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Authors	Guillaume Vandenbroucke
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The Mechanics of Individually- and Socially-Optimal Decisions during an Epidemic*

Guillaume Vandenbroucke†

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Abstract

I present a model where work implies social interactions and the spread of a disease is described by an SIR-type framework. Upon the outbreak of a disease reduced social contacts are decided at the cost of lower consumption. Private individuals do not internalize the effects of their decisions on the evolution of the epidemic while the planner does. Specifically, the planner internalizes that an early reduction in contacts implies fewer infectious in the future and, therefore, a lower risk of infection. This additional (relative to private individuals) benefit of reduced contacts implies that the planner's solution feature more social distancing early in the epidemics. The planner also internalizes that some infectious eventually recover and contribute further to a lower risk of infection. These mechanisms imply that the planner obtains a flatter infection curve than that generated by private individuals' responses.

Keywords: SIR model; epidemic; social optimum; social distance; contact rate

JEL codes: E1, H1, I1

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†Research Division, Federal Reserve Bank of St. Louis, P.O. Box 442, St. Louis, MO 63166, USA. Email: guillaumevdb@gmail.com.

1 Introduction

Why should social distancing be mandatory during an epidemic? In other words why would individual incentives lead to less social distancing than is socially optimal? These are the questions I discuss in this paper.

I develop a model where ex-ante identical individuals value consumption and leisure and receive income from work. Both work and leisure entail social interactions in such a way that social interactions are increasing (in a sense to be made precise below) in work time. I do not model savings nor testing, and I consider the wage rate to be exogenous.

I use an SIR-type description of the evolution of the epidemic. Specifically, I consider four compartments of the population: the susceptible, the infectious, the symptomatic and the recovered. I assume that both susceptible and infectious are asymptomatic. Thus, their behavior is the same. Infectious individuals become symptomatic after an incubation period of random length. Symptomatic individuals are quarantined and face, each period, a probability of dying and a probability of recovering. Recovered individuals cannot become infectious again. The socially- and economically-active population is the union of susceptible, infectious and recovered individuals.

A key object of interest is the contact rate: the number of contacts made by an average individual with other individuals in a period. In SIR-type models the contact rate, together with the relative sizes of the socially-active compartments of the population, dictate the flow of infectious each period. The contact rate is the choice variable in this analysis. At the individual's level a "low" contact rate (i.e. social distancing) implies a "low" risk of infection at the cost of "low" consumption. The model features heterogeneous contact rates since susceptible and infectious individuals on the one hand, and recovered individuals on the other hand, do not behave similarly. An SIR-type model with heterogeneous (albeit exogenous) contact rates can be found in Brauer (2008b).

A contribution of the model is the description of the contact rate. From the perspective of individuals, the contact rate is the mean λ of a Poisson distribution governing the number of meetings with other individuals in a period. I assume that λ is tied to individual work/leisure decisions in such a way that is is increasing in work time and consumption, i.e. working individuals are more likely to meet other individuals. Conversely, individuals seeking to reduce their exposure to others, need to reduce their labor supply and, thereby, their consumption. The law of large number implies that in the aggregate the number of contacts by an average individual is λ.

My focus is on the differences between the individually-optimal and the socially-optimal responses to the epidemic. There are two sources of differences. The first is that the planner internalizes the effect of the contact rate on the evolution of the disease over time. Individuals take this as given. The second is that the planner internalizes the effect of the contact rate on the number of meetings **and** on the probability that any single meeting involves an infectious. Individuals takes the latter as given—Section 2 describes this formally.

Results

I simulate a calibrated version of the model. The planner internalizes that lowering the contact rate—hence work and consumption—early in the outbreak implies a low stock of infectious later. This is utility enhancing since it implies that asymptomatic individuals are less likely to incur the cost of becoming sick and/or die in the future. Private individuals do not internalize this benefit of a low contact rate. Thus, the planner optimally chooses a lower contact rate, i.e. more social distancing, than individuals would choose early in the epidemics. Later in the epidemics, the planner raises the contact rate more than individuals would, because the planner internalizes that: (i) with a lower stock of infectious a higher contact rate does not imply a higher flow of infections; and (ii) some infectious eventually recover, and contribute to a reduction of the risk of infections for all. In sum the planner uses the contact rate as an instrument to reach herd immunity which is utility enhancing because it implies a low (or zero) risk that asymptomatic individuals become sick and/or dies.

A lesson from these simulations is that social distancing should be mandated early in an epidemic because individuals, left to their own devices, do not internalize all the benefits of social distancing.

Literature

There is a a growing list of papers on the "economics of the COVID-19 crisis," for lack of a better term. Garriga et al. (2020) give an organized literature review. I provide a (non-exhaustive) list of relevant papers in the reference section below.

Jones et al. (2020), among others, is close in spirit to the work I present: The authors analyze the differences between private and public incentives to implement mitigation strategies during the epidemic. They find, as I do, that the planner's incentives to mitigate—that is to impose social-distancing measures—early in the pandemic are stronger than private individual's. Differences between my approach and that of Jones et al. (2020) is the modeling of contacts between

individuals, and the emphasis on a dynamic incentive for the planner that is not shared by private individuals.

Garibaldi et al. (2020) also emphasize the difference between private and socially optimal solutions in a SIR-type model. Their model does not feature asymptomatic infectious individuals, but it features the medical congestions generated by large number of infectious individuals. They too find that private individuals restrict their social contacts to reduce the probability of an infection, but not enough compared with what the social optimum prescribes. They emphasize static and dynamic externalities as I do in Section 2.4.2.

The combination of SIR-type modeling and an economic framework can be found in many other recent papers such as Eichenbaum et al. (2020a) or Alvarez et al. (2020). These papers, including my own, face the following difficulties. First, on the "epidemiological front:" the COVID-19 epidemic is recent and key epidemiological features of the virus may not yet be well-known and/or widely accepted. It follows that (i) there is no such thing as a "standard" model of the COVID-19 epidemic; (ii) different authors will emphasize different epidemiological aspects (e.g. the presence, or not, of asymptomatic carriers; vaccines that may, or not, affect the transmission of the virus; the importance, or not, of age/gender/race/location/type-of-work heterogeneity, etc...); and (iii) the empirical values of the key epidemiological parameters of the model may not yet be well-known and/or reasonably reduced to a single value (e.g. the incubation period). Second, on the "economic front:" the COVID-19 epidemic is so disruptive to every aspect of the World's economic life that one is bound to choose to model only limited aspects of the economy (e.g. should it be the labor markets, or the financial markets or should the focus be on international trade? or on a closed economy? etc...)

