\alpha}\left(\int_{0}^{T} a(t \mid \omega) d t+\frac{1}{\gamma_{a}} a(T \mid \omega)\right) .
$$

Average transmission rate. The transmission rate of the disease is the rate at which a susceptible individual is contaminated. In average, the transmission rate in epidemic $\omega$ is $T R_{\bar{\mu}}(\omega):=\frac{1}{T} \int_{0}^{T} \beta a(t \mid \omega) \hat{k}_{\bar{\mu}}^{2}(t) d t$. By equation (1), $\beta a(t \mid \omega) \hat{k}_{\bar{\mu}}^{2}(t)=-\frac{\dot{s}(t \mid \omega)}{s(t \mid \omega)}$ hence

$$
T R_{\bar{\mu}}(\omega)=\frac{1}{T}(\ln (\bar{s})-\ln (s(T \mid \omega)) .
$$

Effective reproduction number The effective reproduction number is the expected proportion of the population contaminated by a randomly chosen infected individual. In our model, only asymptomatic individuals can effectively contaminate others, hence the effective reproduction number at time $t$ in epidemic $\omega$ is $E R N_{\bar{\mu}}(t \mid \omega)=\frac{1}{\gamma_{a}} \frac{a(t \mid \omega)}{a(t \mid \omega)+i(t \mid \omega)} \beta s(t \mid$ $\omega) \hat{k}_{\bar{\mu}}(t)^{2}$, which simplifies to $E R N(t \mid \omega)=\frac{1}{\gamma_{a}} \alpha \beta s(t \mid \omega) \hat{k}_{\bar{\mu}}(t)^{2}$.

[^8]
### 4.2 The dynamics of the epidemics and behaviors without uncertainty.

As a benchmark, we simulate the dynamics of both epidemics without uncertainty. Figure 1 exhibits the dynamics of the fraction of infected individuals at the equilibrium and the equilibrium social activity level $\hat{k}_{\bar{\mu}}(t)$ when individuals know the epidemic state (precisely, when $\bar{\mu}=1$ individuals know that epidemic state is $\omega_{L}$, and when $\bar{\mu}=0$ they know it is $\omega_{H}$ ).


Figure 1: Dynamics of each epidemic without uncertainty.

In both epidemics, the equilibrium social activity level drops to $\hat{k}_{1}(20)=0.042$ and $\hat{k}_{0}(20)=0.012$ right after the announcement. Afterwards, the level of social activity increases in both epidemics, first rapidly then at a slower pace, and remains smaller than 1 until the arrival of the vaccine.

The initial risk of infection is larger in $\omega_{H}$ than in $\omega_{L}$, this is why $\hat{k}_{0}(t)$ is smaller than $\hat{k}_{1}(t)$ at the beginning of the epidemic. Interestingly, after time 90, individuals self-isolate more in epidemic $\omega_{L}$ than in epidemic $\omega_{H}$. Let us explain why. The subjective probability of having symptoms depends on the fraction of infected individuals without symptoms $a(t \mid \omega)$ and the subjective belief of being the symptomatic type $p(t \mid \omega)$. Individuals are more confident in being of the asymptomatic type in epidemic $\omega_{H}$ (i.e., $p\left(t \mid \omega_{H}\right)<p\left(t \mid \omega_{L}\right)$ ), because, at the beginning, the virus circulated more intensively. Also, after date 100, the fraction of infected individuals with symptoms is higher in $\omega_{L}$. Therefore, the subjective probability of having symptoms is smaller $\omega_{H}$ after date 90 . Figure 2 illustrates this point.


Figure 2: Dynamics of the epidemic and the subjective belief without uncertainty after date 90 .

### 4.3 The dynamics of the epidemics and behaviors under uncertainty

We now simulate the dynamics of the two epidemics when the population has prior belief $\bar{\mu} \in\{0,0.25,1\}$ that the epidemic is $\omega_{L}$. As illustrated in Figure 3, uncertainty has important consequences on the spread of the disease in $\omega_{L}$ but not in $\omega_{H}$.

Interestingly, there is a second wave of infection in epidemic $\omega_{L}$ : for each prior $\bar{\mu} \in$ $\{0,0.25\}$, the proportion of infected decreases from $t=50$ to $t=150$ before increasing again. When $\bar{\mu}=0$, the peak of the second wave is reached at $t=325$ approximately. When $\bar{\mu}=0.25$, the peak has not been reached before $T$. This second wave arises because, after $t=90$, the social activity level is higher when $\bar{\mu} \in\{0,0.25\}$ than when individuals form correct anticipations on $\omega_{L}$. Note that the second wave is particularly high when agents (mistakenly) believe that the epidemic is $\omega_{H}$ with probability 1 (cf. the solid line), because they continuously increase their level of social activity. On the other hand, when $\bar{\mu}=0.25$, agents decrease their social activity after $t=100$ because they anticipate that the fraction of cases will increase again if $\omega=\omega_{L}$, which results in a flattening of the curve. Surprisingly, the second wave arises only in epidemic $\omega_{L}$. In epidemic $\omega_{H}$, uncertainty implies that individuals are more cautious than when they know that $\omega=\omega_{H}$ for sure, because without uncertainty they would have learned faster that they are likely to be the asymptomatic type and would thus have self-isolate less.

This result is consistent with the dynamics of the ERN, which is interpreted as the fraction of people an infected individual contaminates while infectious. Figure 4 suggests that without uncertainty individuals behave in a such a way the ERN is close to one from date 80 to the


Figure 3: Top right: Dynamics of the fraction of infected individuals in $\omega_{L}$ when $\bar{\mu} \in\{0,0.25,1\}$. Top left: Dynamics of the fraction of infected individuals in $\omega_{H}$ when $\bar{\mu} \in\{0,0.25,1\}$. Below: Dynamics of the equilibrium social activity level $\hat{k}(t)$.
end. For this reason, there is a single wave in this case. Under uncertainty, the ERN is above one after date 80 when the epidemic is $\omega_{L}$ : each infected individual contaminates more than many individuals, which explains why the infection cases increase after date 80. In epidemic $\omega_{H}$, the ERN stays below 1 under uncertainty, which is consistant with the fact that there is a single wave for every value of the prior $\mu_{0}$.

The average social activity level over time, defined as $\bar{k}_{\bar{\mu}}=\frac{1}{T} \int_{0}^{T} \hat{k}_{\bar{\mu}}(t) d t$ can be lower under uncertainty than when individuals are confident about the epidemic. For instance, when $\bar{\mu}=0.6, \bar{k}_{\bar{\mu}}=0.502$ while $\bar{k}_{\bar{\mu}}=0.534$ and $\bar{k}_{\bar{\mu}}=0.504$ when $\bar{\mu}=0$ and 1 respectively (see Figure 5).

In epidemic $\omega_{L}$, the average transmission rate is $T R_{\bar{\mu}}\left(\omega_{L}\right)=0.46$ when $\bar{\mu}=1$ and $T R_{\bar{\mu}}\left(\omega_{L}\right)=0.14$ when $\bar{\mu}=0$. This means that the disease is (much) more transmitted when the population wrongly believes that the epidemic is $\omega_{H}$. Figure 6 suggests that there


Figure 4: Dynamics of the ERN and average ERN in epidemics $\omega_{L}$ and $\omega_{H}$.


