Equilibrium social distancing efforts, public policies and the COVID-19 epidemics

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Abstract

The COVID-19 pandemic is a global threat that has caused the lockdown of about one half of the world's population for weeks. Containing the epidemics comes however at a very large economic and welfare cost, so that lockdown is being removed in many countries. We model the dynamics of the epidemics after (full or partial) lockdown removal, incorporating the equilibrium behavior of individuals. Individuals optimally choose self-protection efforts (a reduction in contacts) given their risk aversion, beliefs and perceptions, utility from contacts, effectiveness of efforts, and the current prevalence of the disease. We show that contrary to imposed social distancing, individually optimal self-protection does not lead to a constant reduction in contacts intensity. In equilibrium, self-protection efforts adjust at each date in a non-proportional way given the number of reported cases. We calibrate the model on French data. The general dynamics of epidemics differs markedly from the one obtained when neglecting to account for equilibrium self-protection efforts. It involves a rebound in the epidemics, with a peak number of cases that is about 1/10th of the one with business-as-usual, and a cumulated number of deaths that is about 1/6th lower.

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1 Introduction

The COVID-19 pandemic started in Wuhan, in December 2019. The World Health Organization (WHO) recognized it as a pandemic on 11 March 2020. On the 13^{th} of May 2020, more than 4.1 million cases of COVID-19 have been reported in over 212 countries and territories over the world resulting in more than 292,000 deaths worldwide – more than 82,000 deaths in the US, than 30,000 in the UK and Italy, 26,000 in France and Spain ([25]). As no treatment or vaccine exists yet, the only way to contain the spread of the epidemics is lockdown, to ensure maximal social distancing. Maier and Brockmann (2020) show that lockdown (and the ensuing social distancing) has been effective at curbing the spread of the epidemics in China, despite an initial exponential growth (exponential growth has been observed e.g., in Italy, France, Great Britain and the US). Social distancing measures have also been more effective in China than mobility restrictions [14]. In this context, compliance with social distancing during lockdown but especially at the end of the lockdown period is an essential determinant of the dynamics of the epidemics. While Zhang et al. (2020) show that contacts have been reduced 7- to 8-fold in Wuhan and Shanghai, which has contained the epidemics, such a drastic reduction may not be imposed for a long period of time in most countries, and involves extremely large economic and welfare costs. It is therefore particularly important to model individual behavior after lowkdown to be able to simulate dynamics and possible rebounds.

The aim of this paper is to integrate in a SIRU epidemiologic model, an economic model of self-protection choices. After lockdown ends, individuals will choose how much to maintain some social distancing effort, and how much to undertake other efforts that help reduce virus spread, such as wearing a mask, washing hands, etc. Because these efforts are individual choices in most countries, in the process of going back to normal, they should be explicitly modeled. Doing so enables us to assess the impact of important variables on individual choices, and thereby on the spread of the virus: These variables include the degree of risk aversion, the psychological and monetary attractiveness of contacts and the perceived efficiency of various self-protection measures, in addition to the epidemics prevalence and severity of the disease.

This article studies how individual choices interact with the dynamics of the epidemics. Because individuals do not internalize the positive impact of their prevention efforts on others, their choices are typically inefficient and exhibit too little prevention. This is a well-known feature of vaccination decisions for instance, and justifies public intervention. In the context of the Covid-19, the prevention effort consists in reducing the number and intensity of contacts with others in the population. As for vaccines, the positive externality generated by more effort (more reduction in the number of contacts) can justify imposing lockdown on individuals. However, once lockdown will be removed, individuals will be free to choose the intensity of their contacts with other individuals, and a rebound of the epidemics may occur. We focus on how individuals' perception of the infection risk (given the specificity of the Covid-19), their perception of the severity of the disease, and their degree of risk aversion, together determine their behavior, and how this behavior, in turn, determines the dynamics of the epidemics. Importantly, because there are many asymptotic infected individuals, and because of the salience of the epidemics in the media and public discourse, risk perception is likely to be much higher than the risk computed based on reported cases. We show that perceptions and attention to the infection risk are very important drivers of the epidemics. This highlights the potential effectiveness of policy measures to credicly communicate concern about infection risks.

There is ample evidence that individuals change their behavior in reaction to an epidemics. In a documented study about the SARS 2003 epidemics in Hong-Kong and Singapore, Ferguson [7] describes how the epididemic modified individual behavior, especially with respect to social contacts. Based on a survey on H1N1 and SARS, Balinska and Rizzo [2] show that epidemics induce behavioral changes that then affect the spread of the epidemics. This change of social behavior has also been stressed for the HIV epidemics in San Francisco in the gay community (Mc Kusik et al. [18]) and in Africa (Green et al.

[11]).

Much theoretical work has shown that taking into account the bahavior of individuals modifies the spread of the epidemics. D'Onofrio and Manfredi [21] present a survey of these models, since the seminal work of Capassio and Serio ([4]). Surveys on behavior and epidemics have also been realized by Funk et al ([8]) from papers published before 2010 and Verhelst et al. [23] (from 2010 to 2015). Most of the behavioral models concerns vaccine uptakes.

Behavioral models differ one from another on the way individuals perceive prevalence (which is based on the information which is avalaible to make one's own judgement, Funk et al. [8]), and on the way in which individals evaluate risk. D'Onofrio and Manfredi [21] propose a general form written as a delayed function of a general form of prevalence. The aim of the individual is then to choose the optimal strategy that maximizes her expected utility. In most models, individuals are risk neutral. A novelty of our approach is that we incorporate risk aversion and self-protection efforts to our epidemiological model.

The aim of our work is to understand how the intensity of contact and other self-protection efforts are endogeneously chosen by the individual and how this in turn changes the epidemics dynamics. Our model differs from previous models because the only information which is available to an individual is the number of (either newly or cumulative) reported cases or deaths, and individuals maximize their utility over some self-protection effort [13]. As far as the COVID-19 epidemics is concerned, the transmission of the disease is mainly due to either exposed or unreported cases. We study how the intensity of contacts depends on prevalence and risk perception, perception of health loss due to the disease and risk aversion. We then use the characterization of the optimal self-protection efforts to understand how the epidemics dynamics is modified.

Self-protection consists in an action that reduces the probability of becoming infected [6]. We study a first type of self-protection effort: It entails a utility loss and is particularly adequate to model contacts reduction, that have both psychological and financial consequences that are risky in the sense that they depend on one's health status. We then add the possibility for the individual to also make a second type of effort. This second type of effort entails a deterministic cost (such as an upfront payment or a discomfort) and is more adequate to model wearing a mask and other protective measures, including washing hands, that require initial changes in habits, and time and attention. We study the substitutability / complementarity of the two types of effort. Being able to use two types of instruments to reduce one's risk of infection does not necessarily ultimately reduce the infection risk. We also study the effectiveness of public measures to impose partial confinement (for a proportion of the population) and the impact of the salience of the disease. We show that even with a large overestimation of the probability of getting infected compared with available data (due to strong salience for instance), individual efforts are not sufficient to avoid sizable rebounds. The evolution of the epidemics is however very dependent on individual efforts and on behavioral parameters.

1.1 Our approach

Our modeling of the dynamics of the epidemics is original in that we incorporate features specific to the virus as well as features specific to human behavior. Among the former, a prominent element is the uncertainty about the proportion of non immunized, contagious individuals. Among the latter, we focus especially on risk aversion, that affects the choice of the number of contacts for each individual, and on the length of the lockdown period, that affects the desirability of contacts.

