Policy During an Epidemic With Super-Spreaders

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Abstract

I study mitigation policies during an epidemic when individuals vary in their degree of interaction with others. Individuals with a high degree are more likely to contract the disease and, once infected, they transmit the disease to more individuals in expectation. Thus, the presence of high degree individuals fuels the epidemic initially. However, this also means that the high degree individuals are more likely to become immune, and this lowers the reproduction number relatively quickly. That the buildup of immunity is correlated with social interactions has implications for policy. In an SIR model I compare a lockdown in which all individuals are forced to have the same degree with a more liberal policy in which the degree is lowered proportional to the original degree. The lockdown has to be in place longer than the liberal policy to limit the number of deaths to the same extent. The reason is that under the lockdown the high degree agents do not build up immunity faster than the low degree agents. When the policy is lifted, these high degree agents will fuel a second wave of infections. Under the liberal policy, average immunity is the same as under the lockdown, but degree-weighted immunity is larger, leading to a less severe second wave once policy is lifted. I show that the same implications for (optimal) policy hold in a model with economic behavior.

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JEL codes: E1, I1, D62, H0

Super-spreaders are individuals who infect a disproportionate number of other individuals. Super-spreaders are thought to be important for the spread of respiratory diseases such as MERS and SARS (Wong et al., 2015). There is evidence that super-spreaders also play an important role in the spread of COVID-19 (Hu et al., 2020).

It is not well understood what makes that some individuals turn out to be a super-spreader (Stein, 2011). The expected number of people an infected individual transmits the disease to equals the number of (susceptible) people one meets (the degree) times the probability of transmission conditional on meeting someone (the transmission risk). When super-spreaders, for instance, produce more of the virus and/or sneeze and cough more this corresponds to variation in the transmission risk. When on the other hand super-spreaders have more social contacts this corresponds to heterogeneity in the degree. In this paper, I study the phenomenon of super-spreaders through the lens of an epidemiological model with heterogeneity in the degree and assume that the transmission risk is the same across individuals.[1] Clearly, the degree constitutes an upper bound on how many individuals one can transmit a virus to, and agents with a high degree are more likely to become a super-spreader.

I first analyze an SIR model with a degree distribution. Individuals can be in one of four possible health sates. They can be susceptible to the disease and if they get in contact with an infected individual there is a probability that they get infected as well. Infected individuals recover and die according to a Poisson process. If the individual has recovered, I assume that this individual is immune to the disease for the foreseeable future.

The probability that a susceptible individual gets infected during a (short) period of time equals the transmission risk times the number of infected individuals one meets.[2] The latter equals the degree times the probability that a given interaction is with an infected individual. I assume random meetings such that the probability that a given interaction is with an infected individual is the same across individuals. As high degree individuals meet more people they are more likely to get infected during the early phase of the epidemic. Furthermore, because an individual is more likely to meet someone who has a high degree, this implies that the probability that a given interaction is with an infected individual exceeds the overall infection rate in the population. This makes that the disease spreads initially rapidly compared to a model in which all agents have the same degree.[3] However, as time progresses, a larger share of the high degree individuals will be immune to the disease compared to the low degree individuals, which limits the spread of the disease.

That high degree individuals are more likely to get infected and, therefore, are more likely to become immune has implications for policy. I compare two types of temporary policies that affect the degree. The first policy is an authoritarian policy in which all agents are forced to have the same (low) degree. This corresponds to a strict lockdown in which

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[1]For instance, Reich et al. (2020) also model super-spreaders by heterogeneity in the degree.

[2]I assume that all infected individual are infectious.

[3]When all individuals have the same degree, the probability that a given social contact is infected equals the infection ration in the population.

individuals are only allowed to go out for essential reasons such as doing groceries. Such a lockdown was implemented in, for instance, Wuhan and several Southern European countries during early 2020. I contrast this with a more liberal policy in which all agents are discouraged to interact with others. I assume this takes the form of all individuals having their degree lowered by the same proportion. An example of a liberal policy is the approach that Sweden adopted. Since during the authoritarian regime the degree is the same for all individuals everyone is equally prone to the disease. Therefore, once the policy is lifted the high degree individuals have not build up excess immunity compared to the average individual. Hence, when the authoritarian policy is lifted the high degree agents fuel the epidemic again, leading to a severe second peak. Instead, during the liberal regime high degree agents are still more prone to the disease than low degree agents and therefore build up excess immunity. At the time the liberal policy is lifted average immunity in the population is the same as when the authoritarian policy is lifted, but *average immunity weighted by the degree* is larger for the liberal policy. This makes that under the liberal policy the second peak will be less severe as there are fewer susceptible/infected super-spreaders that can fuel the epidemic. Thus, if the two policies are in place for the same amount of time there will be fewer deaths in the liberal regime than in the authoritarian regime. In order to limit the number of deaths to the same extent the authoritarian policy has to be in place longer, which might come at a larger cost to the economy and welfare.

The above two policy experiments are not optimal as a benevolent social planner can for instance improve by lifting the policy gradually, while in the above I assume that the policy is revoked abruptly. In order to speak about welfare and optimal policy I bring the above model to a simple economic model. Individuals can consume a regular good and a social good. The social good entails meeting other people (e.g., going to a restaurant, the gym or taking the subway) while the regular good does not come with social interactions (e.g., cooking at home, doing exercises at home or driving a car). Individuals vary in their preference for the social good relative to the regular good which leads to heterogeneity in the degree. I refer to individuals that have a high preference for the social good as high degree or social individuals. Individuals receive a flow utility that is positive and therefore do not want to die. Thus, as the disease emerges susceptible individuals will lower their social consumption such that the probability that they get infected drops. In the competitive equilibrium all individuals lower their consumption of the social good which makes that the peak is lower and that the total number of deaths is lower than in the SIR model without behavior (this is for instance also found by Eichenbaum et al. (2020) when all agents have the same contact rate). It turns out that the social individuals lower their social consumption to a smaller extent in relative terms than the non-social individuals.