The choices I made in this paper are to emphasize the role of asymptomatic carriers, to abstract from ex-ante heterogeneity, and to assume that economic activity is associated with social interactions. My modelling of the contact rate as the mean of a Poisson distribution allows for an easy distinction between an individual's contact rate and the aggregate contact rate. It thus reveals clearly the difference of incentives between the planner and individuals.

2 Model

2.1 Population dynamics

Time is discrete. The economy is populated by a large number of ex-ante a priori identical individuals. I denote the population size by P in a given period, and by P' in the following

period—I use a $'$ (prime) to denote next period's value for any object in the model. Upon the outbreak of a disease the following compartmentalization of the population can be devised.

1. There is a group of susceptible individuals denoted P_S and a group of infectious but asymptomatic individuals, P_I. Since members of P_S and P_I are asymptomatic, they do not know to which group they belong and their behavior, which I will describe in Section 2.4, is identical.

2. Members of the infectious group become symptomatic with probability σ each period. The size of the symptomatic population is denoted by P_M—M stands for the Medical care they may require. I assume that members of P_M are quarantined and do not participate in social activities.

3. Members of the symptomatic population die with probability γ or recover with probability ρ, such that $\gamma+\rho \leq 1$. The size of the recovered population is P_R. I assume that members of the recovered population may not become infectious again.

I refer to $P_S + P_I$ as the asymptomatic population. I refer to members of P_R as the recovered population. The socially-active population is $P_S + P_I + P_R$.

Let Λ denote the number of contacts—the contact rate—made by the average member of P_S or P_I in a period. Let Λ_R denote the contact rate of the average member of P_R. I discuss the determination of Λ and Λ_R in Section 2.4. A susceptible individual meeting an infectious individual becomes infectious with probability ϕ. I show in Appendix A that the flow of (new) infectious in a given period is

$$\text{new infections} = \phi\Lambda P_S \frac{P_I}{P_I + P_S + P_R\Lambda_R/\Lambda}. \tag{1}$$

The term ΛP_S is the number of meetings involving susceptible individuals in a period. The fraction $P_I/(P_I + P_S + P_R\Lambda_R/\Lambda)$ indicates the probability that a meeting is with an infectious individual. Note that, if $\Lambda = \Lambda_R$, that is if all individuals have the same contact rate, the probability of meeting an infectious is the proportion of infectious in the socially-active population. If recovered individuals have a relatively higher meeting rate than others, i.e., if $\Lambda_R/\Lambda > 1$, the probability of meeting an infectious is lower, all else equal.

The laws of motion for the various compartments of the population are

$$\begin{aligned} P_S' &= P_S - \phi\Lambda P_S \frac{P_I}{P_I + P_S + P_R\Lambda_R/\Lambda} \\ P_I' &= P_I\left(1-\sigma\right) + \phi\Lambda P_S \frac{P_I}{P_I + P_S + P_R\Lambda_R/\Lambda} \\ P_M' &= P_M\left(1-\rho-\gamma\right) + \sigma P_I \\ P_R' &= P_R + \rho P_M \end{aligned}$$

By construction, the total population is the addition of each compartment: $P = P_S + P_I + P_M + P_R$. It follows that $P' = P - \gamma P_M$. I use lower cases to represent population shares: $s = P_S/P$, $i = P_I/P$, etc... The dynamics of population shares is then

$$s' = S(s,i,r,m,\Lambda) = (1-\gamma m)^{-1}\left[s - \phi\Lambda\frac{si}{i+s+r\Lambda_R/\Lambda}\right] \quad (2)$$

$$i' = I(s,i,r,m,\Lambda) = (1-\gamma m)^{-1}\left[i\left(1-\sigma\right) + \phi\Lambda\frac{si}{i+s+r\Lambda_R/\Lambda}\right] \quad (3)$$

$$m' = M(i,m) = (1-\gamma m)^{-1}\left[m\left(1-\rho-\gamma\right) + \sigma i\right] \quad (4)$$

$$r' = R(r,m) = (1-\gamma m)^{-1}\left[r + \rho m\right]. \quad (5)$$

Note that P_S, P_I, s, i, etc... are stock variables while Equation (1) describes a flow. Similarly, the flow of symptomatic in a period is σP_I, the flow of recovered is ρP_M, etc...

This description of population dynamics is standard in discrete-time versions of SIR-type models in epidemiology—see Brauer (2008a) for a description of these models. The typical dynamics of an SIR-type model is as follows. At the start of an epidemic almost all the population is in the susceptible compartment and a small fraction is exogenously assigned to the infectious compartment. The stock of susceptible only decreases over the course of the epidemic, and the stock of recovered only increases. The stock of infectious and symptomatic exhibit $\cap$-shape trajectories. The tipping-point for the stock of infectious occurs when the flow of infectious equals the flow of symptomatic. This tipping point is certain to obtain because, as the stock of susceptible decreases, the flow of infectious eventually decreases. At some point, therefore, the flow of infectious is offset by the flow of symptomatic. Similarly, the tipping point for the stock of symptomatic occurs when the flow of symptomatic is eventually offset by the flow of recovered and death.

I conclude this section by a remark on the flow of infectious described by Equation (1): This flow is increasing in the contact rate and decreasing in the population of susceptible and infectious. It follows that a "high" contact rate does not imply a "high" flow of infectious when the stock of susceptible and/or the stock of infectious are "low" enough. I will refer to that observation

when I discuss the behavior of the model in Section 3.2.