The SIR model much used in the litterature is ill-fitted to study the Covid-19, because of the importance of asymptomatic infected individuals. While this category of individuals can easily be added to the dynamic structure of the model, this is not sufficient to fully account for its role in the dynamics of the epidemics. One of the consequences of infection via asymptomatic individuals is that it generates additional uncertainty.

The fact that many individuals may be infected (and contagious) without exhibiting symptoms indeed creates an intrinsic uncertainty about the probability of being infected. Not only are individuals unable to observe the real state of others in the population, and themselves; Scientists also face much uncertainty about the proportion of asymptotic infected individuals, so that one cannot easily use statistics to correct for the lack of observability. We highlight how this characteristic of the disease leads to an underestimation of the real probability of becoming infected, and hence the choice of an excessive number of contacts.

We model how the individual perception about the infection risk is based on the number of new reported cases, which is the prominent information available in the media. But this number falls short of the real number of contagious individuals, given the proportion of asymptomatic cases. Because an individual will optimally choose her prevention effort (or equivalently, optimally choose the intensity of the contacts she has with other individuals), this bias in perception will have a strong impact on the dynamics of the epidemics. Risk aversion may to some extent mitigate this bias.

We model important features of the choice of contacts intensity, that may differ across individuals but may also vary according to the length of the lockdown period: Risk aversion, the desirability of contacts, the perceived severity of the disease, the attention paid to reported cases. Our model thus links the specifics of the epidemics to individual decisions under the intrinsic uncertainty generated by the epidemics, and subsequently to the spread of the epidemics itself.

2 The model

2.1 The epidemiological system

Our model of the epidemics is based on Liu et al. ([15],[16]) and is given by System 1:

$$\begin{aligned} S' &= -\tau(t)S\,(I+U) \\ I' &= \tau(t)S\,(I+U) - \nu I \\ R' &= f\nu I - \eta R - \delta R \\ U' &= (1-f)\nu I - \eta U \end{aligned} \tag{1}$$

with initial data

$$S(t_0) = S_0, I(t_0) = I_0, R(t_0) = 0, U(t_0) = U_0.$$

$I(t)$ is the number of infectious asymptomatic individuals, $R(t)$ the number of severe symptomatic infectious individuals, who are tested COVID-19 and classified as Reported and $U(t)$ the number of mild symptomatic infectious individuals, who are undetected. $S(t)$ is the number of individuals susceptible to be contaminated by the infectious $I(t)$ and $U(t)$. We assume that infectious severe symptomatic are not active in the transmission of the disease because they are isolated.

Contaminated susceptible individuals then become infectious asymptomatic for an average time ν^{-1}. Then, a fraction f of infectious asymptomatic develop severe symptoms, which last for a mean time η^{-1}. However, severe asymptomatic individuals may die at rate γ. A fraction $1-f$ of infectious asymptomatic individuals become mild symptomatic and lose infectiousness after an average time η^{-1}.

The transmission rate $\tau\,(t)$ depends on the infectiousness of the disease but also on a behavioural component, the number of contacts by time unit. We assume that before individuals take any social distancing measures $\tau\,(t) = \tau_0$. The impact of reducing social contact $\varphi(t) \in [0,1]$ lowers the transmission rate so that $\tau\,(t) = \tau_0\varphi(t)$.

We assume that lockdown is applied at time $t_1 > t_0$ and is lifted at time $t_2 > t_1$. For $t \in [t_0, t_2]$, φ is exogenously given by Equation (2). This formulation has been considered in Chowell et al. [5] for Ebola disease and Augeraud [1] for COVID-19.

$$\varphi(t) = \begin{cases} 1 \text{ for } t < t_1 \\ \varphi^0 + (1 - \varphi^0)e^{-\mu(t-t_1)}, \text{ for } t_1 < t < t_2. \end{cases} \tag{2}$$

Table 1 presents the parameters we consider

Symbol	Interpretation	Value
t_0	Time when the epidemic started	February 3rd [17]
t_1	Time when the lockdown started	March 17th [[?]]
t_2	Time when the lockdown ends	May 11th
S_0	Number of susceptible at time t_0	$66.99.10^6$
I_0	Number of asymptomatic infectious at time t_0	3.675 [17]
U_0	Number of unreported symptomatic infectious cases at time t_0	0.892 [17]
R_0	Number of reported symptomatic infectious cases at time t_0	1
τ	Transmission rate	$4.23.10^{-9}$ [17]
γ	Death due to the disease	fitted
ν^{-1}	Average duration of the asymptomatic infectious period	7
f	Fraction of asymptomatic infectious who become reported symptomatic infectious	0.4
$\nu_1 = f\nu$	Rate at which asymptomatic infectious become reported symptomatic infectious	
$\nu_2 = (1-f)\nu$	Rate at which asymptomatic infectious become unreported symptomatic infectious	
η^{-1}	Average time symptomatic infectious have symptoms	7
$\varepsilon_1(t)$	intensity of contacts among unreported infected individuals at time t	fitted
ε_2	intensity of contacts among reported infected individuals	0.0001

Table 1: Table of parameters

We are interested in understanding the behaviour of the individuals after the lift of lockdown, that is for time $t > t_2$. More precisely, we assume that from time $t \geq t_2$, individuals will rationally chose the effort ε they would do to reduce their social contacts.

Thus from time $t \geq t_2$, the impact of reducing social contact $\varphi(t)$ is defined as $\varphi(t) = \phi(\varepsilon(t))$ where $\phi(\varepsilon)$ is the impact of doing an effort ε. The following paragraph explains how ε is determined by individuals.

2.2 The individual's preferences and perceptions

We assume that individuals are myopic (they are not able to compute and anticipate the dynamics of the epidemics) and that each chooses to do an effort ε to reduce her contacts, and thereby reduce her perceived probability of infection. Reducing contacts creates a disutility, as it involves psychological costs and economic foregone opportunities (reducing contacts may entail working only very few hours). The chosen self-protection effort maximizes at time $t \geq t_2$ the individual's expected utility defined as:

$$J(\varepsilon, y) = (1 - p(\varepsilon, t; y))\, u(U_S, \varepsilon; y) + p(\varepsilon, t; y)\, u(U_M, \varepsilon; y)$$

were $p(\varepsilon, t; y)$ is the perception of the risk of being infected at time t and decreases in the effort ε that the individual makes to reduce contacts. Function $u(H, \varepsilon; y)$ is the utility function, which depends on two variables: the well-being $H \in \{S, M\}$ associated to having $(H = M)$ or not having $(H = S)$ the disease, and the effort of reducing contacts, ε. It may also depends on the type of the individual, $y \in T$, which density function is π. We consider an homogeneous population in a first step of our study.

To obtain tractable solutions and to be able to assess the impact of risk aversion on individual decisions, we make the following assumptions.

The utility function.

$$\begin{aligned} u(U_S, \varepsilon; y) &= \frac{U_S^{1-\frac{1}{\sigma}} (1 - \theta\varepsilon)^{\left(1-\frac{1}{\sigma}\right)}}{1 - \frac{1}{\sigma}} \\ u(U_M, \varepsilon; y) &= \frac{(\lambda(y)\, U_s)^{1-\frac{1}{\sigma}} (1 - \theta\varepsilon)^{\left(1-\frac{1}{\sigma}\right)}}{1 - \frac{1}{\sigma}}, \text{ with } 0 < \lambda(y) < 1 \end{aligned}$$

with $\sigma > 1$ and $\theta > 0$. We would assume in the following that $U_S = 1$.

Parameter σ measures the degree of risk aversion of the individual, θ is a measure of disutility of the effort, and λ measures the perception of the individual about the severity of the disease. All three parameters are fundamental determinants of the behavior of the individual.