There are several externalities in this model, providing an opportunity for policy intervention.[4] I consider a social planner that does not know in which health state an individual is, but that can let the policy depend on the individuals' preference for the

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[4]See Section II for an overview of the externalities.

social good.[5] When the virus arrives the social planner does not find it optimal to lower consumption of the social good for the social individual to the same extent as for the non-social individual. The reason is the same as in the SIR model. The planner wants to build up degree-weighted immunity. Therefore, it finds it optimal to keep consumption of the social good initially high, after which it limits consumption severely such that the currently infected individuals do not spread the virus to too many other individuals. Importantly, as long as the value of life and the case fatality rate are sufficiently low it is not optimal to lower the amount of social interactions to a level that is about the same for both social and non-social individuals. If anything, the planner wants to constrain the social agent less. This is very different from the lockdowns that have been implemented around the world in the spring of 2020. They affect the social agents disproportionally.

In my calibration, the social planner chooses a severe but short-lived peak compared to the competitive equilibrium. One reason could be that the baseline model does not feature an ICU constraint. When I include an ICU constraint the social planner indeed wants to flatten the curve. However, it still wants to do so by reducing consumption of the social good more for the low social agent than for the high social agent.

Finally, I solve two version of a constrained planner. In the first version the planner chooses a time-varying maximum amount of social consumption, and this maximum is the same for all individuals. This is reminiscent of the above authoritarian regime and disproportionally affects the social agent. In fact, it turns out that the planner does not want to set this constraint at a very low level, and therefore the non-social agent is not directly affected by the constraint. The reason is that setting this constraint so low such that it is binding for the non-social agent would hurt welfare of the social agent a lot while immunity only builds up slowly. The constrained planner chooses to limit consumption of the social agent only after the first eight weeks of the epidemic, and then gradually lifts the policy. The second version of the constrained planner is a tax on consumption of the social good. This tax affects both the social and non-social agents. It is optimal to start with a low tax that increases gradually and reaches its maximum after the epidemic has peaked. It then slowly declines. In the liberal regime around 10% fewer people die due to the virus than in the authoritarian regime.

Related Literature

There is a growing body of research studying the interaction between the macroeconomy and an epidemic, and studying optimal policies during an epidemic. A subset of this literature includes Eichenbaum et al. (2020), Alvarez et al. (2020) and Jones et al. (2020). A large share of this literature does not take heterogeneity into account and the papers that do so focus on heterogeneity in age and heterogeneous risks (see, e.g., Acemoglu et al., 2020 and Glover et al., 2020). I add to this literature by studying policy when there

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[5]The social planner can however infer how likely it is that an individual is in each health state, based on its past social activity.

is heterogeneity in the degree. Brotherhood et al. (2020) show that constraining the young (i.e., the socially active) might backfire and might lead to more deaths in a model in which the young are less likely to die of the virus. I obtain similar results, but without having heterogeneity in the case fatality rate. My model shares with Krueger et al. (2020) that agents can substitute between different goods that differ in their risk of contagion.

Papers in the epidemiological literature that take the degree distribution into account include Pastor-Satorras and Vespignani (2001), Newman (2002) and Jackson and Rogers (2007). A large share of this literature focuses on how the degree distribution affects the basic reproduction number and the size of the population that will be infected eventually. In terms of policy, this literature focuses on preventive measures such as vaccination campaigns (Britton et al., 2007; Cohen et al., 2003). There is less work on how the degree distribution affects the results of mitigation policies during an epidemic. An exception is Britton et al. (2020) who recently made a similar argument as presented here. They show that, in a model with heterogeneity in social activity, less strict policies can lead to fewer deaths by making the second wave less severe. They consider preventive measures that affect all contact rates proportionally while I also study policies that can affect agents differently depending on their degree, which is closer to the lockdowns that have been implemented in reality. I furthermore add to this literature by studying optimal policy.

I SIR Model

I first present a standard deterministic SIR model (Kermack and McKendrick, 1927) with random meetings and a degree distribution. In this model, behavior does not respond to the epidemic. In the next section I embed this model in a simple economic model to study how behavior responds to the epidemic, and to study welfare and optimal policy.

Suppose there is a unit mass of individuals, that are in one of four possible health states. The four states are: susceptible to the disease (S), infected and infectious (I), recovered (R) and death (D). A susceptible individual can get infected when it meets an infectious individual. Therefore, the probability of getting infected during a period depends on the number of social interactions d during a period. Denote by f the probability that a given social interaction is with an infectious individual. Thus, $f \cdot d$ is the expected number of infectious individuals one meets during a period. When the transmission risk, p, is less than 1 an interaction with an infectious individual does not necessarily lead to transmission. The probability of getting infected during a period is the product of these three terms,

$$\text{Probability getting infected} = p \cdot f \cdot d. \tag{1}$$

Here, I have assumed that a period is short such that an individual can only be infected once during a period.

Let $P(d)$ be the measure of agents who have degree d. As individuals differ in the

probability of getting infected, the fraction of agents that is in each state depends on the degree. Therefore, denote by $S(d)$ the fraction of agents with a degree d that is susceptible, and define $I(d)$, $R(d)$, and $D(d)$ likewise. We have that $S(d) + I(d) + R(d) + D(d) = 1$ for each d. Then, the overall mass of agents that is susceptible equals $S = \sum_d P(d)S(d)$ and similarly for the other states.