Figure 5: Average social activity at the symmetric equilibrium.
is a positive relationship between the average transmission rate and the prior in the epidemic $\omega_{L}$. In contrast, in state $\omega_{H}$ this relationship is non-monotonic in $\bar{\mu}$ : the average transmission rate is $T R_{\bar{\mu}}\left(\omega_{H}\right)=0.35$ when $\bar{\mu}=0.4$ and $T R_{\bar{\mu}}\left(\omega_{H}\right)=0.38$ and $T R_{\bar{\mu}}\left(\omega_{H}\right)=0.37$ in when $\bar{\mu}=0$ and $\bar{\mu}=1$, respectively.

### 4.4 The value of information

In this section we investigate whether it is always a good idea for a government to give the population all the information they have about the characteristic of an outbreak. We focus on two possible welfare objectives: minimizing the fraction of deaths and maximizing payoffs.

Figure 7 describes the total fraction of deaths for different values of $\bar{\mu}$ in each epidemic. In epidemic $\omega_{L}$, the fraction of deaths decreases with the prior belief $\bar{\mu}$ that the state is $\omega_{L}$. When individuals believe that the epidemic is $\omega_{H}$ with probability 1 (total delusion), the


Figure 6: Average transmission rate in epidemics $\omega_{H}$ and $\omega_{L}$.
fraction of deaths is twice higher than when they know that the state is $\omega_{L}$. In contrast, in epidemic $\omega_{H}$ the fraction of deaths decreases with uncertainty, since it is $0.112 \%$ when $\bar{\mu}=0.4$ and $0.12 \%$ and $0.115 \%$ when $\bar{\mu}=0$ and 1, respectively. In Figure 8 one can observe the same pattern for equilibrium payoffs: in epidemic $\omega_{L}$, the payoffs increase with $\bar{\mu}$, while in $\omega_{H}$, the payoffs are higher for $\bar{\mu}=0.4$ than for $\bar{\mu} \in\{0,1\}$. Therefore, transparency improves welfare - both in terms of deaths and payoffs - when the epidemic is $\omega_{L}$, while it decreases welfare when $\omega=\omega_{H}$.

What if the government has to commit to a disclosure policy before knowing the state of the epidemic?Recall that $T D_{\bar{\mu}}(\omega)$ is the fraction of deaths in epidemic $\omega$ when the prior is $\bar{\mu}$. The information value in terms of deaths is the fraction of deaths that can be avoided when the government disclose information. It depends on the prior $\bar{\mu}$ and is defined by:

$$
I V D_{\bar{\mu}}=-\underbrace{\bar{\mu} T D_{1}\left(\omega_{L}\right)+(1-\bar{\mu}) T D_{0}\left(\omega_{H}\right)}_{\text {Ex-ante fraction of deaths without uncertainty }}+\underbrace{\bar{\mu} T D_{\bar{\mu}}\left(\omega_{L}\right)+(1-\bar{\mu}) T D_{\bar{\mu}}\left(\omega_{H}\right)}_{\text {Expected fraction of deaths with uncertainty }}
$$

As one can see in Figure $7, I V D_{\bar{\mu}} \leq 0$ for every $\bar{\mu} \in(0,1)$, which suggests that, ex-ante, the value of information is negative when the objective is to reduce the fraction of deaths.

We now address the same question in terms of payoffs. Recall that $v\left(\hat{k}_{\bar{\mu}} \mid \omega\right)$ is the equilibrium payoff in $\omega$ when the prior is $\bar{\mu}$. The information value in terms of payoffs is ex-ante payoff gain from knowing whether the epidemic is $\omega_{L}$ or $\omega_{H}$. It also depends on the


Figure 7: Above: Total fraction of deaths in each epidemic. Below: information value in terms of deaths
prior $\bar{\mu}$ and is defined by:

$$
I V P_{\bar{\mu}}=\underbrace{\bar{\mu} v\left(\hat{k}_{1} \mid \omega_{L}\right)+(1-\bar{\mu}) v\left(\hat{k}_{0} \mid \omega_{H}\right)}_{\text {Ex-ante payoff without uncertainty }}-\underbrace{\bar{\mu} v\left(\hat{k}_{\bar{\mu}} \mid \omega_{L}\right)-(1-\bar{\mu}) v\left(\hat{k}_{\bar{\mu}} \mid \omega_{H}\right)}_{\text {Expected payoff with uncertainty }}
$$

In contrast with the information value in terms of deaths, $I V P_{\bar{\mu}}$ is positive for small values of $\bar{\mu}$ and negative for large values of $\bar{\mu}$, as depicted by Figure 8. Transparency seems to be ex-ante welfare improving for low prior probabilities but not otherwise.

## 5 The social planner problem

The problem of the social planner is to determine the level of social activity that maximizes the average payoff in the population over the infinite horizon. As the population is homogenous, we restrict the attention to symmetric profiles, i.e., such that $k_{j}(t)=k(t)$ for every $j$.

Let us determine the total average payoff of strategy $k$ conditional on some epidemic


Figure 8: Above: expected discounted payoff in epidemic $\omega_{L}$ and $\omega_{H}$. Below: Information Value in terms of Payoffs
$\omega$. After time $T$, every living individual without symptoms gets vaccinated and stops selfisolating, thereby gets payoff 0 . Sick individuals continue to bear the flow cost $c_{S}+c_{I}$ as long as they have symptoms, and dead individuals bear the cost $c_{D}$. Therefore, the average continuation payoff at $T$ conditional on $\omega$ is

$$
W(T \mid \omega)=\int_{T}^{\infty} e^{-r t}\left(-\left(c_{S}+c_{I}\right) i(t \mid \omega)-c_{D} d(t \mid \omega)\right) d t
$$

After the arrival of the vaccine at time $T$, the contagiousness rate of the disease drops to $\beta=0$, hence $\dot{s}(t \mid \omega)=0 \forall t \geq T$. Plugging this into (3) and (5) and integrating between $T$ and $t>T$, we obtain:

$$
\begin{aligned}
& i(t \mid \omega)=i(T \mid \omega) e^{-\gamma_{a}(t-T)} \\
& d(t \mid \omega)=d(T \mid \omega)+\frac{\nu}{\gamma_{a}} i(T \mid \omega)\left(1-e^{-\gamma_{a}(t-T)}\right)
\end{aligned}
$$

Plugging this into the latter expression, we obtain:

$$
\begin{equation*}
W(T \mid \omega)=-e^{-r T}\left(d(T \mid \omega) \frac{c_{D}}{r}+i(T \mid \omega) \frac{1}{r+\gamma_{a}}\left(\frac{c_{D}}{r} \nu+c_{S}+c_{I}\right)\right) \tag{13}
\end{equation*}
$$

At each time $t$ before the arrival of the vaccine, the population can be divided into four groups:

1. those who have never had symptoms before $t$, i.e., susceptible people, asymptomatic infected people and asymptomatic recovered people. They represent a total fraction $s(t \mid \omega)+\alpha(1-s(t \mid \omega))$ of the population and, as they spend a fraction $1-k(t)$ of their day home, bear the flow cost $c_{S}(1-k(t))$.
2. those who are infected with symptoms. They represent a fraction $i(t \mid \omega)$ of the population and bear the flow cost $c_{S}+c_{I}$.
3. those who had symptoms in the past and have healed from the disease before time $t$. They represent a fraction $(1-\alpha)(1-s(t \mid \omega))-i(t \mid \omega)-d(t \mid \omega)$ of the population and bear no cost as they do not self-isolate anymore.
4. those who died from the disease before $t$. They represent a fraction $d(t \mid \omega)$ of the population and bear the flow $\operatorname{cost} c_{D}$.