The risk perception. We assume that the perceived probability of being infected $p(\varepsilon, t; y)$ is computed as a function of the perceived prevalence of the disease (d'Onofrio and Mandredi [21]) times the impact of the effort of reducting contact. The disease is transmited by asymtomatic infectious and mild symptomatic infectious whose number is unknown. The risk perception is thus computed by a proxy of these disease transmitter. As the number of daily reported cases $(f\nu I)$ is indeed one of the main information provided by the media, we assume that the perceived prevalence of the disease is

$$\frac{k(y)\,\tau_0 \varphi(t)\,\nu_1 I}{\nu + k(y)\,\tau_0 \varphi(t)\,\nu_1 I}$$

Parameter $k(y)$, which may depends on the type of the individual, can measure the attention paid to this information by the individual, or the confidence the individual gives to official information. Thus the perceived prevalence is computed by individuals as a proportion $k(y) > 1$ of the number of daily reported cases at time t that amounts to $\nu_1 I$. The impact of reducing social contacts $\varphi(t)$ depends on the average effort $\overline{\varepsilon}$ of reducing contacts

$$\overline{\varepsilon} = \int_T \varepsilon(y)\,\pi(y)\,dy$$

To explicit the dependence on the average effort , we denote the perceived prevalence of the disease

$$\pi(I, \overline{\varepsilon}; y) = \frac{k(y)\,\tau_0 \phi(\overline{\varepsilon})\,\nu_1 I}{\nu + k(y)\,\tau_0 \phi(\overline{\varepsilon})\,\nu_1 I}$$

The perceived probability of being infected can thus be written

$$p(\varepsilon, \overline{\varepsilon}, I; y) = \phi(\varepsilon)\,\pi(I, \overline{\varepsilon}; y)$$

We consider Assumption 1

Assumption 1 *We assume* $\phi'(\varepsilon) < 0$, *and* $\phi(0) = 1$, $\phi^{(2)}(\varepsilon) \geq 0$, *and that* $\lim_{\varepsilon \to 0} \phi'(\varepsilon) < \infty$.

We consider the following as an example $\phi(\varepsilon) = 1 - \varepsilon$.

3 The behavioral model in a homogeneous population

As we assume that all individuals have the same type, we will drop index y in this paragraph.

3.1 Optimal effort to reduce contacts

Due to large population, one single individuals has on influence on the mean contact effort $\overline{\varepsilon}$, which is considerated as given for the optimization problem.

The optimal strategy $\varepsilon \in [0, 1]$ maximizes expected utility fonction $J(\varepsilon)$. If interior solution $\widehat{\varepsilon}$ exists, it satisfies $J'_{\varepsilon}(\widehat{\varepsilon}) = 0$, where

$$\begin{aligned} J'_{\varepsilon}(\varepsilon) &= -p'_{\varepsilon}(\varepsilon, \overline{\varepsilon}, I)\left[u(U_S, \varepsilon) - u(U_M, \varepsilon)\right] \\ &\quad + (1 - p(\varepsilon, \overline{\varepsilon}, I))\, u'_{\varepsilon}(U_S, \varepsilon) + p(\varepsilon, \overline{\varepsilon}, I)\, u'_{\varepsilon}(U_M, \varepsilon) \end{aligned}$$

As $J_{\varepsilon}^{(2)}(\varepsilon)$ satisfies

$$
\begin{aligned}
J_{\varepsilon}^{(2)}(\varepsilon) &= -p_{\varepsilon}^{(2)}(\varepsilon,\overline{\varepsilon},I)\left[u(U_S,\varepsilon)-u(U_M,\varepsilon)\right] \\
&\quad -2p_{\varepsilon}'(\varepsilon,\overline{\varepsilon},I)\left[u_{\varepsilon}'(U_S,\varepsilon)-u_{\varepsilon}'(U_M,\varepsilon)\right] \\
&\quad +(1-p(\varepsilon,\overline{\varepsilon},I))\,u_{\varepsilon}^{(2)}(U_S,\varepsilon)+p(\varepsilon,\overline{\varepsilon},I)\,u^{(2)}(U_M,\varepsilon)
\end{aligned}
$$

Thus $J(\varepsilon)$ is a concave function. The optimal effort of reducting contact is $\varepsilon^* = \max(\min(1,\overline{\varepsilon}),0)$.

More explicitely, denoting $\Lambda = \lambda^{1-\frac{1}{\sigma}}$, this yields

$$
\begin{aligned}
J_{\varepsilon}'(\varepsilon) &= -\phi'(\varepsilon)\,\pi(I,\overline{\varepsilon})\,\frac{(1-\theta\varepsilon)^{1-\frac{1}{\sigma}}}{1-\frac{1}{\sigma}}\left[1-\Lambda\right] \\
&\quad -\theta(1-\theta\varepsilon)^{-\frac{1}{\sigma}}\left[1-\phi(\varepsilon)\,\pi(I,\overline{\varepsilon})(1-\Lambda)\right]
\end{aligned}
$$

The interior solution $\widehat{\varepsilon}$ thus satisfies

$$
-\phi'(\widehat{\varepsilon})\,\frac{(1-\theta\widehat{\varepsilon})}{1-\frac{1}{\sigma}}+\theta\phi(\widehat{\varepsilon}) = \frac{\theta}{\pi(I,\overline{\varepsilon})(1-\Lambda)}
$$

As $\pi_{\overline{\varepsilon}}'(I,\overline{\varepsilon}) = \frac{\kappa\tau_0\phi'(\overline{\varepsilon})\nu f I}{\nu+\kappa\tau_0\phi(\overline{\varepsilon})\nu f I} < 0$, $\widehat{\varepsilon}$ is a strictly decreasing function of $\overline{\varepsilon}$. Indeed:

$$
\frac{\partial\widehat{\varepsilon}}{\partial\overline{\varepsilon}} = \left(\frac{-\pi_{\overline{\varepsilon}}'(I,\overline{\varepsilon})}{(1-\Lambda)}\frac{\theta}{\pi^2(I,\overline{\varepsilon})}\right)\frac{1}{-\phi^{(2)}(\overline{\varepsilon})\,\frac{(1-\theta\varepsilon)}{1-\frac{1}{\sigma}}+\theta\left(\frac{2-\frac{1}{\sigma}}{1-\frac{1}{\sigma}}\right)\phi'(\overline{\varepsilon})} < 0
$$

The variation of $\widehat{\varepsilon}$ according to the other parameters of the model can be studied accordingly.

$$\frac{\partial\widehat{\varepsilon}}{\partial\Lambda} = \left(\frac{-1}{(1-\Lambda)^2}\frac{\theta}{\pi(I,\overline{\varepsilon})}\right)\frac{1}{-\phi^{(2)}(\overline{\varepsilon})\frac{(1-\theta\varepsilon)}{1-\frac{1}{\sigma}}+\theta\left(\frac{2-\frac{1}{\sigma}}{1-\frac{1}{\sigma}}\right)\phi'(\overline{\varepsilon})} > 0$$

$$\frac{\partial\widehat{\varepsilon}}{\partial I} = \left(\frac{-\pi_I'(I,\overline{\varepsilon})}{(1-\Lambda)}\frac{\theta}{\pi^2(I,\overline{\varepsilon})}\right)\frac{1}{-\phi^{(2)}(\overline{\varepsilon})\frac{(1-\theta\overline{\varepsilon})}{1-\frac{1}{\sigma}}+\theta\left(\frac{2-\frac{1}{\sigma}}{1-\frac{1}{\sigma}}\right)\phi'(\overline{\varepsilon})} > 0$$

$$\frac{\partial\widehat{\varepsilon}}{\partial\sigma} = \frac{-1}{-\phi^{(2)}(\overline{\varepsilon})\frac{(1-\theta\overline{\varepsilon})}{1-\frac{1}{\sigma}}+\theta\left(\frac{2-\frac{1}{\sigma}}{1-\frac{1}{\sigma}}\right)\phi'(\overline{\varepsilon})}\left(\phi'(\overline{\varepsilon})\frac{(1-\theta\overline{\varepsilon})}{(\sigma-1)^2}\right) < 0$$