The following difference equations hold for the four states

$$S_{t+1}(d) = (1 - p \cdot f_t \cdot d) \, S_t(d) \tag{2}$$

$$I_{t+1}(d) = (1 - \lambda_R - \lambda_D) \, I_t(d) + p \cdot f_t \cdot d \cdot S_t(d) \tag{3}$$

$$R_{t+1}(d) = R_t + \lambda_R I_t(d) \tag{4}$$

$$D_{t+1}(d) = D_t + \lambda_D I_t(d) \, . \tag{5}$$

The law of motion for S_t is based on (1). As there is a continuum of agents, the mass of agents that gets infected equals the mass of susceptible agents times the probability of getting infected. An infectious agent either recovers or dies at the rates λ_R and λ_D, respectively. Thus the outflow of the infectious state equals $(\lambda_R + \lambda_D)I_t(d)$ whereas the inflow equals the susceptible agents that get infected. The recovered and death states are absorbing states. This system of difference equations depends on the infection rates of the individuals with a different degree through f. That high degree individuals are more likely to be infected (during the early stage of the epidemic) implies that f is larger than the average infection rate in the population. The reason is that an individual is more likely to meet someone who has more social interactions. Thus, the chance that a given interaction is with an infectious individual equals

$$f_t = \frac{\text{Number of infected meetings}}{\text{Number of total meetings}} = \frac{\sum_d P(d)I_t(d)d}{\sum_d P(d)L_t(d)d} \, , \tag{6}$$

where in the denominator we have to take into account that only a mass of agents $L(d)$ is alive ($L(d) = S(d) + I(d) + R(d)$). When f is larger than the overall infection rate the disease spreads relatively fast. This is the case when high degree individuals are more likely to be infectious. Loosely speaking, this case corresponds to there being relatively many super-spreaders.

In the special case where all individuals have the same degree, f equals the overall infection rate as a share of the population alive: $f = \frac{I}{L}$. This is the model that is typically studied in the recent Macro-SIR literature, where $p \cdot d$ is governed by one parameter, the (effective) contact rate.

Reproduction Number

The basic reproduction number, $\mathcal{R}_0$, is the number of people that is infected by one infectious individual when the entire population is susceptible. And the effective reproduction number, $\mathcal{R}_e$, is the number of people that is infected on average by an infectious individual.

Holding behavior constant, the fewer susceptible individuals there are the lower $\mathcal{R}_e$. I will here study how the degree distribution affects these reproduction numbers, while holding the transmission risk p constant.

For simplicity, I assume for now that the lethality of the disease λ_D is equal to zero. Each infectious individual infects in expectation $\frac{p \cdot d}{\lambda_R}$ agents when the whole population is susceptible, as $p \cdot d$ is the number of effective contacts per period and $1/\lambda_R$ is the expected number of periods an individual is infectious. To get the basic reproduction number we need to use the average degree of the infectious agent: $\sum_d P(d)\frac{I(d)}{I}d$,

$$\mathcal{R}_0 = \frac{p}{\lambda_R} \sum_d P(d)\frac{I(d)}{I}d \tag{7}$$

$$= \frac{p}{\lambda_R \overline{d}} \sum_d P(d)d^2 \, . \tag{8}$$

The second equality follows from observing that at the onset of the epidemic the probability that an individual gets infected is proportional to d such that $\frac{I(d)}{I} = \frac{d}{\overline{d}}$, where $\overline{d}$ is the average degree. Equation (8) shows that holding the average degree constant, the higher the dispersion of the degree the higher $\mathcal{R}_0$. This result is well-known in the epidemiological literature (see, e.g., May and Anderson, 1988)

To get the effective reproduction number we need to multiply equation (7) with the probability that a meeting is with a susceptible agent, $\frac{\sum_d P(d)S(d)d}{\overline{d}}$,

$$\mathcal{R}_e = \frac{p}{\lambda_R} \left(\sum_d P(d)\frac{I(d)}{I}d \right) \cdot \left(\frac{\sum_d P(d)S(d)d}{\overline{d}} \right) \, . \tag{9}$$

Thus, both when the high degree nodes are less likely to be susceptible and less likely to be infectious the reproduction number goes down. When the number of infected is small, $\frac{I(d)}{I}$ is approximated by $\frac{S(d)d}{\sum_d P(d)S(d)d}$. Using this to rewrite equation (9) gives that

$$\mathcal{R}_e \approx \mathcal{R}_0 - \frac{p}{\lambda_R \overline{d}} \sum_d P(d)R(d)d^2 \, .$$

Thus, the higher immunity weighted by the squared degree, the lower the effective reproduction number and the slower the spread of the disease. For simplicity, I will refer to immunity weighted by the squared degree as degree-weighted immunity.