Therefore, the total average payoff conditional on $\omega$ is
$W(k \mid \omega)=W(T \mid \omega)+$

$$
\int_{0}^{T} e^{-r t}\left(-c_{S}(1-k(t))(\alpha+s(t \mid \omega)(1-\alpha))-\left(c_{S}+c_{I}\right) i(t \mid \omega)-c_{D} d(t \mid \omega)\right) d t
$$

Proposition 4 (The optimal strategy). The problem of the social planner has a unique symmetric solution $\check{k}$ defined by

$$
\check{k}(t)= \begin{cases}\tilde{k}(t) & \text { if } \tilde{k}(t) \in(0,1] \\ 1, & \text { otherwise }\end{cases}
$$

where

$$
\tilde{k}(t)=\frac{c_{S}}{2 \alpha \beta} \frac{\alpha+(1-\alpha) \sum_{\omega} \mu^{0}(\omega) s(t \mid \omega)}{\sum_{\omega} s(t \mid \omega) i(t \mid \omega)\left(\frac{1}{1-\alpha} \psi_{s}(t \mid \omega)-\psi_{i}(t \mid \omega)\right)}
$$

and $\forall t \in[0, T], \forall \omega \in \Omega$,
$\left\{\begin{array}{l}\dot{s}(t \mid \omega)=-\beta \check{k}(t)^{2} s(t \mid \omega) \frac{\alpha}{1-\alpha} i(t \mid \omega), \\ \dot{i}(t \mid \omega)=-(1-\alpha) \dot{s}(t \mid \omega)-\gamma_{a} i(t \mid \omega), \\ \dot{d}(t \mid \omega)=\nu i(t \mid \omega)\end{array}\right.$
$\left\{\begin{aligned} \dot{\psi}_{s}(t \mid \omega)-r \psi_{s}(t \mid \omega) & \left.=-\check{k}(t)^{2} \alpha \beta i(t \mid \omega)\left(\psi_{i}(t \mid \omega)-\frac{1}{1-\alpha} \psi_{s}(t \mid \omega)\right)+\mu^{0}(\omega) c_{S}(1-\alpha)(1-k(t))\right), \\ \dot{\psi}_{i}(t \mid \omega)-r \psi_{i}(t \mid \omega) & =-\check{k}(t)^{2} \alpha \beta s(t \mid \omega)\left(\psi_{i}(t \mid \omega)-\frac{1}{1-\alpha} \psi_{s}(t \mid \omega)\right)+\mu^{0}(\omega)\left(c_{S}+c_{I}+\gamma_{a} \psi_{i}(t \mid \omega)-\nu \psi_{d}(t \mid \omega)\right), \\ \dot{\psi}_{d}(t \mid \omega)-r \psi_{d}(t \mid \omega) & =\mu^{0}(\omega) c_{D},\end{aligned}\right.$
and $\psi_{s}(T \mid \omega)=0, \psi_{i}(T \mid \omega)=-\mu^{0}(\omega) \frac{1}{r+\gamma_{a}}\left(\frac{c_{D}}{r} \nu+c_{S}+c_{I}\right)$, and $\psi_{d}(T \mid \omega)=-\mu^{0}(\omega) \frac{c_{D}}{r}$.
Proof. See the Appendix.

## Appendix

### 5.1 Detailed derivation of 9

Fix $\omega \in \Omega$ and a strategy $k_{j}$ for player $j$. If player $j$ gets symptoms at time $\tau$, her payoff is:

$$
\begin{array}{ll}
e^{-r \tau} v_{I}-\int_{0}^{\tau} e^{-r s}\left(1-k_{j}(s)\right) c_{S} d s & \text { if } \tau \leq T \\
-\int_{0}^{T} e^{-r s}\left(1-k_{j}(s)\right) c_{S} d s & \text { if } \tau>T
\end{array}
$$

As $\tau$ is a random variable, player $j$ 's expected payoff is:

$$
v\left(k_{j} \mid \omega\right)=E\left[\left(e^{-r \tau} v_{I}-u(\tau)\right) \mathbb{1}_{\tau \leq T}\right]-u(T) P(\tau>T)
$$

with $u(t):=\int_{0}^{t} e^{-r s}\left(1-k_{j}(s)\right) c_{S} d s$.
For $t \leq T, P(\tau>t)=1-p_{0}+p_{0} e^{-\int_{0}^{t} k_{j}(s) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s}$, which, by integrating (6), simplifies to

$$
P(\tau>t)=e^{-\int_{0}^{t} p_{j}(s \mid \omega) k_{j}(s) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s}
$$

and implies that $\tau$ is distributed with density

$$
f_{\tau}(t)=p_{j}(t \mid \omega) k_{j}(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega) P(\tau>t)
$$

Therefore,

$$
\begin{aligned}
v\left(k_{j} \mid \omega\right) & =\int_{0}^{T}\left(e^{-r t} v_{I}-u(t)\right) f_{\tau}(t) d t-u(T) P(\tau>T) \\
& =\int_{0}^{T} e^{-r t} v_{I} f_{\tau}(t) d t-\int_{0}^{T} u(t) f_{\tau}(t) d t-u(T) P(\tau>T)
\end{aligned}
$$

Integrating by parts and simplifying, we obtain:

$$
\begin{aligned}
v\left(k_{j} \mid \omega\right) & =\int_{0}^{T} e^{-r \tau} v_{I} f_{\tau}(t) d t-[u(t) P(\tau \leq t)]_{0}^{T}+\int_{0}^{T} u^{\prime}(t) P(\tau \leq t) d t-u(T) P(\tau>T) \\
& =\int_{0}^{T} e^{-r \tau} v_{I} f_{\tau}(t) d t-u(T) P(\tau \leq T)+\int_{0}^{T} e^{-r t}\left(1-k_{j}(t)\right) c_{S} P(\tau \leq t) d t-u(T) P(\tau>T) \\
& =\int_{0}^{T} e^{-r \tau} v_{I} f_{\tau}(t) d t-u(T)+\int_{0}^{T} e^{-r t}\left(1-k_{j}(t)\right) c_{S}(1-P(\tau>t)) d t \\
& =\int_{0}^{T} e^{-r \tau} v_{I} f_{\tau}(t) d t-u(T)+u(T)-\int_{0}^{T} e^{-r t}\left(1-k_{j}(t)\right) c_{S} P(\tau>t) d t \\
& =\int_{0}^{T} e^{-r \tau} P(\tau>t)\left[v_{I} p_{j}(t \mid \omega) k_{j}(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)-\left(1-k_{j}(t)\right) c_{S}\right] d t
\end{aligned}
$$