2.2 Preferences

The disease is the only cause of death, i.e. conditional on not dying from it, individuals are infinitely-lived. They are endowed with 1 unit of time per period. The utility index is

$$\tilde{U}(c,\ell) = \omega \ln(c) + (1-\omega)\ln(\ell)$$

where c is consumption and ℓ is leisure time. I assume an individual's contact rate to depend on work and leisure time:

$$\lambda = \lambda_w(1-\ell) + \lambda_\ell \ell \Leftrightarrow \ell = \frac{\lambda_w - \lambda}{\lambda_w - \lambda_\ell}, \tag{6}$$

where λ_w and λ_ℓ are positive parameters such that $\lambda_w > \lambda_\ell$: one is more likely to meet others at work than off from work. Work time is thus increasing in an individual's contact rate, and leisure time is decreasing. An individual's budget constraint is

$$c = 1 - \ell, \tag{7}$$

where the wage rate is exogenous, constant and normalized to 1. Under these assumptions an individual's decision can be described either as a time allocation decision, i.e. a choice of leisure time, implying work, consumption and a contact rate; or it can be described as the choice of a contact rate, i.e. a decision to be more or less "social," implying work, leisure and consumption. I adopt the later approach. Given (6) and (7), the preferences over (c,ℓ) can be equivalently represented by the the function U:

$$U(\lambda) = \omega \ln(\lambda - \lambda_\ell) + (1-\omega)\ln(\lambda_w - \lambda). \tag{8}$$

This representation of contacts, work and leisure is stylized for simplicity. My goal is to represent a tradeoff between activities implying different intensity of contacts such that, at the same time, there is an economic cost associated with choosing the activity generating the least contacts. Individuals working at home may not be adequately represented by this model since they can, at the same time, work and experience fewer contacts. A possible extension of the model would be to allow for more activities, including work from home, each with their activity-specific contact rate.

2.3 The probability of remaining asymptomatic

The number of meetings an individual has in a period is a random variable, Z, following a Poisson distribution with mean given by the individual's contact rate, $\mathbb{P}(z,\lambda) \equiv \Pr(Z=z) = \lambda^z e^{-\lambda}/z!$ In Section 2.1 I defined Λ as the contact rate of the average member of P_S or P_I. I distinguish λ from Λ: the former is a choice by an individual taking the latter as given. Since all susceptible and infectious individuals are identical the following consistency condition must hold:

$$\lambda = \Lambda. \tag{9}$$

By the law of large numbers, the total number of meetings induced by susceptible individuals is ΛP_S, and the flow of new infections in a period is, indeed, given by Equation (1).

I now define the probability that an asymptomatic individual remains asymptomatic at the end of the period. Let $Q(\lambda,\Lambda)$ denote this probability for an asymptomatic with contact rate λ when the average contact rate of asymptomatic individuals is Λ. Given Λ, the probability that any meeting is with an infectious individual is

$$\iota(\Lambda) = \frac{i}{i+s+r\Lambda_R/\Lambda}. \tag{10}$$

The probability of being infected in one meeting is $\iota(\Lambda)\phi$, i.e. the probability that the meeting is with an infectious multiplied by the probability that this meeting ends with an infection. The probability of not becoming infected after z meetings is then $(1-\iota(\Lambda)\phi)^z$. It follows that the probability of not becoming infected in a period is

$$\pi(\lambda,\Lambda) = \sum_{z=0}^{\infty} \mathbb{P}(z,\lambda)(1-\iota(\Lambda)\phi)^z = e^{-\lambda\iota(\Lambda)\phi}. \tag{11}$$

Then

$$Q(\lambda,\Lambda) = \frac{i}{i+s}(1-\sigma) + \frac{s}{i+s}\left[\pi(\lambda,\Lambda) + (1-\pi(\lambda,\Lambda))(1-\sigma)\right],$$

where the first term is the probability of being infectious and not developing symptoms—recall that asymptomatic do not know whether they are susceptible of infectious. The second term is the probability of being susceptible and not developing symptoms. The latter, in brackets, is the probability of not becoming infected or becoming infected and not developing symptoms.

Some explanations are necessary. I assume a form of bounded rationality for individuals: they do not keep track of the number of realized contacts in a period. The contacts that could lead to an infection during a day are not all "planned meetings" or "conversations" easily remembered. They may include people "met" in an elevator or in a waiting line, a clerk in a store or a waiter

at a restaurant etc... Thus, it is sensible to assume that individuals do not keep a record of them. This assumption voids the heterogeneity that develops during a period since different individuals may experience different number of contacts even though they may have chosen the same λ. Under the bounded rationality assumption, asymptomatic individuals are identical at the start of any period, and consider $i/(i+s)$ as their best estimate for the probability of being infectious.

It is worth noting that this bounded rationality assumption is for simplicity. Absent this assumption, the model would feature heterogeneity in the history of contacts for each individual. Individuals would also have to form beliefs about being susceptible or infectious, and would update them each period after observing their realized contacts. Such model would be significantly more complicated.

The expression for $Q(\lambda, \Lambda)$ simplifies to

$$Q(\lambda, \Lambda) = \frac{i}{i+s}(1-\sigma) + \frac{s}{i+s}(1-\sigma(1-\pi(\lambda, \Lambda))). \tag{12}$$

Note that the first component of $Q(\lambda, \Lambda)$ does not depend on λ. When the proportion of infectious is large an asymptomatic assesses the probability of being infectious to be large and the probability to remain asymptomatic to be mostly determined by the duration of the incubation period, regardless of attitude toward social interactions.

2.4 Value functions

Recovered individuals are immune and are aware of it. Their value is therefore

$$V_R = \max_{\lambda \in [\lambda_\ell, \lambda_w]} U(\lambda) + \beta V_R \tag{13}$$

where β is a subjective discount factor. As indicated earlier, I use Λ_R to denote the optimal choice of recovered individuals: $\Lambda_R = \omega\lambda_w + (1-\omega)\lambda_\ell$ and $V_R = U(\Lambda_R) + \beta V_R$.

Symptomatic individuals are not socially active. Their value is

$$V_M = \delta + \beta\left[(1-\gamma)(1-\rho)V_M + (1-\gamma)\rho V_R + \gamma\Delta\right]. \tag{14}$$

In this expression δ is the flow of utils in the current period. The continuation value comprises three terms. These are, in order: (i) the individual does not die and remains symptomatic; (ii) the individual does not die and recovers; and (iii) the individual dies. The parameter Δ

indicates the "(dis)utility of death."