To see how the degree distribution affects transmission over the course of the epidemic I find it helpful to plot the effective reproduction number versus the susceptible population. Figure 1a shows this both when all individuals have the same degree and when there is heterogeneity in the degree. The disease parameters and the average degree are identical. Disease parameters are such that agents are on average infectious for two weeks, and $p = 0.07$. For the case of a degree distribution, I assume there are two type of agents. 30% of the population meets 25 people per week and the remaining 70% of the population meets 10 people per week. This is an assumption I maintain throughout. I will also
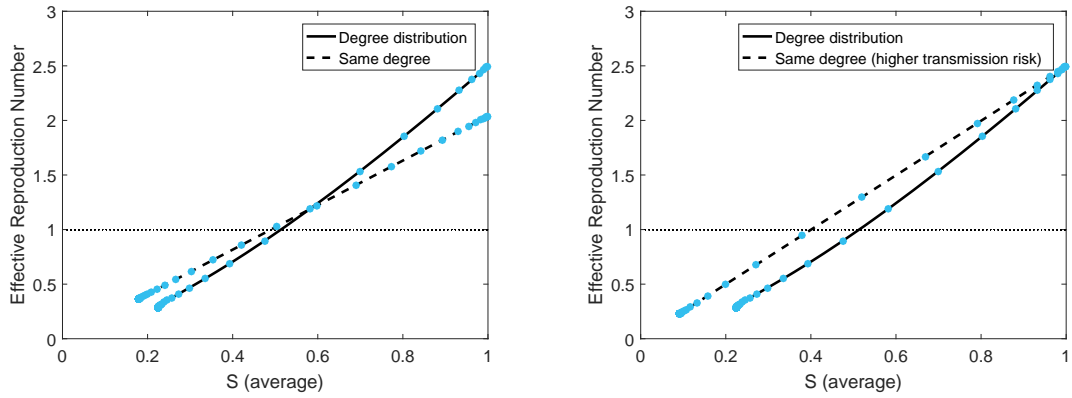
refer to the high degree agents as super-spreaders, as they will infect more people on average. Meeting 25 people per week might seem low. Whether this is a reasonable number depends on how intense social contact needs to be such that the virus has a reasonable chance of transmitting. These numbers are easily adjusted and the results will be identical as long as the degree is proportionally increased while the transmission risk falls at the same rate.[6] These numbers mean that (in a susceptible population) a high degree infectious individual will infect on average 3.5 people ($0.07 \cdot 25 \cdot 2$ weeks) while a low degree individual will infect 1.4 people. Taking into account that the social agents are more likely to be infected initially gives an $\mathcal{R}_0$ of 2.5. When individuals would have the same degree, $\mathcal{R}_0$ would be 2. 2.5 seems to be a reasonable approximation of the COVID-19 basic reproduction number (Riou and Althaus, 2020).

Let us first focus on the case of all individuals having the same degree. In this case, the relationship between the effective reproduction number and the susceptible population is linear. The slope of this line is $\frac{p\bar{d}}{\lambda_R}$. At the beginning of time $S = 1$ and over time S decreases and also the reproduction number decreases (thus in Figure 1a we go from right to left as time progresses and each blue dot represents a week). Once the reproduction rate is below one the disease will slowly die out. The S for which the reproduction rate equals one is also called the herd immunity threshold (to be precise 1 minus this S is the herd immunity threshold). When the mass of susceptible agents is below this threshold a new imported case will not lead to a re-occurrence of the epidemic. Of course, this does not mean that once this threshold is achieved by the epidemic running its course, nobody can be infected anymore. There is still a share of agents that is infectious of which each one will infect one other individual, which in turn will infect 0.99 individuals etc. Thus, when the epidemic runs its course without a policy intervention, the total mass of agents that contracts the disease is somewhat larger than the herd immunity threshold. This is shown by the point where the dashed line stops. Thus, in this example a bit more than 80% of individuals gets infected while the herd immunity threshold is at 50%.

That $\mathcal{R}_0$ is larger the more dispersed the degree is means that the initial spread of the disease is worse than when all individuals have the same degree. Does this mean that the epidemic is worse over the entire course of the epidemic when there is dispersion in the degree? No, not necessarily. The relationship between the effective reproduction number and the susceptible population is no longer linear when there is variation in the degree. When S is close to 1 the slope is larger than $\frac{p\bar{d}}{\lambda_R}$. The reason is that initially it are mainly the high degree individuals that become infected, which means they are also the mostly likely to become immune early on. The presence of these immune individuals with a high degree lowers the chance that an infectious individual meets a susceptible individual, and therefore, the reproduction number drops relatively quick. When $S = 0$ the slope is less than $\frac{p\bar{d}}{\lambda_R}$ (not shown in the graph because the epidemic never reaches that point in the state space). The reason why this slope is less than $\frac{p\bar{d}}{\lambda_R}$ is that by the time

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[6]For example, a calibration with $p = 0.007$ and the degree being 100 and 250 would give the same results.

(a) Different degree distribution but same transmission risk

(b) Different degree distribution but different transmission risk such that the basic reproduction number is the same

Figure 1: Relationship between susceptible population and effective reproduction number when the epidemic runs its course in the absence of policy intervention. Each blue dot represents a week.

the epidemic has ran its course most of the susceptible agents have a low degree. The probability that an infectious agent meets one of these individuals is low and therefore the reproduction number does not change much by varying S when S is close to zero. To summarize the above, when S is low the slope of the reproduction number curve is smaller when there is dispersion in the degree compared with no dispersion, while the slope is larger when S is large. Furthermore, when $S = 1$ the reproduction number is larger when there is degree dispersion, and for both cases, obviously, the reproduction number is zero when $S = 0$. Thus, the two curves must intersect and there is a threshold $\hat{S}$ below which the reproduction rate is lower for the case of degree dispersion. In Figure 1a this threshold is reached when the reproduction number is still above 1 which means that the herd immunity threshold is lower when there is dispersion in the degree, and eventually fewer people will contract the disease. This is of course does not hold generally. Suppose the transmission risk p would be sufficiently low such that the basic reproduction number in the case of a constant degree is only marginally above 1. Then, after an instant herd immunity is reached, while with dispersion in the degree, $\mathcal{R}_0$ is substantially above 1 such that more people need to contract disease before herd immunity is reached.