### 5.2 Proofs for Section 2 and Section 3

Lemma 1. For every $\omega \in \Omega, t \in \mathbb{R}_{+}$,

$$
\mu_{j}(t, \omega)=\frac{\mu^{0}(\omega) /\left(1-p_{j}(t \mid \omega)\right)}{\sum_{\omega^{\prime}} \mu^{0}\left(\omega^{\prime}\right) /\left(1-p_{j}\left(t \mid \omega^{\prime}\right)\right)}
$$

Proof. Let $S_{j}(t)$ stands for the event " $j$ has symptoms in $t$ " and $\bar{S}_{j}\left(t^{-}\right)$for the event " $j$ never had symptoms before $t^{\prime \prime}$. By definition, $\mu_{j}(t, \omega)=P\left(\omega \mid \bar{S}_{j}\left(t^{-}\right)\right)$. By Bayes's rule,

$$
P\left(\omega \mid \bar{S}_{j}\left(t^{-}\right)\right)=\frac{P\left(\bar{S}_{j}\left(t^{-}\right) \mid \omega\right) P(\omega)}{P\left(\bar{S}_{j}\left(t^{-}\right)\right)}=\frac{P\left(\bar{S}_{j}\left(t^{-}\right) \mid \omega\right) \mu^{0}(\omega)}{\sum_{\omega^{\prime}} P\left(\bar{S}_{j}\left(t^{-}\right) \mid \omega^{\prime}\right) \mu^{0}\left(\omega^{\prime}\right)} .
$$

As individuals of type $\theta_{a}$ never have symptoms,

$$
P\left(\bar{S}_{j}\left(t^{-}\right) \mid \omega\right)=1-P\left(\theta_{s}\right)+P\left(\bar{S}_{j}\left(t^{-}\right) \mid \omega, \theta_{s}\right) P\left(\theta_{s}\right)
$$

As $P\left(\theta_{s}\right)=1-\alpha$ and $P\left(\bar{S}_{j}\left(t^{-}\right) \mid \omega, \theta_{s}\right)=e^{-\int_{0}^{t} k_{j}(s) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s}$, we can write:

$$
P\left(\bar{S}_{j}\left(t^{-}\right) \mid \omega\right)=\alpha+(1-\alpha) e^{-\int_{0}^{t} k_{j}(s) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s} .
$$

Moreover, integrating (6) between 0 and $t$, we obtain:

$$
\left\{\begin{array}{l}
p(0 \mid \omega) e^{-\int_{0}^{t} k_{j}(s) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s}=p_{j}(t \mid \omega) e^{-\int_{0}^{t} k_{j}(s) p(s \mid \omega) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s} \\
e^{-\int_{0}^{t} k_{j}(s) p(s \mid \omega) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s}=\frac{1-p(0 \mid \omega)}{1-p_{j}(t \mid \omega)}
\end{array}\right.
$$

Using the latter identities together with $p(0 \mid \omega)=1-\alpha$, we obtain:

$$
P\left(\bar{S}_{j}\left(t^{-}\right) \mid \omega\right)=\frac{\alpha}{1-p_{j}(t \mid \omega)} .
$$

The result follows.

Lemma 2. Let $\tau_{H}$ and $\tau_{D}$ be independent random variables distributed according to $f(t)=$ $\gamma e^{-\gamma t}$ and $f(t)=\nu e^{-\nu t}$, respectively. The following equality holds:

$$
E\left[\int_{0}^{\min \left\{\tau_{H}, \tau_{D}\right\}} e^{-r t}\left(c_{S}+c_{I}\right) d t+\frac{c_{D}}{r} e^{-r \tau_{D}} \mathbb{1}_{\tau_{D}<\tau_{H}}\right]=\frac{1}{r+\gamma+\nu}\left(c_{S}+c_{I}+\nu \frac{c_{D}}{r}\right) .
$$

Proof. Let $g\left(\tau_{H}, \tau_{D}\right):=\int_{0}^{\min \left\{\tau_{H}, \tau_{D}\right\}} e^{-r t}\left(c_{S}+c_{I}\right) d t+\frac{c_{D}}{r} e^{-r \tau_{D}} \mathbb{1}_{\tau_{D}<\tau_{H}}$. Straightforwardly,

$$
g\left(\tau_{H}, \tau_{D}\right)=\frac{c_{S}+c_{I}}{r}\left(1-e^{-r \min \left\{\tau_{H}, \tau_{D}\right\}}\right)+\frac{c_{D}}{r} e^{-r \tau_{D}} \mathbb{1}_{\tau_{D}<\tau_{H}} .
$$

The random variable $\min \left\{\tau_{H}, \tau_{D}\right\}$ is distributed according to $f(t)=(\gamma+\nu) e^{-(\gamma+\nu) t}$. Therefore,

$$
E\left[e^{-r \min \left\{\tau_{H}, \tau_{D}\right\}}\right]=\frac{\gamma+\nu}{r+\gamma+\nu} .
$$

Moreover,

$$
E\left[e^{-r \tau_{D}} \mathbb{1}_{\tau_{D}<\tau_{H}}\right]=\int_{0}^{\infty}\left(\int_{0}^{\tau_{H}} e^{-(r+\nu) \tau_{D}} \nu d \tau_{D}\right) \gamma e^{-\gamma \tau_{H}} d \tau_{H}=\frac{\nu}{r+\gamma+\nu}
$$

Therefore,

$$
E\left[g\left(\tau_{H}, \tau_{D}\right)\right]=\left(c_{S}+c_{I}\right) \frac{1}{r+\gamma+\nu}+\frac{c_{D}}{r} \frac{\nu}{r+\gamma+\nu} .
$$

## Proof of Proposition 1

The best-response problem of a player is to determine the strategy $k$ that maximizes her expected discounted payoff, with, for every $\omega \in \Omega$, the functions $s(. \mid \omega)$ and $a(. \mid \omega)$ being fixed and defined by the dynamic system:

$$
\left\{\begin{array}{l}
\forall t \in[0, T] \\
\dot{s}(t \mid \omega)=-\beta \bar{k}_{S}(t \mid \omega) s(t \mid \omega) \bar{k}_{A}(t \mid \omega) a(t \mid \omega), \text { with } s(0 \mid \omega)=\bar{s} \in(0,1), \\
\dot{a}(t \mid \omega)=-\alpha \dot{s}(t \mid \omega)-\gamma_{a} a(t \mid \omega), \text { with } a(0 \mid \omega)=\bar{a} \in(0,1)
\end{array}\right.
$$