2.4.1 *The individual's solution*

The optimization problem for an asymptomatic individual is

$$\begin{aligned} V_A(s,i,r,m) &= \max_{\lambda\in[\lambda_\ell,\lambda_w]} U(\lambda) + \beta Q(\lambda,\Lambda) V_A(s',i',r',m') + \beta\left(1-Q(\lambda,\Lambda)\right) V_M \\ \text{s.t.} &\quad \text{Equations (2)–(5).} \end{aligned} \tag{15}$$

Let λ^* denote the solution. At an interior λ^* satisfies the following first-order condition

$$U_\lambda(\lambda^*) + \beta Q_\lambda(\lambda^*,\lambda^*)\left[V_A(s',i',r',m') - V_M\right] = 0. \tag{16}$$

Equation (16) indicates that, at an optimum, the marginal cost of a reduction in the contact rate is offset by the marginal value of remaining asymptomatic. The latter is the product of the marginal effect of the contact rate on the probability of remaining asymptomatic and the value of being asymptomatic over being symptomatic.

A few observations are in order at this stage. First, the marginal probability, Q_λ, is evaluated at (λ^*,λ^*) to impose consistency between individual and aggregate behavior. Second, Equation (16) is derived under the assumption that: (i) An asymptomatic individual does not internalize the effect of his decisions on the evolution of the disease; (ii) An asymptomatic individual does not internalize the effect of his decisions on the probability that any given meeting be with an infectious, i.e. the individual takes Λ as given.

It is convenient to write (16) as

$$U_\lambda(\lambda^*) = -\beta\frac{s}{i+s}\sigma\pi_\lambda(\lambda^*,\lambda^*)\left[V_A(s',i',r',m') - V_M\right], \tag{17}$$

where $\pi_\lambda(\lambda^*,\lambda^*) = -\iota(\lambda^*)\phi e^{-\lambda^*\iota(\lambda^*)\phi} < 0$. The left-hand side of (17) equals 0 at $\lambda = \Lambda_R$ since Λ_R is the solution of problem (13). It is immediate that $U_\lambda(\lambda) \to +\infty$ as $\lambda \to \lambda_\ell$. The right-hand side of (17) is decreasing in λ but it is finite at $\lambda = \lambda_\ell$ and strictly positive at $\lambda = \Lambda_R$ as long as $V_A > V_M$.[1] It follows from these observations that the individual's solution is such that $\lambda^* \leq \Lambda_R$.

It follows from Equations (11) and (12) that $\pi(\lambda,\Lambda)\big|_{i=0} = 1$ and $Q(\lambda,\Lambda)\big|_{i=0} = 1$. Thus, at the end of an epidemic, when the proportion of infectious reaches 0, the probability of remaining

[1] If there existed a state in which $V_M \geq V_A$, the "disease" would not be a nuisance. The condition $V_A > V_M$ can be guaranteed to hold by appropriately choosing the parameters δ and Δ.

asymptomatic reaches 1. Furthermore, Equations (2)-(5) reveal that: the proportion of susceptible remains constant at a level (possibly 0) which I label s_{LR}; the population of symptomatic converges to 0; and the population of recovered converges to a constant (possibly 1) which I label r_{LR}. Thus, the state of the population is constant. In that case problem (15) reads

$$V_A(s_{LR}, 0, r_{LR}, 0) = \max_{\lambda \in [\lambda_\ell, \lambda_w]} U(\lambda) + \beta V_A(s_{LR}, 0, s_{LR}, 0).$$

This is the same as Equation (13). Thus, $V_A(s_{LR}, 0, r_{LR}, 0) = V_R$, and the long-run value of λ^* is Λ_R.[2]

2.4.2 *The planner's solution*

The planner solves an optimization problem similar to that of an individual:

$$\begin{aligned} V_A(s,i,r,m) &= \max_{\Lambda \in [\lambda_\ell, \lambda_w]} U(\Lambda) + \beta Q(\Lambda,\Lambda) V_A(s',i',r',m') + \beta\left(1 - Q(\Lambda,\Lambda)\right) V_M \\ \text{s.t.} &\quad \text{Equations (2)–(5).} \end{aligned} \tag{18}$$

Let Λ^* denote the planner's solution. At an interior, Λ^* satisfies a first-order condition different than that of an individual (see Equation 16) because the planner internalizes effects that an individual does not. Using the fact that $S_\Lambda = -I_\Lambda$ (see Equations 2 and 3) the planner's first-order condition is

$$\overbrace{U_\lambda(\Lambda^*) + \beta\left[Q_\lambda(\Lambda^*,\Lambda^*) + Q_\Lambda(\Lambda^*,\Lambda^*)\right]\left[V_A(s',i',r',m') - V_M\right]}^{\text{static incentive}} + \underbrace{\beta Q(\Lambda^*,\Lambda^*)\left[V_{A,s}(s',i',r',m') - V_{A,i}(s',i',r',m')\right] S_\Lambda(s,i,r,m,\Lambda^*)}_{\text{dynamic incentive}} = 0. \tag{19}$$

The term "static incentive" refers to the fact that the trade-off summarized by this part of the first-order condition deals with static considerations: the effect of Λ on contemporaneous utility and on the probability of remaining asymptomatic between the current and the next period. Similarly, the term "dynamic incentive" refers to the fact that this part of the first-order condition represents the planner's ability to internalize that a change in Λ affects the trajectory of the disease in the future.

The envelope condition implies the following derivatives for the planner's value of an asymp-

[2]The same observation can be made by inspecting Equation (16). Since Q is constant its derivative is 0 and, therefore, $U_1(\lambda^*) = 0$ which is also the solution of problem (13).

tomatic (I use Q to denote $Q\left(\Lambda^*, \Lambda^*\right)$ and a prime to denote a function evaluated at (s', i', r', m')):

$$\begin{aligned}
V_{A,s} &= \beta Q\left[V'_{A,s}S_s + V'_{A,i}I_s\right], \\
V_{A,i} &= \beta Q\left[V'_{A,s}S_i + V'_{A,i}I_i + V'_{A,m}M_i\right], \\
V_{A,m} &= \beta Q\left[V'_{A,s}S_m + V'_{A,i}I_m + V'_{A,r}R_m + V'_{A,m}M_m\right], \\
V_{A,r} &= \beta Q\left[V'_{A,s}S_r + V'_{A,i}I_r + V'_{A,r}R_r\right].
\end{aligned}$$

Consider the planner's value of a marginal susceptible, $V_{A,s}$. The marginal susceptible implies S_s susceptible and I_s infectious in the next period. Thus, the value of the marginal susceptible is the discounted value of these changes in the stock of susceptible and infectious. A marginal infectious implies M_i symptomatic in the next period which may die or recover. Specifically, the marginal symptomatic implies R_m recovered individuals in the next period. From the planner's perspective a marginal infectious may have a positive value since it may, eventually, lead to enough recovered to lower the probability of a meeting being with an infectious.