Now let us consider another experiment. Again consider one world in which all individuals have the same degree and one world where there is dispersion in the degree. But now assume that the transmission risk is larger when there is a homogeneous population such that $\mathcal{R}_0$ is identical in both worlds. This experiment corresponds to a world in which we empirically observe $\mathcal{R}_0$ but do not know the underlying degree distribution. Figure 1b shows the corresponding relationship between S and the reproduction number. Holding $\mathcal{R}_0$ constant, the larger the degree dispersion the lower the herd immunity threshold. With these numbers, a constant degree implies a herd immunity threshold of 60% and a threshold of 50% when there is degree dispersion. That heterogeneity in the degree lowers
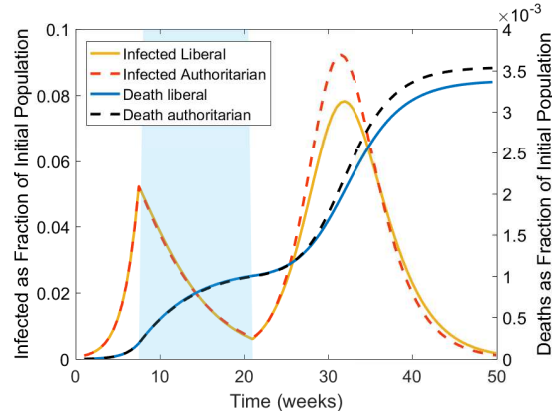
herd immunity is also discussed in Britton et al. (2020) and Gomes et al. (2020).

Now suppose there is a vaccine available before the virus arrives in a population. How many people do we need to vaccinate to ensure that there will be no epidemic? The answer is that, independent of the degree distribution, 60% of the population needs to be vaccinated when people are vaccinated at random. Thus, in that sense the herd immunity threshold does not depend on the degree distribution, and this number is usually what is reported in the media. But we can do better than random vaccination. If we would vaccinate mainly the high degree individuals we need to vaccinate fewer people. However, this is usually infeasible because the social structure is unknown. But one way to improve on random vaccination is to use social interactions. This works as follows. Vaccinate one person at random and ask this individual to name someone she interacted with during recent time. Vaccinate this person and ask him for a contact as well etc. This way, high degree individuals are more likely to be vaccinated and therefore less than 60% of the population has to be vaccinated to achieve herd immunity. This strategy is also called acquaintance vaccination (Cohen et al., 2003). In a world where there is no vaccine available, herd immunity can be achieved by people getting infected by the virus and subsequently recovering. And because the virus follows the same path as the above vaccination program, less than 60% needs to get the disease in order to achieve herd immunity.

This logic will be important when contemplating policies that mitigate the effect of the epidemic. All else equal, policies that maximize degree-weighted immunity are preferred. Not only as this achieves herd immunity more rapidly. But also because having a higher degree-weighted immunity, while holding average immunity constant, lowers the reproduction number and therefore limits a potential second peak.

Policies

I compare two simple policies in the SIR framework. In one policy experiment social behavior is limited such that all agents have the same degree irrespective of their initial degree. This corresponds to a severe lockdown in which people are only allowed to go outside for essential reasons. I refer to this as an "authoritarian" policy. The other policy is a policy in which the degree is limited proportional to the initial degree of each person. This corresponds to a policy that discourages people to meet others, but still lets the people decide themselves how much social activity to engage in. I refer to this as a "liberal" policy. For both policy experiments I assume that the epidemic first runs uncontrolled for 7 weeks, that the policy is then in place for 13 weeks, after which we return to normal. In the authoritarian policy each individual meets 6 people per week (compared to 10 and 25 without policy) while during the liberal policy each person's social activity is limited by around 65%. These numbers are chosen such that the effect on the number of infectious individuals during the policy is identical across both policy experiments. I now also assume that 0.5% of the infectious will eventually die.
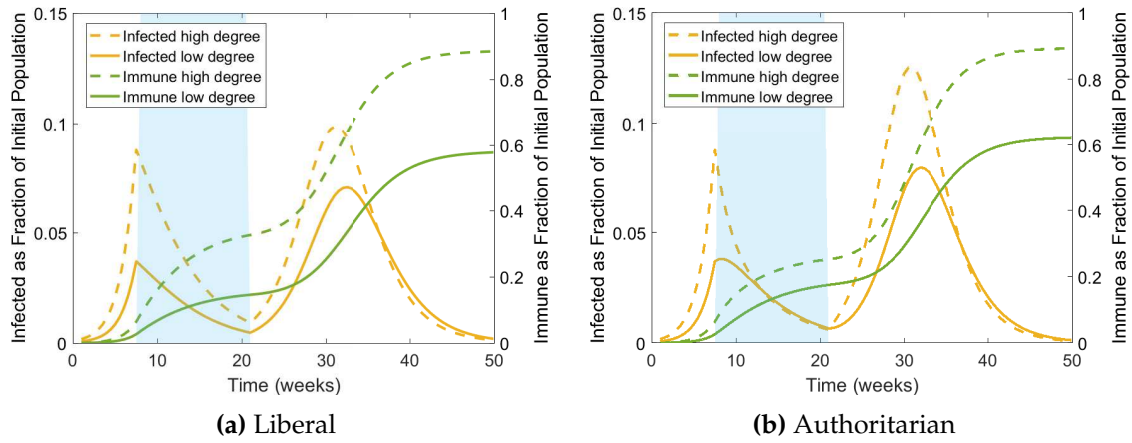
Figure 2: Evolution of the epidemic for the authoritarian and liberal policy. The shaded area indicates the time during which the policies were in place.

Figure 2 shows the evolution of the epidemic for the two different policies. When the policy is enacted around 5% of the population is infectious, and during the policy (highlighted by the shaded area) the number of infections drops quickly. The policy is lifted before herd immunity is reached making that a second peak will arise. However, the second peak will be lower when the liberal policy has been in place compared with the authoritarian policy. This also means that fewer people will die with the liberal policy.