However, the sign of $\frac{\partial\widehat{\varepsilon}}{\partial\theta}$ is ambiguous.

$$\frac{\partial\widehat{\varepsilon}}{\partial\theta} = -\left(\frac{\widehat{\varepsilon}\phi'(\widehat{\varepsilon})}{1-\frac{1}{\sigma}}+\phi(\widehat{\varepsilon})-\frac{1}{\pi(I,\overline{\varepsilon})(1-\Lambda)}\right)\frac{1}{\phi^{(2)}(\widehat{\varepsilon})\frac{(1-\theta\widehat{\varepsilon})}{1-\frac{1}{\sigma}}+\theta\phi'(\widehat{\varepsilon})}$$

The Best Response function.

Optimal contact effort, given the average contact effort accross the population can be seen as a function of the infecAs $\frac{\partial\widehat{\varepsilon}}{\partial I} > 0$, and according to Assumption 1 there exists $0 < I_1(\overline{\varepsilon})$ and $I_2(\overline{\varepsilon})$, with $I_1(\overline{\varepsilon}) \leq I_2(\overline{\varepsilon}) \leq \infty$, such that

$$\varepsilon^* = \begin{cases} 0, \text{ if } I < I_1(\overline{\varepsilon}) \\ \widehat{\varepsilon}, \text{if } I_1(\overline{\varepsilon}) \leq I \leq I_2(\overline{\varepsilon}) \\ 1, \text{if } I \geq I_2(\overline{\varepsilon}) \end{cases}$$

The best response function gives the optimal solution ε^* as a function of the other player behavior, here $\overline{\varepsilon}$. It is denoted $\varepsilon^* = BR(\overline{\varepsilon})$.

For $\phi(\varepsilon) = 1-\varepsilon$, the interior solution $\widehat{\varepsilon}$ is given by

$$BR(\overline{\varepsilon}) = \min\left(\max\left(\frac{\sigma}{\theta(2\sigma-1)} - \frac{(\nu+\Lambda k\tau_0(1-\overline{\varepsilon})\nu_1 I)(\sigma-1)}{k\tau_0(1-\overline{\varepsilon})\nu_1 I(1-\Lambda)(2\sigma-1)}, 0\right), 1\right)$$

The special case of risk neutrality. In order to better see the effects at play, one can consider the case of a risk neutral individual. Then her utility function is

$$J'_{\varepsilon}(\varepsilon) = \left[-p'_{\varepsilon}(\varepsilon, t)(1-\theta\varepsilon) + \theta p(\varepsilon, t)\right](1-\lambda) - \theta$$

In the absence of risk aversion, the determinants of the self-protection effort are easier to interpret: The optimal effort to reduce contacts is obtained by equating the value of avoiding to become ill, i.e., the additional utility obtained when remaining sane, $U_S - U_M = (1-\lambda)(1-\theta\varepsilon)$ with the marginal desutility of reducing one's contacts for a unit reduction in the probability of becoming ill, i.e., $\frac{-\theta(1-p(\varepsilon)(1-\lambda))}{p'(\varepsilon)}$:

$$(1-\lambda)(1-\theta\varepsilon^*) = \frac{\theta(1-p(\varepsilon^*)(1-\lambda))}{-p'(\varepsilon^*)}$$

Nash Equilibrium

As the population is homogeneous, the Nash Equilibrium is defined by

$$\overline{\varepsilon} = BR(\overline{\varepsilon})$$

For $\phi(\varepsilon) = 1 - \varepsilon$, the Nash Equilibrium solves

$$\overline{\varepsilon} = \min\left(\max\left(\frac{\sigma}{\theta(2\sigma-1)} - \frac{(\nu + \Lambda k\tau_0(1-\overline{\varepsilon})\nu_1 I)(\sigma-1)}{k\tau_0(1-\overline{\varepsilon})\nu_1 I(1-\Lambda)(2\sigma-1)}, 0\right), 1\right)$$

If the Nash Equilibrium is an interior solution, letting $\widetilde{\phi} = 1 - \varepsilon$, we have

$$\widetilde{\phi} = 1 - \frac{\sigma}{\theta(2\sigma-1)} + \frac{\left(\nu + \Lambda k\tau_0\widetilde{\phi}\nu_1 I\right)(\sigma-1)}{k\tau_0\widetilde{\phi}\nu_1 I(1-\Lambda)(2\sigma-1)}$$

z is a solution of the order 2 polynomial $P(z) = 0$, where

$$P(z) = \widetilde{\phi}^2 - \left(1 - \frac{\sigma}{\theta(2\sigma-1)} - \frac{\Lambda(\sigma-1)}{(1-\Lambda)(2\sigma-1)}\right)\widetilde{\phi} - \frac{\nu(\sigma-1)}{k\tau_0\nu_1 I(1-\Lambda)(2\sigma-1)}$$

The polynomial admits exactly one positive root

$$\begin{aligned}\widehat{\phi} &= \frac{1}{2}\left(1-\frac{\sigma}{\theta\left(2\sigma-1\right)}-\frac{\Lambda\left(\sigma-1\right)}{\left(1-\Lambda\right)\left(2\sigma-1\right)}\right) \\ &+\frac{1}{2}\sqrt{\left(1-\frac{\sigma}{\theta\left(2\sigma-1\right)}-\frac{\Lambda\left(\sigma-1\right)}{\left(1-\Lambda\right)\left(2\sigma-1\right)}\right)^{2}+4\frac{\nu\left(\sigma-1\right)}{k\tau_0\nu_1 I\left(1-\Lambda\right)\left(2\sigma-1\right)}}\end{aligned}$$

To emphasize the dependency of the Nash equilibrium on I, we denote the Nash Equilibrium

$$\varepsilon^*\left(I\right)=\min\left(\max(1-\widehat{\phi},0),1\right)$$

The related impact function is $\phi^*\left(I\right)=\min\left(\max(\widehat{\phi},0),1\right)$.

Consequences on the spread of the epidemics now need to be studied.

3.2 The induced epidemics dynamics

In a homogeneous population, the reduction effort is ε^* previously defined. We consider it a function of I and denote it $\varepsilon^*\left(I\right)$.