The differences between an individual's first-order condition (Equation 16) and the planner's first order condition are two-fold. First, the static incentive of the planner comprises the term $Q_\Lambda(\Lambda^*, \Lambda^*)$ to represents the planner's ability to internalize how a change in the contact rate affects the probability that any meeting be with an infectious. This term is absent from the individual's first-order condition. The term $Q_\lambda(\Lambda^*, \Lambda^*)$ represents how a change in the contact rate affects the probability of meetings. It is common to both the individual and the planner's first-order condition. Second, the dynamic incentive is absent from the individual's first-order condition.

Comparing Equations (16) and (19) with their counterpart in Garibaldi et al. (2020) a few observations can be made. First, as in their paper there are similar terms in both first-order conditions. The differences between the individual and planner's first-order conditions indicate the externalities in the environment. Second, one source of externality present in their paper but not in mine is the medical congestion externality. A contribution of the simple model I present here is thus to emphasize how the planner's incentive differ from the individual's even in the absence of considerations such as medical capacity.

I make two final remarks. First, it is immediate that the planner's solution in the long-run, that is once $i = 0$, is the same as the individual solution in the long-run: Λ_R. Second, the planner I discuss in this section does not allocate the contact rate of recovered individuals, Λ_R. I make this assumption for simplicity, and return to its implications in Section 3.2.

3 Numbers

3.1 Calibration

The exercise I consider in this section is a numerical example to illustrate the behavior of the model. It is not a quantitative study of the COVID-19 epidemic. A model period is a day. The Center for Disease Control indicates that the incubation period for COVID-19 ranges from 2 to 14 days with a median of 4-5 days.[3] I use 5 days and set $\sigma = 1/5$ since the expected time before symptoms, in the model, is $E_\sigma \equiv 1/\sigma$.

The WHO-China joint mission on COVID-19 indicates the median time for recovery for mild cases is about 2 weeks (3-6 weeks for patients with severe or critical disease). The time from the appearance of symptoms to death, among the patients who have died, ranges from 2 to 8 weeks.[4] In the model the expected duration before recovery, conditional on not dying, is E_ρ; the expected duration before death, conditional on not recovering, is E_γ:

$$E_\rho \equiv \frac{(1-\gamma)\rho}{\left(1-(1-\gamma)(1-\rho)\right)^2} \quad \text{and} \quad E_\gamma \equiv \frac{(1-\rho)\gamma}{\left(1-(1-\gamma)(1-\rho)\right)^2}.$$

I choose ρ and γ to imply $E_\rho = 14$ and $E_\gamma = 28$. This yields $\rho = 0.79\%$ and $\gamma = 1.58\%$.

I set $\beta = 0.99$. I set $\lambda_\ell = 1$ and $\lambda_w = 60$. At $\lambda = 60$, the 95% confidence interval for the number of individuals met in a day is $[45, 76]$. At $\lambda = 1$ the 95% confidence interval is $[0, 3]$. I set ω such that, in the absence of an epidemic (or equivalently for a recovered individual), work time amounts to 40 hours per week: $1 - \ell = 40/112$.[5] This yields $\omega = 0.36$. Given $\ell = 1 - 40/112$, Equation (6) implies $\Lambda_R = 22.07$. Thus, in the absence of an epidemic, the 95% confidence interval for the number of individuals met in a day is $[13, 32]$.

I follow Jones et al. (2020) and set Δ, the disutility of death, to ten times Gross Domestic Product per capita. In this model this is $10 \times (1 - \ell)$ where $\ell = 1 - 40/112$ as, in the absence of an epidemic, Gross Domestic Product per capita is labor income. I set δ, the disutility of being symptomatic, to $\Delta/2$.

Finally, I set ϕ, the probability of infection in a meeting with an infectious so as to imply a basic reproduction number of 2.5 at the start of the epidemic. The reproduction number is

[3] https://www.cdc.gov/coronavirus/2019-ncov/hcp/clinical-guidance-management-patients.html

[4] https://www.who.int/docs/default-source/coronaviruse/who-china-joint-mission-on-covid-19-final-report.pdf

[5] I assume that one needs 8 hours per day for sleep and minimal personal care. There are, therefore, $7 \times (24 - 8) = 112$ hours in a week.

Preferences	$\beta = 0.99$, $\omega = 0.36$ $\Delta = 3.57$
	$\delta = 1.78$
Epidemic	$\sigma = 1/5$, $\rho = 0.79\%$, $\gamma = 1.58\%$
	$\phi = 2.2\%$
Social interactions	$\lambda_\ell = 1$, $\lambda_w = 60$

Table 1: Parameters

the number of secondary cases generated by 1 infectious in a population where everyone else is susceptible. In the model, at the onset of the epidemics, an infectious individual's contact rate is Λ_R until symptoms develop and that individual becomes quarantined. This takes on average $1/\sigma$ periods. The probability that a meeting yields an infection is ϕ. Hence, the basic reproduction number is $\phi\Lambda_R/\sigma$. This implies $\phi = 2.2\%$.

3.2 Discussion

I assume that the initial proportion of infected is 1/1000. Figure 1 shows the trajectory of the epidemics under both the private and the planner's solution. Figure 2 shows the corresponding contact rate.[6] Under the private response the proportions of infectious and symptomatic peak at significantly higher levels (24% and 82%, respectively) than under the planner's response (3% and 35%, respectively). Under the planner's response the proportion of infectious takes longer to decline, however. It reaches 1/1000 (the initial proportion) on day 224 versus day 67 under the private response. Furthermore, the fraction of population remaining susceptible at the end of the epidemic is significantly higher under the planner's response than under the private response: 4.8% v. 0.5%. That is, under the planner's response 95.2% of the population eventually gets infected while, under the private response it is 99.5% of the population.

The sole reason for the different dynamics presented in Figure 1 is the different behavior of the contact rate displayed in Figure 2. Why does the planner's response features a contact rate that (i) declines more initially than the private contact rate, and (ii) overshoots the long-run solution before settling down on it?