The reason for the differential (long-run) effect of the policies is that during the liberal policy relatively more high degree individuals get immune compared to the authoritarian policy. This is shown in more detail in Figure 3. This figure shows the evolution of infection and immunity rates by degree type, for both the liberal and authoritarian policy. Before the policies are enacted, the high degree agents are more likely to get infected than the low degree agents and therefore, the high degree agents build up immunity quicker. During the liberal policy, the *relative* degree of the high degree individual vs the low degree individual has not changed. Therefore, the high degree agents continue to be more likely to be infected. At the end of the policy, around 32% of the high degree individuals are immune and 14% of the low degree individuals are immune. Compare this with the authoritarian policy. During the authoritarian policy the low degree individuals are as likely to get infected as the high degree individuals. Because the epidemic has spread uncontrolled during the first 7 weeks it is still the case that high degree agents have build up more immunity than the low degree agents. Namely, 25% and 17% respectively by the end of the policy, but the discrepancy is much smaller than under the liberal policy. Thus, the average immunity weighted by the degree is lower after the authoritarian policy than after the liberal policy, although the average immunity is the same for both policies. This means that after the authoritarian policy there are more susceptible high degree individuals left who fuel the second peak.
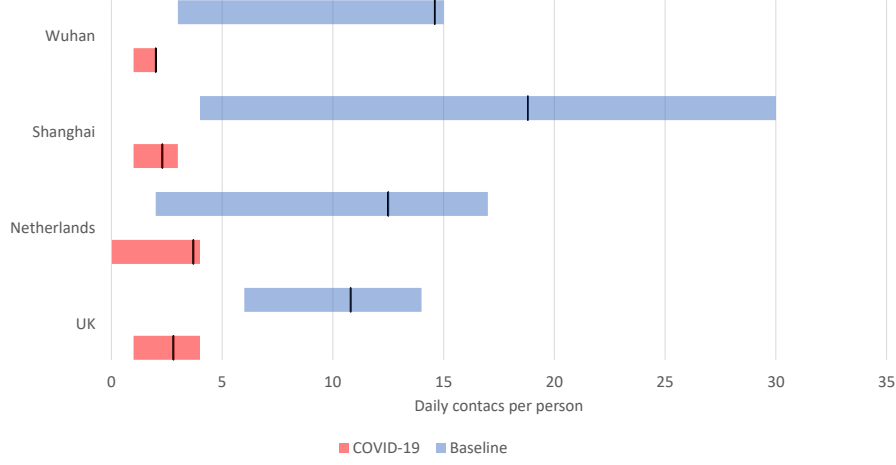
That the liberal policy leads to fewer deaths does not necessarily mean that welfare is higher under the liberal policy compared with the authoritarian policy. For instance, under the liberal policy the average degree goes down more than under the authoritarian policy.

Figure 3: Infection and immunity rates under the liberal and authoritarian policy. The shaded area indicates the time during which the policies were in place.

This might lead to a loss of utility. On the other hand, during the authoritarian policy the high degree individuals are more constrained which would lead to a relatively larger drop in utility when the marginal utility of social interactions is decreasing. Therefore, in the next section I embed the above model in a simple economic model in order to be able to speak about welfare. This also allows me to calculate optimal policy as for the two above policy experiments it is clearly not optimal to lift the restrictions after 13 weeks.

Before turning to the economic model let me briefly discuss to what extent the liberal and authoritarian regime relate to real-world policies. Figure 4 compares the daily number of contacts per person before the onset of COVID-19 and when social distancing measures were in place for four different locations. The data is obtained from Backer et al. (2020), Jarvis et al. (2020) and Zhang et al. (2020), and the COVID-19 period refers to a different time period for each location. For Wuhan and Shanghai this refers to early February 2020, for the Netherlands this refers to late March and April 2020, and for the UK this refers to late March 2020. For all locations this period coincides with social distancing measures being in place in these locations. Arguably, the measures taken in Wuhan and Shanghai were the most strict among these four locations. This is also confirmed by Figure 4 which shows that when these policies were in place the mean number of contacts dropped with 85–90% to a larger extent in Wuhan and Shanghai than in the Netherlands and the UK with 70–75%. In addition, Figure 4 shows that in the locations with a more strict lockdown the dispersion in the number of contacts fell to a larger extent. Namely, in Wuhan and Shanghai the interquartile range fell by 92% while in the Netherlands and the UK this fell by 73% and 63% respectively. Thus, the mild lockdown in the Netherlands and the UK is closer to the above liberal policy. In fact, in these two countries the 75th percentile fell to a smaller extent than the 25th percentile, while in Wuhan and Shanghai the 75th percentile fell to a larger extent. Thus, in the Netherlands and the UK the high degree agents were in *relative* terms more likely to contract the virus when mitigation policies were in place than before these policies were in place.

Figure 4: Daily contacts per person before onset of COVID-19 (baseline) and during social distancing measures. The colored bars show the interquartile range and the black lines show the mean. The COVID-19 period refers to early February 2020 (Wuhan and Shanghai), late March and April 2020 (the Netherlands), and late March 2020 (UK). Sources: Backer et al. (2020); Jarvis et al. (2020); Zhang et al. (2020).

II   SIR Economic Model

Individuals can consume a social good s and a regular good r. Consuming the social good leads to social interactions (e.g., going to a restaurant, the gym or taking the subway) while consuming the regular good does not involve others (e.g., cooking at home, doing exercises at home or driving a car).