The population dynamics satisfies

$$\begin{aligned}S' &= -\beta\phi^*\left(I\right)S\left(I+U\right) \\ I' &= \beta\phi^*\left(I\right)S\left(I+U\right)-\nu I \\ R' &= \nu_1 I-\eta R-\delta R \\ U' &= \nu_2 I-\eta U\end{aligned}$$

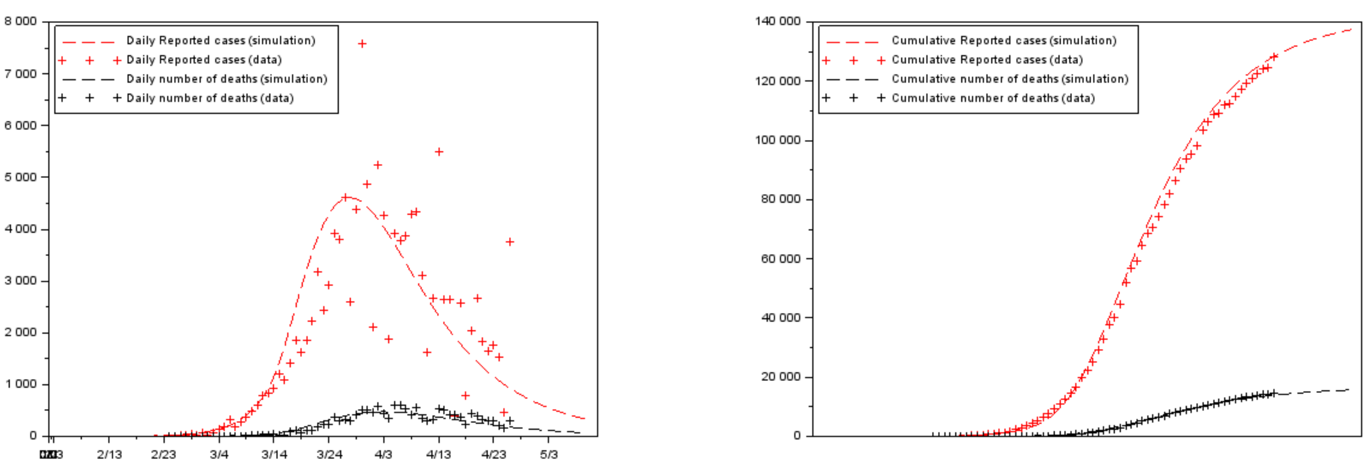
with initial data

$$S(t_0)=S_0, I(t_0)=I_0, R(t_0)=0, U(t_0)=U_0$$

Note that computing the reproduction number $\mathcal{R}_0$ makes little sense in our problem: it indicates how many individuals would be contaminated by one individual in a succeptible population during the length of her infectiousness. We are instead concerned with the epidemics dynamics after a lockdown period, with a large part of the population already infected.

Calibration on French data. We calibrate the model given by Liu et al. [**?**]. The simulation and the actual data are given on Figure **??**. It corresponds to $\tau_0 = 4.238.10^{-09}$, $I_0 = 3.675$, $R_0 = 1$, $U_0 = 0.8927$, with t_0 being February the 3rd. We have assumed that during lockdown, the intensity of the contact is time dependent and given by $\beta e^{-\mu.t}$ with $\mu = 0.65$. The other parameters are: $f = 0.4$, $\eta = 1/7$ and $\nu = 1/7$, with $\nu_1 = f\nu$ and $\nu_2 = (1-f)\nu$.

Figure 3.2 presents our simulations compared to data of the Public Heath Agency from February 25th to April 27th on daily number of reported cases and deaths (Figure 3.2 , (a)) and on cumulative number of reported cases and deaths (Figure 3.2 , (b)). Parameters ε_1^1 and μ have been chosen such that $\varepsilon_1^1 = 0.1088265$, $\mu = 0.11$

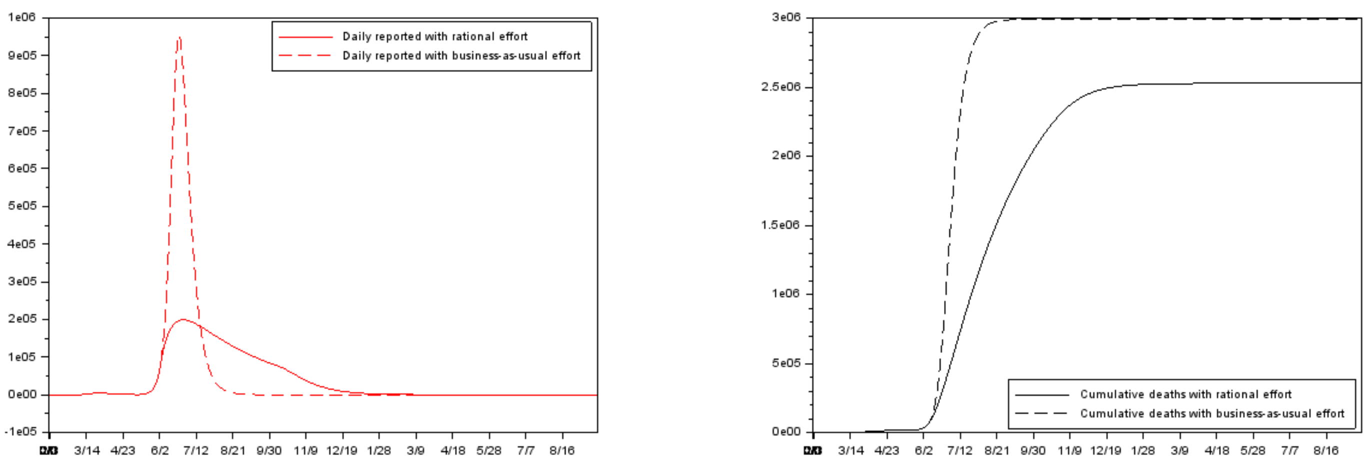


(a) (b)

Figure 1: Daily number of reported cases and deaths (a). Cumulative number of reported cases and deaths (b).

One can note that the fit is very good for death cases, despite their lower number, and less so for infected cases. This is because information on reported infected cases is of lower precision. Cumulative cases are fitted with a better precision.

The impact of self-protection. Figure 2 compares the number of reported cases and deaths with no reduction in contact intensity (dashed curve) and with rationally chosen contact intensity (plain curve). The optimal effort depends on perception parameters (here $\lambda = 0.00001$ and $k = 15$) and on the risk aversion parameter (here $\sigma = 1.5$, in line with the literature, Havranek 2015). Desutility of effort θ is chosen as $\theta = 0.1$. A linear intensity of the contact ϕ has been chosen, with $q = 0.15$. It can be seen that rational behavior does not prevent a 'rebound', that is the re-emergence of an epidemic outbreak after lockdown. However it lowers its magnitude; by doing so, it slows down herd immunity, so that the epidemic outbreak lasts longer.



(a) (b)

Figure 2: Comparison of epidemiological dynamics with rationally chosen effort (plain curves) and no effort (dashed curves. Daily reported cases (a) and cumulative deaths (b).

The corresponding reduction in contact intensity is given in Figure 3. Just after the lockdown, as perceived prevalence is low, contact intensity is equal to 1, and it lowers as soon as the perceived prevalence increases.