In Section 2.4 I indicated that the private solution for the contact rate is such that $\lambda^* \leq \Lambda_R$. The reason why a private individual does not find it optimal to ever raise λ^* above Λ_R is that any such increase entails, at the same time, a loss of current utility since U is maximized at Λ_R, and a reduced likelihood or remaining asymptomatic. The private individual is willing to lower

[6]The "wiggles" in the solution are artifacts of the solution method. I solved the private and the planner's problems on a $40 \times 41 \times 42$ grid for (s, i, m). A finer grid implies less "wiggles."

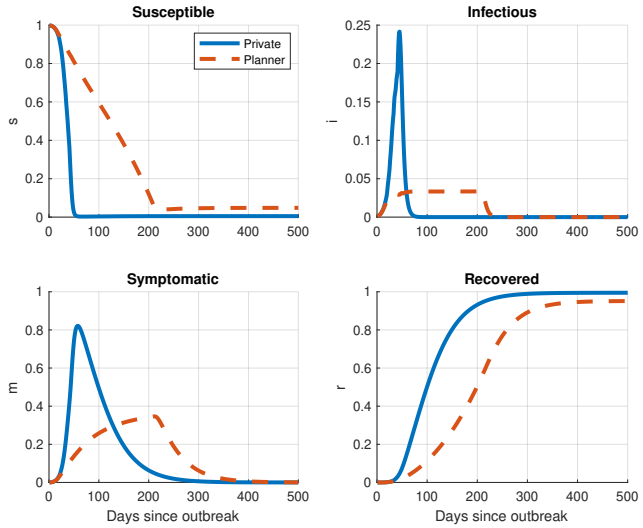

Figure 1: Baseline trajectory of the epidemic

Source: Author's calculations.

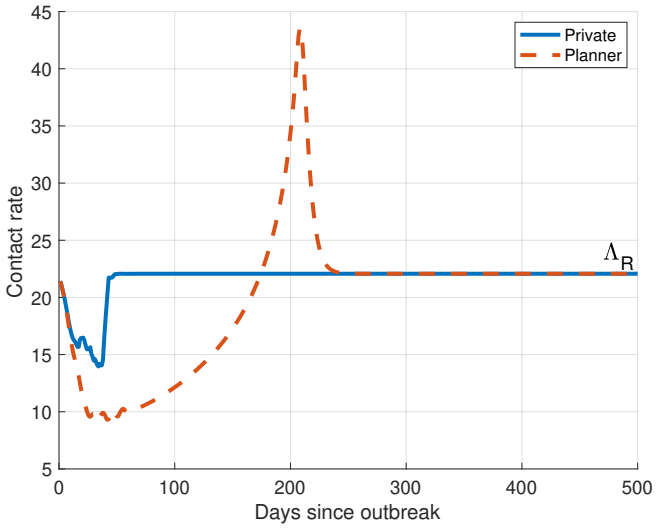

Figure 2: Baseline trajectory of the contact rate

Source: Author's calculations.

λ^* below Λ_R because the loss of current utility is compensated by a reduction in the probability of becoming symptomatic. This explains the behavior of the private contact rate in Figure 2.

The planner faces similar tradeoffs but also internalizes the effect of the contact rate on the evolution of the disease. A low contact rate early in the epidemic yields a benefit for the planner that is not internalized by a private individual: a lower stock of infectious in the future. Because of this added benefit the planner reduces the contact rate more than a private individual would in the early stage of the epidemic. This is visible in Figure 2. How does a low stock of infectious in the future benefit the planner, though? There are two mechanisms at play. First, a low stock of infectious implies a low risk of infection for an asymptomatic—see the effect of i in Equation (10). This is utility enhancing since an asymptomatic is then less likely to incur the cost of being sick and/or dying. Second, with a low stock of infectious the planner can raise the contact rate without raising infections. The planner understands, however, that some infectious eventually recover and contribute to further reducing the risk of infection for an asymptomatic—see the effect of r in Equation (10). These mechanisms can be interpreted as the planner using the contact rate to hasten herd immunity which, in this model, amounts to a low (or zero) risk that the representative asymptomatic individual becomes sick and/or dies. It is not necessary that the contact rate overshoots its long run value for this interpretation to hold. The overshooting itself may or may not take place depending upon parameter values. The key, here, is that the planner internalizes the benefit of a high contact rate later in the epidemics: it contributes to herd immunity and, thus, is utility enhancing.

In Section 2 I indicated that the planner does not choose Λ_R, the contact rate of recovered individuals. I made this assumption for simplicity. If the planner chose Λ_R, it would have an incentive to increase it during the epidemic in order to lower the probability that a susceptible meets an infectious. This would allow the planner to avoid a drastic reduction in the contact rate of asymptomatic. Note however, that this would come at a cost for recovered individuals who may have to work more than is individually optimal. Thus, the planner's objective would have to weight the welfare of asymptomatic individuals against that of recovered individuals.

3.3 Robustness

I conduct robustness experiments with respect to σ, the probability of an infectious becoming symptomatic, ρ the probability that a symptomatic recovers, γ the probability that a symptomatic dies and, finally, with respect to R_0, the reproduction number at the start of the epidemic. Figures 3–10 show the trajectory of the epidemic for a variety of parameter combinations. The main message from these figures is that the general patterns exhibited by the individual and the planner's response to the epidemic remain the same under alternative

calibration of the main "epidemiological" parameters of the model.

4 Conclusion

In this paper I asked: why would individual incentives lead to less social distancing than is socially optimal—and therefore why should social distancing be mandatory? I setup a model where the cost of social distancing is reduced consumption and the benefit is a lower risk of becoming infectious. The model reveals two source of differences between the planner's incentives and an individual's. First, unlike the planner, individuals take the probability that any meeting is with an infectious as given. Second, individuals take the evolution of the disease over time as given. The planner internalizes that a low contact rate early in the epidemic implies a low stock of infectious in the future; and that a low stock of infectious in the future reduces the risk of future infections. These differences imply that the planner chooses more social distancing early in the epidemic and obtains, as a result, a lower infection curve than that obtained by private individuals.