Formally, it is the solution of the optimal control problem
$\begin{cases}\max _{k \in \mathcal{K}} & \int_{0}^{T} e^{-r t} \sum_{\omega} \mu^{0}(\omega) e^{-\int_{0}^{t} p(s \mid \omega) k(s) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s}\left(p(t \mid \omega) k(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega) v_{I}-c_{S}(1-k(t))\right) d t \\ \text { w.r.t. } & \dot{p}(t \mid \omega)=-p(t \mid \omega)(1-p(t \mid \omega)) k(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega) \text { and } p(0 \mid \omega)=1-\alpha \forall \omega \in \Omega,\end{cases}$
where $\mathcal{K}$ denotes the set of piecewise continuous functions from $\mathbb{R}_{+}$into $[0,1]$. Making the change of variable $x(t \mid \omega):=\mu^{0}(\omega) e^{-\int_{0}^{t} p(s \mid \omega) k(s) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s}$, with $X(t):=(x(t \mid \omega))_{\omega}$, and observing that ${ }^{16}$

$$
e^{-\int_{0}^{t} p(s \mid \omega) k(s) \beta \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s}=\frac{\alpha}{1-p(t \mid \omega)}
$$

the player's problem can be rewritten as follows:
$\mathcal{P}(\mathbf{k}): \begin{cases}\max _{k \in \mathcal{K}} & \int_{0}^{+\infty} e^{-r t} F(t, X(t), k(t)) d t \\ \text { w.r.t. } & \dot{x}(t \mid \omega)=-\left(x(t \mid \omega)-\mu^{0}(\omega) \alpha\right) k(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega) \text { and } x(0 \mid \omega)=\mu^{0}(\omega) \forall \omega \in \Omega .\end{cases}$
with

$$
F(t, X(t), k(t)):=\sum_{\omega}\left[\left(x(t \mid \omega)-\mu^{0}(\omega) \alpha\right) k(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega) v_{I}-x(t \mid \omega) c_{S}(1-k(t))\right]
$$

As $F(t, X(t), k(t))$ is negative and bounded below by $v_{I}$, the objective is well defined. Furthermore, by standard results, the problem admits at least one solution. Applying Pontryagin's

[^9]maximum principle, the optimal control $k^{*}$ and the associated trajectory $X^{*}$ must satisfy the following conditions:

Lemma 3 (Necessary conditions). If $\left(X^{*}, k^{*}\right)$ is a solution of $\mathcal{P}(\mathbf{k})$, then there exists $a$ function $\psi: \mathbb{R}_{+} \times \Omega \rightarrow \mathbb{R}, C^{1}$ in the first argument, such that:
(i) $\forall \omega, \dot{\psi}(t \mid \omega)-r \psi(t \mid \omega)=-H_{x(t \mid \omega)}\left(t, X^{*}(t), k^{*}(t), \Psi(t)\right)$,
(ii) $H\left(t, X^{*}(t), k(t), \Psi(t)\right) \leq H\left(t, X^{*}(t), k^{*}(t), \Psi(t)\right)$ for every admissible control $k$,
$($ iii) $\forall \omega, \psi(T \mid \omega)=0$,
where $\Psi(t):=(\psi(t \mid \omega))_{\omega}$ and $H(t, X(t), k(t), \Psi(t)):=F(t, X(t), k(t))+\sum_{\omega} \psi(t \mid \omega) \dot{x}(t \mid \omega)$ is the discounted Hamiltonian of the problem.

The transversality condition (iii) comes from the fact that $x(T \mid \omega)$ is free for every $\omega$.
Observing that
$H(t, X(t), k(t), \Psi(t))=$

$$
\sum_{\omega}\left[\left(x(t \mid \omega)-\mu^{0}(\omega) \alpha\right) k(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(v_{I}-\psi(t \mid \omega)\right)-x(t \mid \omega) c_{S}(1-k(t))\right]
$$

the necessary conditions are rewritten as
(i) $\forall \omega, \dot{\psi}(t \mid \omega)-r \psi(t \mid \omega)=k^{*}(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)+c_{S}\left(1-k^{*}(t)\right)$,
(ii) for every admissible control $k$,

$$
\left(k^{*}(t)-k(t)\right) \sum_{\omega}\left[x^{*}(t \mid \omega) c_{S}-\left(x^{*}(t \mid \omega)-\mu^{0}(\omega) \alpha\right) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)\right] \geq 0
$$

As $x(t \mid \omega)=\mu^{0}(\omega) \alpha /(1-p(t \mid \omega))$, the latter condition can be more conveniently rewritten as:
(ii) $\left(k^{*}(t)-k(t)\right) \sum_{\omega} \frac{\mu^{0}(\omega)}{1-p(t \mid \omega)}\left[c_{S}-p^{*}(t \mid \omega) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)\right] \geq 0 \forall k$.

Dividing by $\sum_{\omega} \mu^{0}(\omega) /(1-p(t \mid \omega))$ and using the identity $\mu(t, \omega)=\frac{\mu^{0}(\omega)(1-p(t \mid \omega))}{\sum_{\omega^{\prime}} \mu^{0}\left(\omega^{\prime}\right)\left(1-p\left(t \mid \omega^{\prime}\right)\right)}$, condition (ii) reduces to:
$(i i) k^{*}(t)= \begin{cases}1, & \text { if } c_{S}-\sum_{\omega} \mu(t, \omega) p^{*}(t \mid \omega) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)>0, \\ 0, & \text { if } c_{S}-\sum_{\omega} \mu(t, \omega) p^{*}(t \mid \omega) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)<0, \\ \in[0,1], & \text { if } c_{S}-\sum_{\omega} \mu(t, \omega) p^{*}(t \mid \omega) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)=0 .\end{cases}$

## Proof of Proposition 2

For all $t$; consider the strategy profile in which $k_{j^{\prime}}(t)=\hat{k}(t) \forall j^{\prime} \neq j, t$ and $k_{j}(t)$ is arbitrary. Suppose that $\hat{k}(t) \in(0,1)$. Replacing $\hat{k}(t)$ in the Hamiltonian, we obtain: $H\left(t, X(t), k_{j}(t), \Psi(t)\right)=-\sum_{\omega} x(t \mid \omega) c_{S}$. It is easy to see that the Hamiltonian is concave with respect to the state variable $X(t)$ and therefore the necessary conditions in Proposition 1 are also sufficient (see e.g. Arrow and Kurz (1970)). Therefore, any $k_{j}(t) \in(0,1)$ is a best response (and in particular $\hat{k}(t)$ since it belongs to $(0,1)$ ). Consequently, $\hat{\mathbf{k}}(t)$ is an equilibrium.

## Proof of Proposition 3

As a preliminary, let us prove the following lemma:

Lemma 4. If $\psi: \mathbb{R}_{+} \times \Omega \rightarrow \mathbb{R}$ satisfies the necessary conditions of Proposition 1 , then $\psi(t \mid \omega) \leq 0$ for every $t \in[0, T]$ and $\omega \in \Omega$.

Proof. Let $\psi: \mathbb{R}_{+} \times \Omega \rightarrow \mathbb{R}$ such that, for every $t$ and $\omega$,

$$
\begin{equation*}
\dot{\psi}(t \mid \omega)-r \psi(t \mid \omega)=k(t) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)+c_{S}(1-k(t)) \tag{14}
\end{equation*}
$$

and $\psi(T \mid \omega)=0$.
We work towards a contradiction. Fix some epidemic $\omega$. Suppose that there exists $t^{\prime}<t^{\prime \prime} \leq T$ such that $\psi(t \mid \omega)>0$ for every $t \in\left[t^{\prime}, t^{\prime \prime}\right]$. It follows that $\psi(t \mid \omega)-v_{I}>0$ on $\left[t^{\prime}, t^{\prime \prime}\right]$, thus $\psi^{\prime}(t \mid \omega)>0$ on $\left[t^{\prime}, t^{\prime \prime}\right]$ by (14). As a consequence, $\psi(t \mid \omega)>0$ in the right neighborhood of $t^{\prime \prime}$, which implies $\psi^{\prime}(t \mid \omega)$ is increasing on the right neighborhood of $t^{\prime \prime}$. The argument can be extended to prove that $\psi(t \mid \omega)>0$ and $\psi^{\prime}(t \mid \omega)>0$ for every $t \in\left[t^{\prime}, T\right]$. This contradicts $\psi(T \mid \omega)=0$.