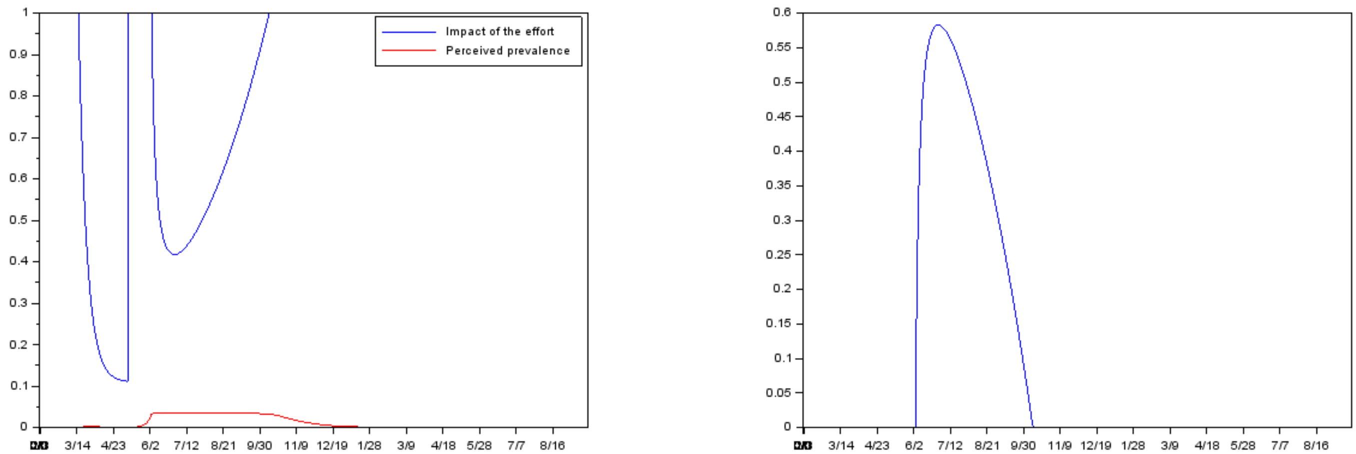
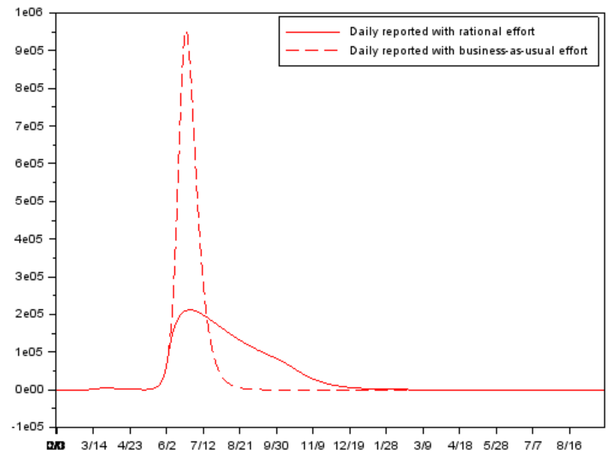


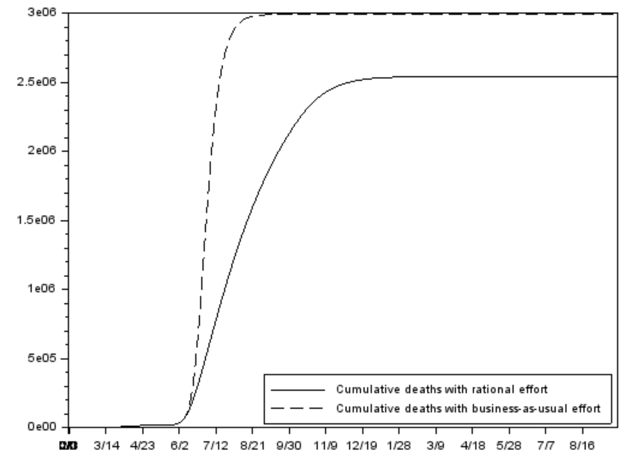
Figure 3: Contact intensity $\phi(\epsilon)$ (blue curve) and perceived prevalence (red curve) (a). Rationally chosen effort ϵ (b).

Interestingly, individuals do not maintain a very high effort level at all times. To the contrary they adapt their self-protection effort in such a way that the number of cases reaches a plateau.

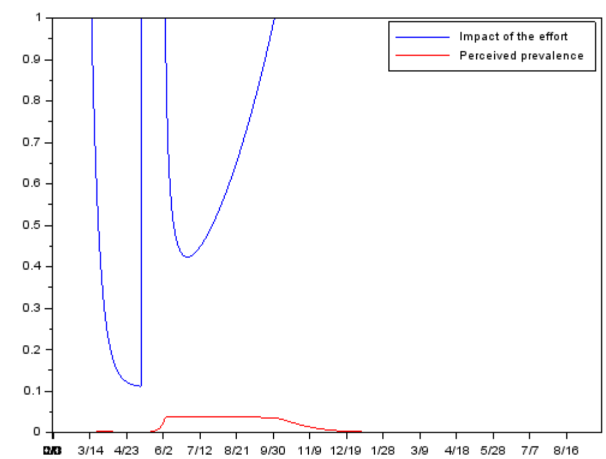
More optimistic perception of the consequences of the disease. Assume now that the utility obtained when one is sick is a higher proportion of the utility when sane. We model this as a higher λ: For $\lambda = 10^{-3}$, simulations are given on Figure 4.



(a)



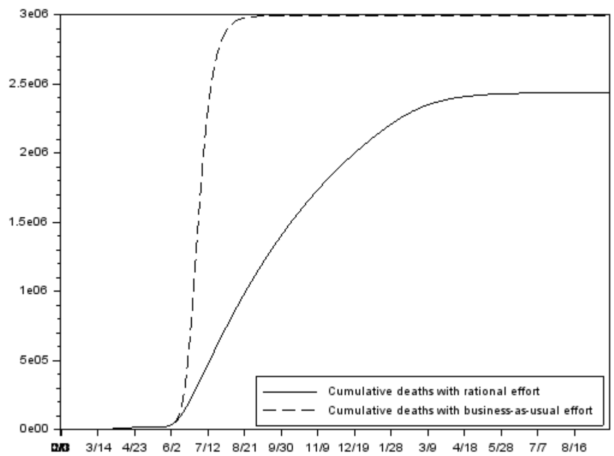
(b)



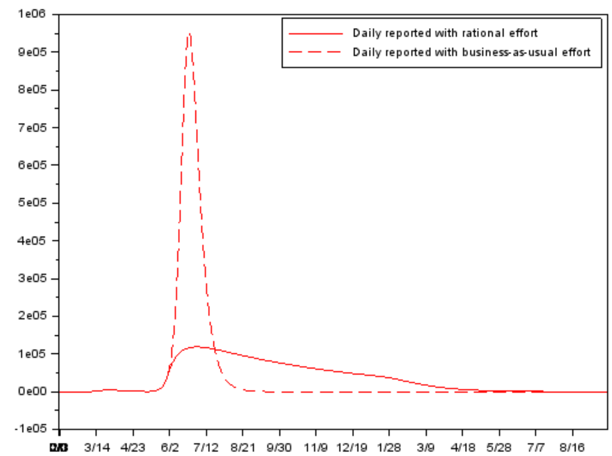
(c)

Figure 4: Impact of optimally chosen effort on daily reported cases (a), on the cumulative number of deaths (b) and on contact intensity $\phi(\epsilon)$ (c) for $\lambda = 10^{-3}$ and $k = 15$.

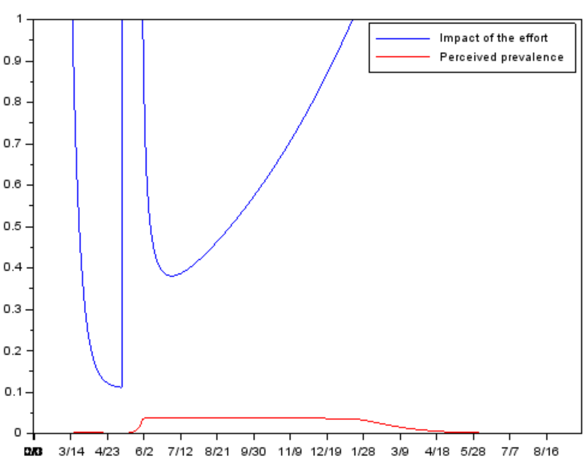
Increasing the perceived infection risk. A public policy may lead to increasing the perceived infection risk (for instance through increased media coverage of serious cases). This can be modeled in our framework as in increase in the weight k that the individual attaches to available data (reported cases). We consider $k = 30$ (instead of $k = 15$), which gives the results in Figure 5.