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A The flow of new infections

Suppose contact rates, that is the number of people met by a given person in a period, are group specific: Λ_I, Λ_S and Λ_R. Let p_{ir} be the fraction of contacts made by a member of group P_I that is with a member of group P_R. Define p_{sr}, p_{rr} etc... similarly. We have

$$\sum_{y\in\{i,s,r\}} p_{xy} = 1 \quad \text{for } x \in \{i,s,r\}.$$

The number of contacts by members of P_I with members of P_S is then $\Lambda_I P_I p_{is}$, and it must equal the number of contacts by members of P_S with members of P_I which is $\Lambda_S P_S p_{si}$. Thus, we have the following consistency conditions

$$\begin{aligned} P_I \leftrightarrow P_S &: \Lambda_I P_I p_{is} = \Lambda_S P_S p_{si} \\ P_I \leftrightarrow P_R &: \Lambda_I P_I p_{ir} = \Lambda_R P_R p_{ri} \\ P_S \leftrightarrow P_R &: \Lambda_S P_S p_{sr} = \Lambda_R P_R p_{rs} \end{aligned}$$

Define

$$p_{xy} = \frac{\Lambda_y P_y}{\Lambda_I P_I + \Lambda_S P_S + \Lambda_R P_R}.$$

It is then immediate that the consistency conditions above are satisfied:

$$\begin{aligned} \Lambda_I P_I p_{is} &= \Lambda_I P_I \frac{\Lambda_S P_S}{\Lambda_I P_I + \Lambda_S P_S + \Lambda_R P_R} = \Lambda_S P_S p_{si}, \\ \Lambda_I P_I p_{ir} &= \Lambda_I P_I \frac{\Lambda_R P_R}{\Lambda_I P_I + \Lambda_S P_S + \Lambda_R P_R} = \Lambda_R P_R p_{ri}, \\ \Lambda_S P_S p_{sr} &= \Lambda_S P_S \frac{\Lambda_R P_R}{\Lambda_I P_I + \Lambda_S P_S + \Lambda_R P_R} = \Lambda_R P_R p_{rs}. \end{aligned}$$

New infections result from a susceptible meeting with an infectious. The number of meetings made by a susceptible is $\Lambda_S P_S$ and a fraction p_{si} are with other infectious. Let ϕ denote the probability that a meeting results in an infection. The number of new infections in a period is then

$$\text{new infections} = \Lambda_S \phi P_S p_{si} = \Lambda_S \phi P_S \frac{\Lambda_I P_I}{\Lambda_I P_I + \Lambda_S P_S + \Lambda_R P_R}$$

If meeting rates were identical, i.e., $\Lambda_S = \Lambda_I = \Lambda_R = \Lambda$, the number of new infections in a period would be

$$\Lambda \phi P_S \frac{P_I}{P_I + P_S + P_R}.$$

If susceptible and infectious people have the same meeting rate, $\Lambda_I = \Lambda_S = \Lambda$, and if the meeting rate of recovered is different, $\Lambda_R \neq \Lambda$, then the number of new infections in a period would be

$$\Lambda \phi P_S \frac{P_I}{P_I + P_S + P_R \Lambda_R / \Lambda}.$$

B Robustness

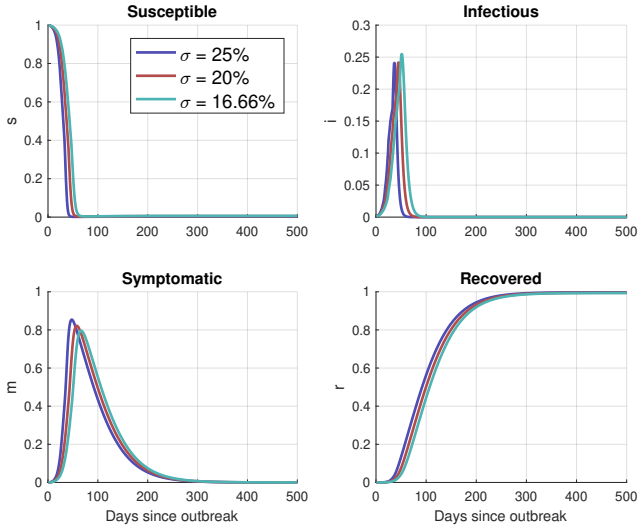

Figure 3: Trajectory of the epidemic – private response, various σ

Source: Author's calculations.

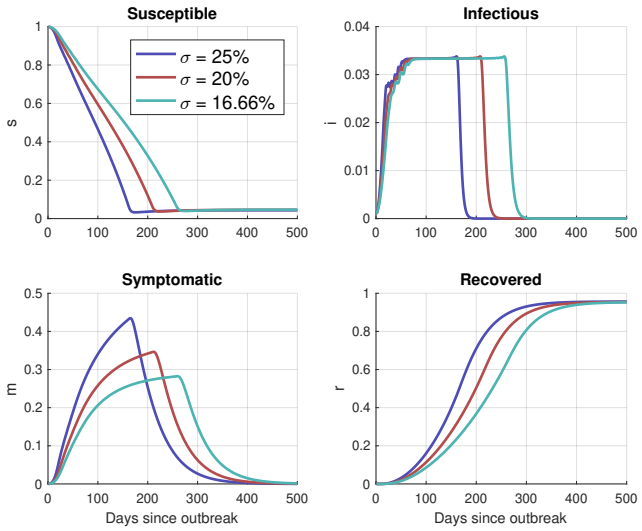

Figure 4: Trajectory of the epidemic – planner's response, various σ

Source: Author's calculations.

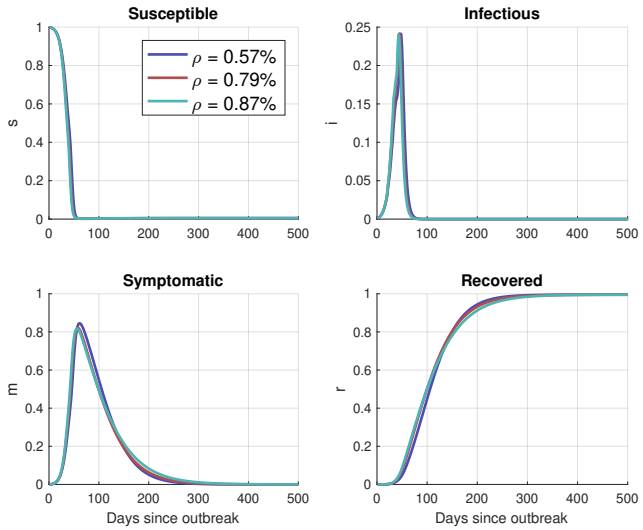

Figure 5: Trajectory of the epidemic – private response, various ρ

Source: Author's calculations.