Fix a player $i$, a date $t$ and a value $\bar{k}_{A}(t \mid \omega)$ for each $\omega$. As $\psi(t \mid \omega)<0$ by Lemma $4, \psi(t \mid \omega)-v_{I}<-v_{I}$. Moreover, $\bar{k}_{A}(t \mid \omega) \leq 1$ and $a(t \mid \omega)<\alpha$. Finally, $p(t \mid \omega)$ is non
increasing in $t$, thus $p(t \mid \omega) \leq 1-\alpha$. Therefore, for every $t$,

$$
\begin{aligned}
\beta \sum_{\omega} \mu(t, \omega) p(t \mid \omega) \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right) & <\sum_{\omega} \mu(t, \omega) p(t \mid \omega) \beta \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(-v_{I}\right), \\
& <\left(-v_{I}\right) \sum_{\omega} \mu(t, \omega)(1-\alpha) \beta \alpha, \\
& <\left(-v_{I}\right)(1-\alpha) \beta \alpha .
\end{aligned}
$$

Suppose that the best response is such that, at some date $t, k_{i}^{*}(t)<1$. If $(1-\alpha) \alpha \beta\left(-v_{I}\right)<$ $c_{S}$ then $\beta \sum_{\omega} \mu(t, \omega) p(t \mid \omega) \bar{k}_{A}(t \mid \omega) a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)$ is smaller then $c_{H}$. According to Proposition ??, $k_{i}^{*}(t)<1$ cannot be a best response. Thus a contradiction. Therefore, $k_{i}^{*}(t)=1$ for all $t$ is a dominant strategy. This proves the result.

Finally, let us determine the players' payoff in the equilibrium where $k^{*}=1$. Plugging $k_{j}(t)=1$ and $\bar{k}_{A}(t \mid \omega)=1$ into the belief dynamics (3), we obtain the players' belief function conditional on $\omega$ as the solution of the ODE:

$$
\dot{p}(t \mid \omega)=-p(t \mid \omega)(1-p(t \mid \omega)) \beta a(t \mid \omega)
$$

with initial condition $p(0 \mid \omega)=1-\alpha$. Integrating between 0 and $t$, we obtain

$$
\frac{p(t \mid \omega)}{1-p(t \mid \omega)}=\frac{1-\alpha}{\alpha} e^{-\int_{0}^{t} \beta a(u \mid \omega) d u}
$$

and

$$
\frac{\alpha}{1-p(t \mid \omega)}=e^{-\int_{0}^{t} \beta p(u \mid \omega) a(u \mid \omega) d u}
$$

Using the latter findings and plugging $k_{j}(t)=1$ into the payoff expression (5) then simplifying, we obtain:

$$
v\left(k^{*} \mid \omega\right)=v_{I}(1-\alpha) \beta \int_{0}^{T} e^{-r t} a(t \mid \omega) e^{-\beta \int_{0}^{t} \beta a(u \mid \omega) d u} d t
$$

The result is obtained by taking the expectation of the latter expression.

### 5.3 Proofs for Section 5

## Proof of Proposition 4.

The problem of the social planner is to determine the strategy that maximizes $E[W(k \mid)$. subject to the evolution of the epidemic. As $a(t \mid \omega)=\frac{\alpha}{1-\alpha} i(t \mid \omega)$, the problem depends only on $3 \times|\Omega|$ state variables represented by $X(t):=\left((s(t \mid \omega))_{\omega},(i(t \mid \omega))_{\omega},(d(t \mid \omega))_{\omega}\right)$. Formally, it is the solution of the optimal control problem

$$
\mathcal{P}^{W}(k): \begin{cases}\max _{k \in \mathcal{K}} & \int_{0}^{T} e^{-r t} F(t, X(t), k(t)) d t+E\left[W_{T}(k \mid .)\right] \\ w . r . t ., \forall \omega \in \Omega, & \dot{s}(t \mid \omega)=-\beta k(t)^{2} s(t \mid \omega) \frac{\alpha}{1-\alpha} i(t \mid \omega), s(0 \mid \omega)=\bar{s} \\ & \dot{i}(t \mid \omega)=-(1-\alpha) \dot{s}(t \mid \omega)-\gamma_{a} i(t \mid \omega), \quad i(0 \mid \omega)=\bar{i} \\ & \dot{d}(t \mid \omega)=\nu i(t \mid \omega), d(0 \mid \omega)=\bar{d}\end{cases}
$$

with

$$
F(t, X(t), k(t)):=\sum_{\omega} \mu^{0}(\omega)\left[-c_{S}(1-k(t))(\alpha+(1-\alpha) s(t \mid \omega))-\left(c_{S}+c_{I}\right) i(t \mid \omega)-c_{D} d(t \mid \omega)\right]
$$

As $F(t, X(t), k(t))$ is negative and bounded below, the objective is well defined. Furthermore, by standard results, the problem admits at least one solution. Applying Pontryagin's maximum principle, the optimal control $k^{*}$ and the associated trajectory $X^{*}$ must satisfy the following conditions:

Lemma 5 (Necessary conditions). If $\left(X^{*}, k^{*}\right)$ is a solution of $\mathcal{P}^{W}(k)$, then there exist functions $\psi_{s}, \psi_{i}, \psi_{d}: \mathbb{R}_{+} \times \Omega \rightarrow \mathbb{R}, C^{1}$ in the first argument, such that:
(i) $\forall \omega \in \Omega$,

$$
\begin{aligned}
& \dot{\psi}_{s}(t \mid \omega)-r \psi_{s}(t \mid \omega)=-H_{s(t \mid \omega)}\left(t, X^{*}(t), k^{*}(t), \Psi(t)\right) \\
& \dot{\psi}_{i}(t \mid \omega)-r \psi_{i}(t \mid \omega)=-H_{i(t \mid \omega)}\left(t, X^{*}(t), k^{*}(t), \Psi(t)\right) \\
& \dot{\psi}_{d}(t \mid \omega)-r \psi_{d}(t \mid \omega)=-H_{d(t \mid \omega)}\left(t, X^{*}(t), k^{*}(t), \Psi(t)\right)
\end{aligned}
$$

(ii) $H\left(t, X^{*}(t), k(t), \Psi(t)\right) \leq H\left(t, X^{*}(t), k^{*}(t), \Psi(t)\right)$ for every admissible control $k$,
(iii) $\forall \omega \in \Omega, \psi_{s}(T \mid \omega)=\frac{\partial E[W(T \mid .)]}{\partial s(T \mid \omega)}, \psi_{i}(T \mid \omega)=\frac{\partial E[W(T \mid .)]}{\partial i(T \mid \omega)}, \psi_{d}(T \mid \omega)=\frac{\partial E[W(T \mid .)]}{\partial d(T \mid \omega)}$, where $\Psi(t):=\left(\left(\psi_{s}(t \mid \omega)\right)_{\omega},\left(\psi_{i}(t \mid \omega)\right)_{\omega},\left(\psi_{d}(t \mid \omega)\right)_{\omega}\right)$ and $H(t, X(t), k(t), \Psi(t)):=F(t, X(t), k(t))+\sum_{\omega}\left(\psi_{s}(t \mid \omega) \dot{s}(t \mid \omega)+\psi_{i}(t \mid \omega) \dot{i}(t \mid \omega)+\psi_{d}(t \mid \omega) \dot{d}(t \mid \omega)\right)$ is the discounted Hamiltonian of the problem.