The flow utility received from consumption equals

$$u(c_t^r, c_t^s) = (1 - \phi) \log \left(\frac{c_t^r}{1 - \phi} \right) + \phi \log \left(\frac{c_t^s}{\phi} \right) + \underline{u}. \tag{10}$$

The parameter ϕ governs the extent to which individuals value the social good. I allow ϕ to vary across individuals. I will refer to individuals with a high ϕ as social or high degree individuals. $\underline{u}$ is a constant of which the value will be set to match the value of life. Agents make decisions to maximize their lifetime utility V,

$$V = \mathbb{E} \sum_{t=0}^{\infty} \beta^t u(c_t^r, c_t^s), \tag{11}$$

where the expectation is taken over the probability of being alive at time t. The agent receives a flow utility of 0 when death. The discount factor is denoted by β. Agents cannot save such that their budget constraint is

$$c_t^r + P_t^s c_t^s \leq B_t, \tag{12}$$

where B_t is income at time t and P_t^s is the price of the social good. The regular good is the numeraire.

I assume that the number of social interactions an individual has grows linearly in consumption of the social good, and that even when consumption of the social good is zero there are still b social interactions. Th latter reflects, for instance, interactions within the household,

$$d(\phi) = b + a \cdot C_t^s \cdot c_t^s(\phi). \tag{13}$$

The degree is not only increasing in ones private consumption of the social good $c_t^s(\phi)$ but also in aggregate consumption of the social good C_t^s. This reflects, for instance, that the more people there are on the subway the more likely it is to get infected. a denotes the sensitivity of the degree to social consumption.

Individuals do not know whether they are susceptible, infectious or have recovered. This seems an appropriate assumption when there is limited testing available, when there are many asymptomatic cases and when the symptoms are similar to those of regular diseases such as the flu. However, I assume that agents are rational and have perfect information about the disease (such as the transmission risk p) and know the probability f_t that a given social interaction is with an infectious individual. This can, for instance, be learned by reading the newspaper. Although each individual does not know whether she has or has had the virus she can infer how likely it is that she has or has had the virus, based on her past social behavior. This works as follows. Suppose she knows that there is a 50% chance that she is susceptible at time t (thus $S_t(\phi) = 0.5$). Then based on f_t and conditional on her social interactions she knows the likelihood that she will get infected during the current period and she can calculate the likelihood that she is susceptible next period using (2). Likewise, she can update how I, R and D evolve. This implies that $S_t(\phi)$ and $I_t(\phi)$ are state variables.[7] The third state variable is time t and reflects how f_t, P_t^s, C_s^t and the mass of agents L_t that is alive at time t evolve over time. C_s^t is part of the state space as this affects the degree and L_t is part of the state space since there are decreasing returns to scale. Think of the latter as the capital-labor ratio increasing as people die.

The first-order condition is as follows

$$\frac{(1-\phi)P_t^s}{B_t - P_t^s c_t^s} - \frac{\phi}{c_t^s} = \tilde{\beta}_t p f_t S_t \frac{\partial d_t}{\partial c_t} \underbrace{\left(\frac{\partial V_{t+1}}{\partial I_{t+1}} - \frac{\partial V_{t+1}}{\partial S_{t+1}} \right)}_{<0}, \tag{14}$$

where $\tilde{\beta}_t$ denotes the discount factor times the probability of survival (which depends on I_t).[8] $\frac{\partial V_{t+1}}{\partial I_{t+1}} - \frac{\partial V_{t+1}}{\partial S_{t+1}}$ is negative as being infected leads to the possibility of dying. Thus, when the disease is present (i.e., $f_t > 0$), (susceptible) individuals will limit their consumption of the social good to lower the probability of getting infected and hence to lower the

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[7] $R_t(\phi)$ and $D_t(\phi)$ are not state variables as these are known conditional on $S_t(\phi)$ and $I_t(\phi)$, using that $S_t(\phi) + I_t(\phi) + R_t(\phi) + D_t(\phi) = 1$ and $R_t(\phi) = \frac{\lambda_R}{\lambda_R + \lambda_D}(R_t(\phi) + D_t(\phi))$.

[8] See Appendix A for the Bellman equation.

probability of dying. The extent to which an individual limits consumption of the social good increases in the transmission risk, p, in the probability a social interaction is with an infectious individual, f_t, in the likelihood of being susceptible, S_t, in the extent to which the degree depends on consumption of the social good, $\frac{\partial d_t}{\partial c_t}$, and in the 'cost' of the disease $\frac{\partial V_{t+1}}{\partial I_{t+1}} - \frac{\partial V_{t+1}}{\partial S_{t+1}}$.

There are several externalities in this model. Individuals that are likely to be infectious do not take into account that they can infect others by consuming the social good. In contrast, the agents that think they might have recovered from the disease do not internalize that their social activity crowds out meetings between susceptible and infectious agents. Individuals that are likely to be susceptible do not take into account that when getting infectious they will also transmit the disease to others, while on the other hand they do not internalize that increasing their social consumption lowers the probability that other susceptible agents meet an infectious agents. Furthermore, the degree does depend on aggregate consumption which is not internalized by the agent. The presence of these externalities makes that there is room for policies that can increase welfare. What the overall effect of these externalities is on socially optimal consumption is not clear ex ante.

To close the model, each individual supplies one unit of labor each period and production in the two sectors is decreasing returns to scale,

$$Y_j = A_j l_j^{\alpha_j}, \quad j \in \{r, s\}. \tag{15}$$

I have in mind a Cobb-Douglas production function, but that capital cannot be reallocated between the two sectors during the epidemic while labor l can reallocate.