(a)



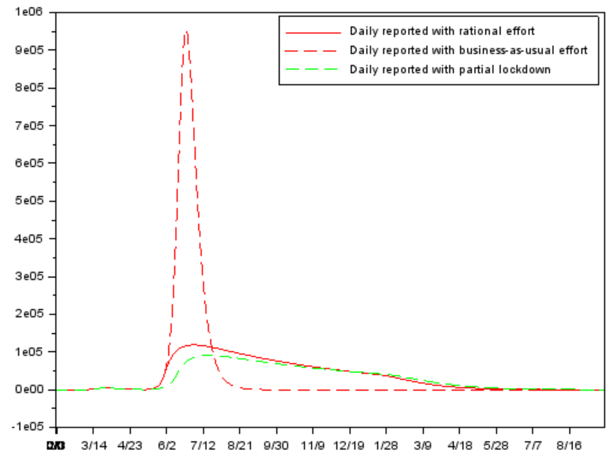
(b)



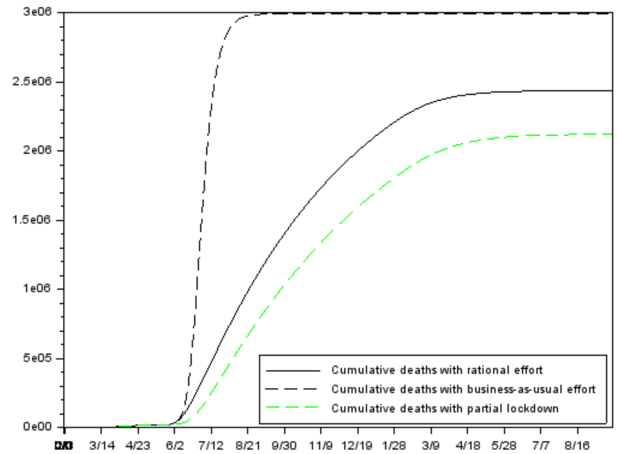
(c)

Figure 5: Impact of optimally chosen effort on daily reported cases (a), on the cumulative number of deaths (b) and on contact intensity $\phi(\epsilon)$ (c) for $\lambda = 10^{-3}$ and $k = 30$.

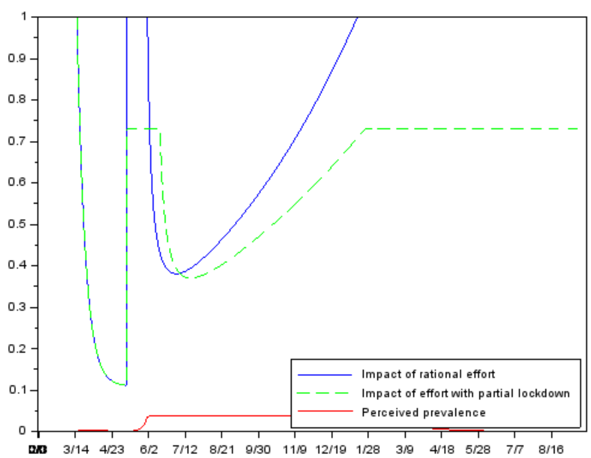
Imposing prolonged lockdown on a proportion of the population. We now consider the impact of a public policy of partial lockdown on the epidemiological dynamics and on the behavior of individuals. We assume that lockdown is still imposed on one third of the population. The result of this policy is given in Figure 6. The result of maintaining in lock down 1/3 of the population is represented by the green curves.



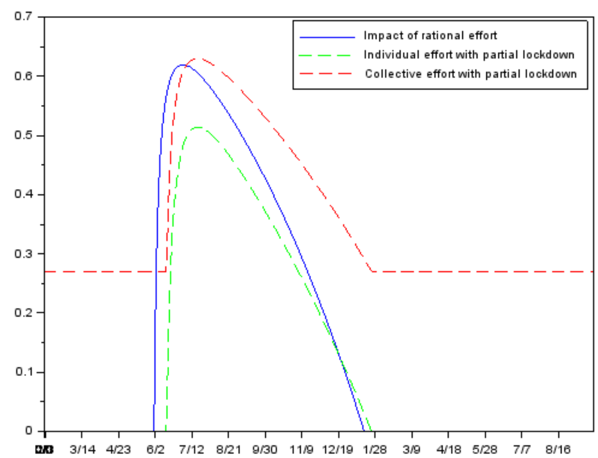
(a)



(b)



(c)



(d)

Figure 6: Impact of maintaining lockdown for 1/3 of the population, on daily reported cases (a), on the cumulative number of deaths (b) and on contact intensity $\phi(\epsilon)$ (c) and on effort (green curve is for individual effort, and red curve for collective effort) (d) for $\lambda = 10^{-3}$ and $k = 30$.

Figure 7 considers the same kind of policy, lockadown affecting 60% of the population. The result is given by the green curves.

3.3 Discussion

It is very clear that individual choices regarding self-protection have an important impact on the dynamics of the epidemics.Individuals adapt their self-protection effort in such a way that the number of reported cases reaches a plateau.

Freely chosen self-protection efforts are however not high enough to avoid a sizable epidemic rebound. And this holds even though we assume that individuals strongly overestimate the infection probability that can be computed from available data, as represented by parameter k (varying the size of this parameter changes the number of cases and deaths on a given period, but the profile of the epidemics remains the same).

The equilibrium self-protection effort to reduce contacts leads to a spread in infected cases, with a much lower maximum number of cases on a given date, but a much longer duration of the epidemics. Imposing prolonged lockdown on some proportion of the population leads to the same type of dynamics, with a still lower maximum number of cases and still longer duration of the epidemics. The number of deaths is very dependent on individual efforts and on public policies, both leading to a strong reduction compared with the situation in which individuals would not self-protect at all (as assumed in most epidemiological models).

References

[1] Augeraud-Véron, E., Lifting the COVID-19 lockdown: different scenarios for France, WP.

[2] Balinska, M., and Rizzo, C. (2009). Behavioural responses to influenza pandemics: what do we know?. PLoS currents, 1.