After some simplifications, we observe that
$H(t, X(t), k(t), \Psi(t))=-\alpha c_{S}$

$$
\begin{aligned}
& +k(t)^{2} \alpha \beta \sum_{\omega} s(t \mid \omega) i(t \mid \omega)\left(\psi_{i}(t \mid \omega)-\frac{1}{1-\alpha} \psi_{s}(t \mid \omega)\right)+k(t) c_{S}\left(\alpha+(1-\alpha) \sum_{\omega} \mu^{0}(\omega) s(t \mid \omega)\right) \\
& +\sum_{\omega} \mu^{0}(\omega)\left[-c_{S}(1-\alpha) s(t \mid \omega)-\left(c_{S}+c_{I}\right) i(t \mid \omega)-c_{D} d(t \mid \omega)\right]+i(t \mid \omega)\left[\nu \psi_{d}(t \mid \omega)-\gamma_{a} \psi_{i}(t \mid \omega)\right]
\end{aligned}
$$

If $Z:=\sum_{\omega} s(t \mid \omega) i(t \mid \omega)\left(\psi_{i}(t \mid \omega)-\frac{1}{1-\alpha} \psi_{s}(t \mid \omega)\right)>0$, then $H(t, X(t), k(t), \Psi(t))$ is increasing in $k(t)$, thus $k^{*}(t)=1$. If $Z<0$, then $H(t, X(t), k(t), \Psi(t))$ is concave in $k(t)$, hence a candidate for $k^{*}$ is the solution of $\frac{\partial H(.)}{\partial k(t)}=0$, i.e.,

$$
\tilde{k}(t)=-c_{S} \frac{\alpha+(1-\alpha) \sum_{\omega} \mu^{0}(\omega) s(t \mid \omega)}{2 \alpha \beta Z}
$$

As $Z<0, \tilde{k}(t)>0$. If $\tilde{k}(t)>1$, then $k^{*}(t)=1$. If $\tilde{k}(t)<1$, then $k^{*}(t)=\tilde{k}(t)$. Therefore,

$$
k^{*}(t)= \begin{cases}\tilde{k}(t), & \text { if } \tilde{k}(t) \in[0,1] \\ 1, & \text { otherwise }\end{cases}
$$

Proceeding as in the proof of Proposition 2, we obtain:

$$
\begin{aligned}
& \left.\dot{\psi}_{s}(t \mid \omega)-r \psi_{s}(t \mid \omega)=-k(t)^{2} \alpha \beta i(t \mid \omega)\left(\psi_{i}(t \mid \omega)-\frac{1}{1-\alpha} \psi_{s}(t \mid \omega)\right)+\mu^{0}(\omega) c_{S}(1-\alpha)(1-k(t))\right) \\
& \dot{\psi}_{i}(t \mid \omega)-r \psi_{i}(t \mid \omega)=-k(t)^{2} \alpha \beta s(t \mid \omega)\left(\psi_{i}(t \mid \omega)-\frac{1}{1-\alpha} \psi_{s}(t \mid \omega)\right)+\mu^{0}(\omega)\left(c_{S}+c_{I}+\gamma_{a} \psi_{i}(t \mid \omega)-\nu \psi_{d}(t \mid \omega)\right) \\
& \dot{\psi}_{d}(t \mid \omega)-r \psi_{d}(t \mid \omega)=\mu^{0}(\omega) c_{D}
\end{aligned}
$$

Finally, the transversality conditions are, $\forall \omega \in \Omega$ :

$$
\begin{aligned}
& \psi_{s}(T \mid \omega)=0 \\
& \psi_{i}(T \mid \omega)=-\mu^{0}(\omega) e^{-r T} \frac{1}{r+\gamma_{a}}\left(\frac{c_{D}}{r} \nu+c_{S}+c_{I}\right) \\
& \psi_{d}(T \mid \omega)=-\mu^{0}(\omega) e^{-r T} \frac{c_{D}}{r}
\end{aligned}
$$

## 6 More uncertainty

The epidemic is characterized by two features:

1) the initial epidemic penetration $(\bar{s}, \bar{i}, \bar{a}, \bar{r}, \bar{d})$, where $\bar{s} \in[0,1]$ is the proportion of individuals who are not immune to the disease at time $0, \bar{i} \in[0,1]$ is the proportion of individuals infected with symptoms at time $0, \bar{a} \in[0,1]$ is the proportion of individuals infected without symptoms
at time $0, \bar{r} \in[0,1]$ is the proportion of individuals who already recovered from the disease at time 0 and are now immune to it and $\bar{d}=1-\bar{s}-\bar{i}-\bar{a}-\bar{r} \in[0,1]$ the proportion of dead individuals at time 0;
2) the medical parameters of the disease $\left(\alpha, \beta, \gamma_{a}, \gamma_{s}, \nu\right)$, where $\alpha \in(0,1)$ is the proportion of asymptomatic types in the population, $\beta>0$ is the contagiousness rate, $\gamma_{a}>0$ is the recovery rate of asymptomatic types, and $\gamma_{s}>0$ and $\nu>0$ are the recovery and death rates of symptomatic types, respectively.

The tuple $\omega=\left\{\bar{s}, \bar{i}, \bar{a}, \bar{r}, \bar{d}, \alpha, \beta, \gamma_{a}, \gamma_{s}, \nu\right\}$ is the epidemic state, and we denote by $\Omega$ the finite set of possible epidemic states.