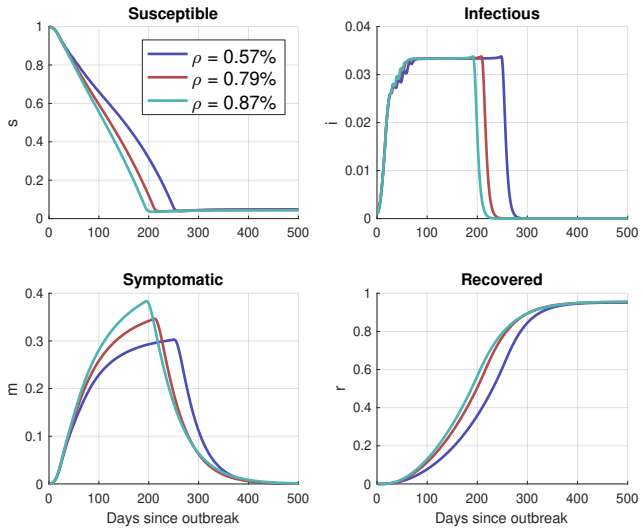

Figure 6: Trajectory of the epidemic – planner's response, various ρ

Source: Author's calculations.

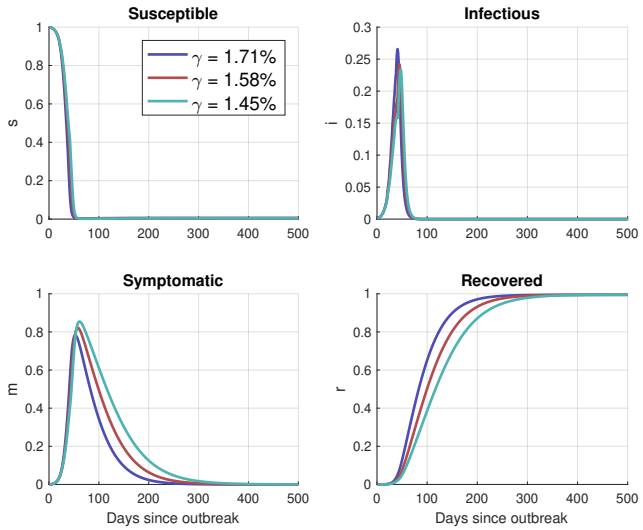

Figure 7: Trajectory of the epidemic – private response, various γ

Source: Author's calculations.

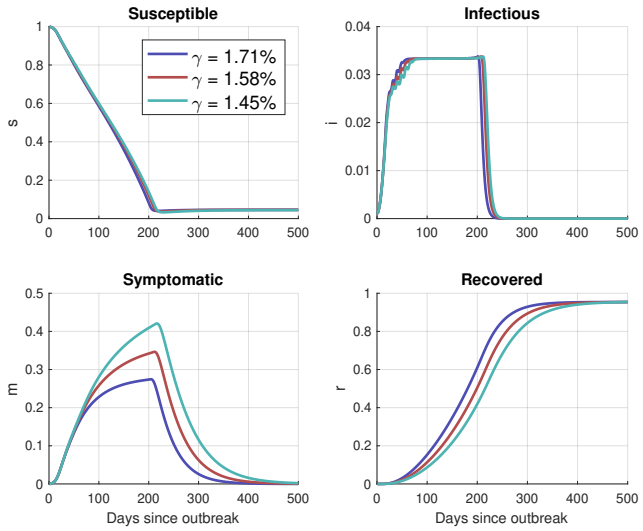

Figure 8: Trajectory of the epidemic – planner's response, various γ

Source: Author's calculations.

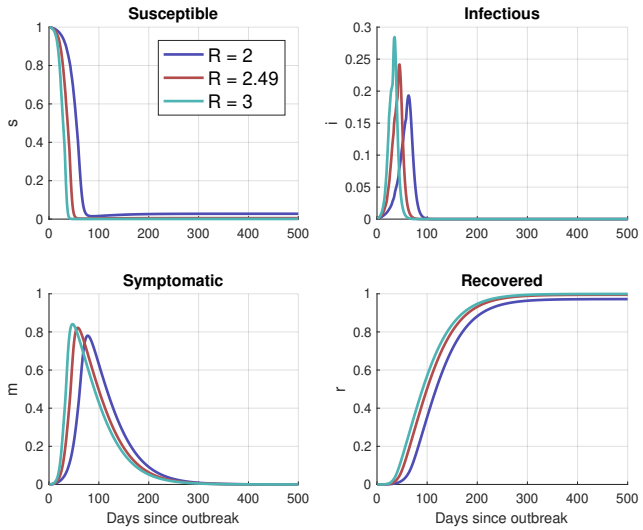

Figure 9: Trajectory of the epidemic – private response, various R_0

Source: Author's calculations.

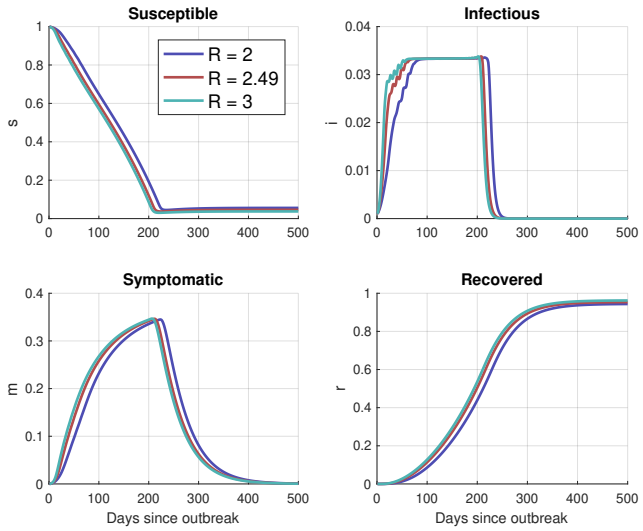

Figure 10: Trajectory of the epidemic – planner's response, various R_0

Source: Author's calculations.