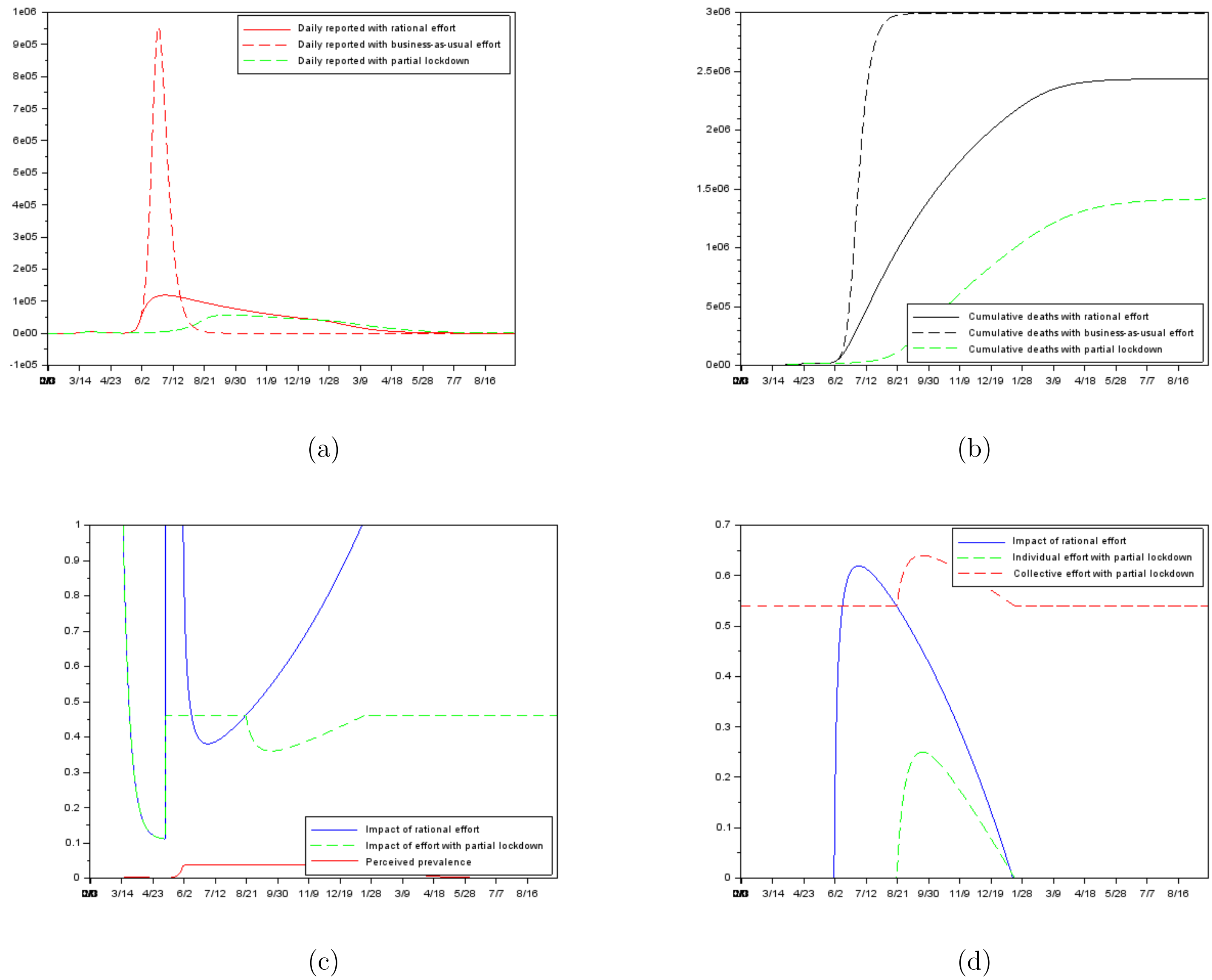


Figure 7: Impact of maintaining lockdown for 6/10 of the population, on daily reported cases (a), on the cumulative number of deaths (b) and on contact intensity $\phi(\epsilon)$ (c) and on effort (green curve is for individual effort, and red curve for collective effort) (d) for $\lambda = 10^{-3}$ and $k = 30$.

[3] Buonomo, B., d'Onofrio, A., and Lacitignola, D. (2008). Global stability of an SIR epidemic model with information dependent vaccination. Mathematical biosciences, 216(1), 9-16.

[4] Capasso, V., and Serio, G. (1978). A generalization of the Kermack-McKendrick deterministic epidemic model. Mathematical Biosciences, 42(1-2), 43-61.

[5] Chowell, G., Hengartner, N. W., Castillo-Chavez, C., Fenimore, P. W., Hyman, J. M. (2004). The basic reproductive number of Ebola and the effects of public health measures: the cases of Congo and Uganda. Journal of theoretical biology, 229(1), 119-126.

[6] Courbage, C., Rey B., and Treich N. (2013). Prevention and Precaution. In: Dionne G. (eds) Handbook of Insurance. Springer, New York, NY.

[7] Ferguson, N. (2007). Capturing human behaviour. Nature, 446(7137), 733-733.

[8] Funk, S., Salathé, M., and Jansen, V. A. (2010). Modelling the influence of human behaviour on the spread of infectious diseases: a review. Journal of the Royal Society Interface, 7(50), 1247-1256.

[9] Geoffard, P. Y., and Philipson, T. (1995). The empirical content of canonical models of infectious diseases: the proportional hazard specification. Biometrika, 82(1), 101-111.

[10] Geoffard, P. Y., and Philipson, T. (1997). Disease eradication: private versus public vaccination. The American Economic Review, 87(1), 222-230.

[11] Green, E. C., Halperin, D. T., Nantulya, V., and Hogle, J. A. (2006). Uganda's HIV prevention success: the role of sexual behavior change and the national response. AIDS and Behavior, 10(4), 335-346.

[12] Havrànek, T. (2015). Measuring intertemporal substitution: The importance of method choices and selective reporting. Journal of the European Economic Association, 13(6): 1180-1204.

[13] Jullien, B., Salanié, B., Salanié, F. (1999). Should More Risk-Averse Agents Exert More Effort?. Geneva Risk and Insurance Review, 24: 1928. https://doi.org/10.1023/A:1008729115022

[14] Kraemer, M., Yang, C-H, Gutierrez, B., Wu, C-H, Klein, B., Pigott, D.M., Open COVID-19 Data Working Group, du Plessis, L., Faria, N.R., Li, R., Hanage, W.P., Brownstein, J.S., Layan, M., Vespignani, A., Tian, H., Dye, C., Pybus, O., Scarpino, S. (2020). The effect of human mobility and control measures on the COVID-19 epidemic in China. Science 01 May 2020: Vol. 368, Issue 6490, pp. 493-497. DOI: 10.1126/science.abb4218

[15] Z. Liu, P. Magal, O. Seydi, and G. Webb, Understanding unreported cases in the 2019-nCov epidemic outbreak in Wuhan, China, and the importance of major public health interventions, MPDI Biology Biology 2020, 9(3), 50

[16] Liu, Z., Magal, P., Seydi, O., and Webb, G. (2020). Predicting the cumulative number of cases for the COVID-19 epidemic in China from early data. arXiv preprint arXiv:2002.12298.

[17] Magal, P., Webb, G. (2020). Predicting the number of reported and unreported cases for the COVID-19 epidemic in South Korea, Italy, France and Germany. Italy, France and Germany (March 19, 2020).

[18] McKusick, L., Coates, T. J., Morin, S. F., Pollack, L., Hoff, C. (1990). Longitudinal predictors of reductions in unprotected anal intercourse among gay men in San Francisco: the AIDS Behavioral Research Project. American Journal of Public Health, 80(8), 978-983.

[19] Maier, Benjamin F., Dirk Brockmann (2020). Effective containment explains subexponential growth in recent confirmed COVID-19 cases in China. Science, 08 Apr 2020:eabb4557. DOI: 10.1126/science.abb4557

[20] A. Mignon Colombet and D. Floreancig, L'infraction de "mise en danger d'autrui" est à manier avec précaution , Tribune, Le Monde, 25 mars 2020.

[21] d'Onofrio, A. and Manfredi, P. (2009). Information-related changes in contact patterns may trigger oscillations in the endemic prevalence of infectious diseases. Journal of Theoretical Biology, 256(3), 473-478.

[22] Reluga, T. C., Bauch, C. T., Galvani, A. P. (2006). Evolving public perceptions and stability in vaccine uptake. Mathematical biosciences, 204(2), 185-198.

[23] Verelst, F., Willem, L., and Beutels, P. (2016). Behavioural change models for infectious disease transmission: a systematic review (2010–2015). Journal of The Royal Society Interface, 13(125), 20160820.

[24] Wang, W. (2012). Modeling adaptive behavior in influenza transmission. Mathematical Modelling of Natural Phenomena, 7(3), 253-262.

[25] https://www.worldometers.info/coronavirus/coronavirus-death-toll/ (accessed on May 10, 2020).

[26] Zhang, J., Litvinova, M., Liang Y., Wang, Y., Wang, W., Zhao, S., Wu, Q., Merler, S., Viboud, C., Vespignani, A., Ajelli, M., Yu, H. (2020). Changes in contact patterns shape the dynamics of the COVID-19 outbreak in China. Science. 29 Apr 2020: eabb8001. DOI: 10.1126/science.abb8001