The evolution of the epidemics is now:

$$
\begin{align*}
\dot{s}(t \mid \omega) & =-\beta^{\omega} \bar{k}_{S}(t \mid \omega) s(t \mid \omega) \bar{k}_{A}(t \mid \omega) a(t \mid \omega)  \tag{15}\\
\dot{a}(t \mid \omega) & =-\alpha^{\omega} \dot{s}(t \mid \omega)-\gamma_{a}^{\omega} a(t \mid \omega)  \tag{16}\\
\dot{i}(t \mid \omega) & =-\left(1-\alpha^{\omega}\right) \dot{s}(t \mid \omega)-\left(\gamma_{s}^{\omega}+\nu^{\omega}\right) i(t \mid \omega)  \tag{17}\\
\dot{r}(t \mid \omega) & =\gamma_{a}^{\omega} a(t \mid \omega)+\gamma_{s}^{\omega} i(t \mid \omega)  \tag{18}\\
\dot{d}(t \mid \omega) & =\nu^{\omega} i(t \mid \omega) \tag{19}
\end{align*}
$$

The discounted expected payoff conditional on the epidemic being $\omega$ is thus:
$v\left(k_{j} \mid \omega\right)=\int_{0}^{T} e^{-r t} e^{-\int_{0}^{t} p_{j}(s \mid \omega) k_{j}(s) \beta^{\omega} \bar{k}_{A}(s \mid \omega) a(s \mid \omega) d s}\left(p_{j}(t \mid \omega) k_{j}(t) \beta^{\omega} \bar{k}_{A}(t \mid \omega) a(t \mid \omega) v_{I}^{\omega}-c_{S}\left(1-k_{j}(t)\right)\right) d t$.
where:

$$
\begin{equation*}
v_{I}^{\omega}=-\frac{1}{r+\gamma_{s}^{\omega}+\nu}\left(c_{S}+c_{I}+\nu^{\omega} \frac{c_{D}}{r}\right) \tag{20}
\end{equation*}
$$

The symmetric equilibrium is given by:

Proposition 5 (The symmetric equilibrium). The game has a unique symmetric equilibrium where all individuals play $\hat{k}$ defined by

$$
\hat{k}(t)=\min \left\{\frac{c_{S}}{\beta^{\omega} \sum_{\omega} \mu(t, \omega) p(t \mid \omega) a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)}, 1\right\}
$$

where

$$
\left\{\begin{array}{l}
\forall t \in[0, T], \forall \omega \in \Omega,  \tag{21}\\
\dot{\psi}(t \mid \omega)-r \psi(t \mid \omega)=\hat{k}^{2}(t) \beta^{\omega} a(t \mid \omega)\left(\psi(t \mid \omega)-v_{I}\right)+(1-\hat{k}(t)) c_{S}, \\
\dot{p}(t \mid \omega)=-p(t \mid \omega)(1-p(t \mid \omega)) \beta^{\omega} \hat{k}^{2}(t) a(t \mid \omega), \\
\dot{s}(t \mid \omega)=-\beta^{\omega} \hat{k}^{2}(t) s(t \mid \omega) a(t \mid \omega), \\
\dot{a}(t \mid \omega)=-\alpha^{\omega} \dot{s}(t \mid \omega)-\gamma_{a}^{\omega} a(t \mid \omega), \\
\mu(t, \omega)=\frac{\mu^{0}(\omega) /(1-p(t \mid \omega)}{\sum_{\omega^{\prime}} \mu^{0}\left(\omega^{\prime}\right) /\left(1-p\left(t \mid \omega^{\prime}\right)\right)},
\end{array}\right.
$$

and $\psi(T \mid \omega)=0$.


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[^1]:    ${ }^{1}$ Delamater et al. (2019) and Britton et al. (2020) show that the herd immunity level against COVID-19 is reduced when the model encompasses the possibility that some social groups of individuals are more socially active. 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. Cowling et al. (2020) show that border restrictions and changes in individual behaviors are partly responsible for reduced transmission in Hong Kong in February 2020.
    ${ }^{2}$ 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. Ahituv et al. (1996) show that the demand for condoms increases in regions where HIV is prevalent.

[^2]:    ${ }^{3}$ The usual assumption in the literature is that infected individuals chose a constant social activity level $\bar{k}$ during the symptomatic period. Assuming $\bar{k}=0$ is without loss of generality for the purpose of this paper and lightens the analytic expressions.
    ${ }^{4}$ By the law of large numbers, the initial proportion of infected individuals with symptoms represents a share $1-\alpha$ of the population of infected people, thus $\bar{i}=(1-\alpha)(\bar{i}+\bar{a})$.

[^3]:    ${ }^{5}$ As $s(t+d t \mid \omega)-s(t \mid \omega)=-\int_{j \in S(t \mid \omega)} k_{j}(t) d j \times \int_{j \in A(t \mid \omega)} k_{j}(t) d j \times \beta d t$. The result follows from the fact that $\dot{s}(t \mid \omega)=\lim _{d t \rightarrow 0} \frac{s(t+d t \mid \omega)-s(t \mid \omega)}{d t}$.

[^4]:    ${ }^{6}$ In Section we describe a more general model where uncertainty pertains also to the epidemiological parameters $\beta, \alpha, \gamma^{s}, \gamma^{a}, \nu$. The analysis is similar with heavier notation.
    ${ }^{7}$ Fix $\omega \in \Omega$ and a strategy profile $\mathbf{k}$. A susceptible individual $j$ develops symptoms in $[t, t+d t)$ with probability 0 when she is of type $\theta_{a}$; when she is of type $\theta_{s}$, she develops symptoms if she meets and is infected by some asymptomatic individual, which occurs with instantaneous probability $k_{j}(t) \bar{k}_{A}(t \mid \omega) a(t \mid \omega) \times \beta d t$. By Bayes' rule, the law of motion of the subjective belief of individual $j$ is thus (6).
    ${ }^{8}$ See Lemma 1 in the Appendix.

[^5]:    ${ }^{9}$ See Lemma 2 in the Appendix for the detailed calculations.
    ${ }^{10}$ See Appendix 5.1 for the detailed calculation.

[^6]:    ${ }^{11}$ See for instance van Dorp et al. (2020).

[^7]:    ${ }^{12}$ Precisely, at stage 1 a value $\psi(0 \mid \omega)[1]$ is uniformly drawn from an interval of reasonable values and is temporarily designed "best candidate". The final value of $\psi$ given $\psi(0 \mid \omega)[1]$, i.e., $\psi(T \mid \omega)[1]$, is computed. At stage 2 , another value $\psi(0 \mid \omega)$ [2] is drawn at random. If the corresponding final value $\psi(T \mid \omega)[2]$ is closer to 0 than $\psi(T \mid \omega)(1)$, then $\psi(0 \mid \omega)[2]$ becomes the new best candidate. The process does on iteratively and stops after a deterministic number of rounds $N$, which is large enough to guarantee that $\psi(T \mid \omega)[N]$ is almost 0 with the final best candidate.
    ${ }^{13}$ 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 $r=-\ln (0.99986)$.
    ${ }^{14}$ See e.g., Remuzzi and Remuzzi (2020).

[^8]:    ${ }^{15}$ In our model, an infected of the symptomatic type dies if the event "Death" occurs for her before the event "Healing". Therefore, the probability of death (conditional on being infected and the symptomatic type) is $P\left(\tau_{D}<\tau_{H}\right)$, with $\tau_{H}$ and $\tau_{D}$ denoting the random times of healing and death, respectively. Straightforwardly, $P\left(\tau_{D}<\tau_{H}\right)=\int_{0}^{\infty} F_{\tau_{D}}(t) f_{\tau_{H}}(t) d t=\nu /\left(\gamma^{s}+\nu\right)$ since $f_{\tau_{D}}(t)=\nu e^{-\nu t}$ and $f_{\tau_{H}}(t)=\gamma^{s} e^{-\gamma^{s} t}$.

[^9]:    ${ }^{16}$ See the proof of Lemma 1.