I solve the model by backward induction and I assume that after 100 weeks the virus is gone. This could, for instance, be because a vaccine has arrived, a perfect cure is found or the virus has ran its course.[9] See Appendix A for details on the model and the solution method.
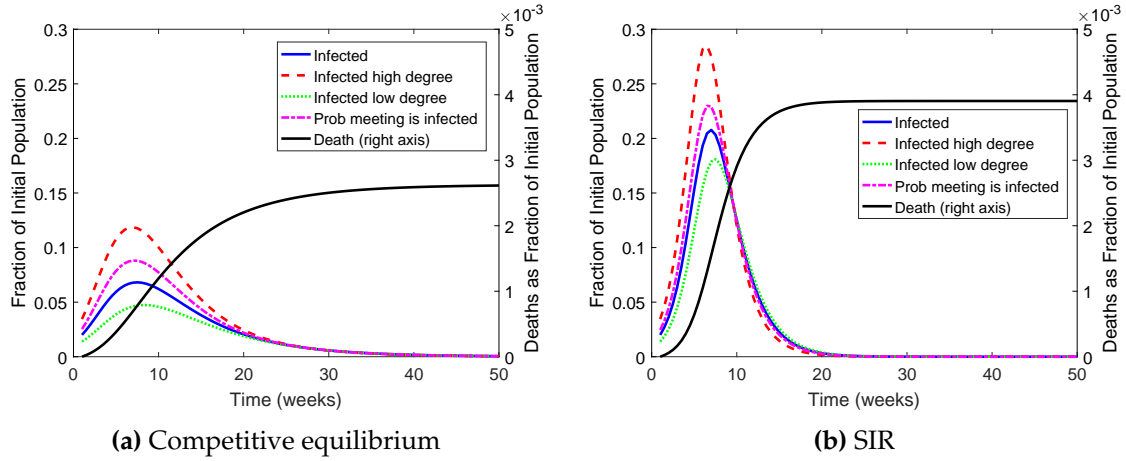
Calibration

I take each period as half a week and the disease parameters are set the same as in the SIR model of the previous section. Namely, the transmission risk p equals 0.07, the rate at which infectious individuals recover λ_R is 0.24875 and the rate at which they die λ_D is 0.00125. This entails that individuals are on average infectious for two weeks and that 0.5% of those that have contracted the disease will die.

70% of individuals is low social with a ϕ of 0.07 and 30% is highly social with a ϕ of 0.2. I set a and b to target a degree in the steady-state (before the arrival of the virus) of 5 and 12.5 per half-week period, respectively. b is equal to 0.95, meaning that an infectious agent will infect on average 0.27 individuals when it does not consume any of the social good and when all others in the population are susceptible.

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[9]I have also solved the model in continuous time for which I do not have to assume a terminal condition. This leads to similar results.

(a) Competitive equilibrium   **(b)** SIR

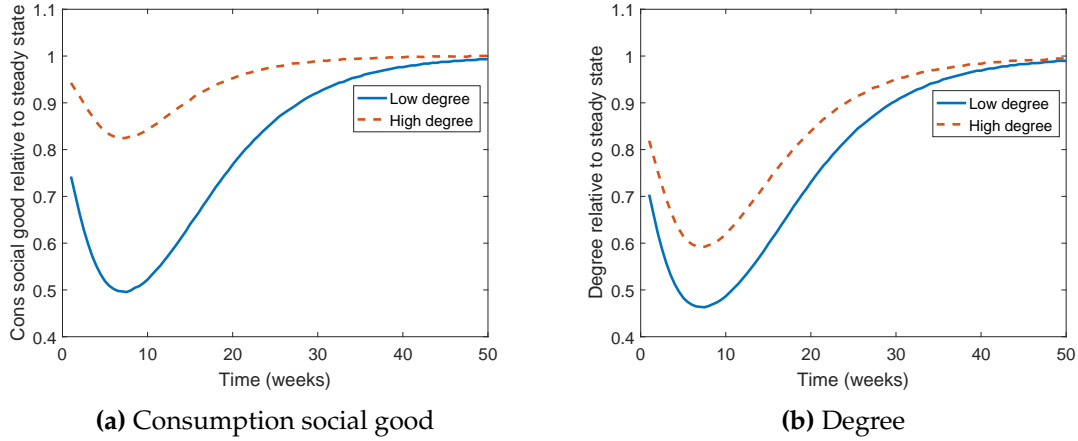**Figure 5:** Evolution of the epidemic.

I set the discount factor β to $0.96^{1/104}$ to target an annual discount rate of 0.04. I assume the returns to scale α are 0.7 in both sectors. I normalize TFP in the regular sector A_r to 1 and set A_s such that P^s is equal to 1 before the virus arrives. I set $\underline{u}$ to target a value of life of 1 million US dollar. This is relatively low. I have in mind that a large fraction of the people dying from COVID-19 is relatively old and is suffering from other diseases. Therefore, their expected remaining lifetime is relatively short, let us say 10 years. With a value of 100 thousand US dollar per quality adjusted life year, this gives a value of 1 million dollar.

Finally, I assume that at onset of the disease 2% of the population gets infected, proportional to their degree. This represents that the epidemic first spreads a few weeks uncontrolled before behavior and policy responds.

Competitive Equilibrium

Figure 5 shows how the disease evolves over time for the first 50 weeks both in the competitive equilibrium and in the SIR model. The blue solid line shows the fraction of the initial population that is infectious. The figures also show the share of agents that is infectious for the high degree and low degree agents. When a larger share of the high degree agents is infectious compared with the low degree agents, the probability that a meeting is with an infectious agent is higher than the average infectious share. This is indicated by the pink line. The black solid line shows the (cumulative) fraction of the population that dies. When behavior responds to the epidemic the peak is less severe but the epidemic lasts longer than when behavior is fixed. When behavior is endogenous 0.26% of the population dies while in the SIR model 0.39% dies.

The reason that the epidemic is less severe in the competitive equilibrium is that individuals start to consume less of the social good. The first panel of Figure 6 shows consumption of the social good relative to the steady state. Individuals respond to the virus and lower their consumption initially. However, the reproduction number is still

(a) Consumption social good   **(b)** Degree

Figure 6: Consumption of social good and degree in competitive equilibrium relative to the steady state (before the arrival of the virus).
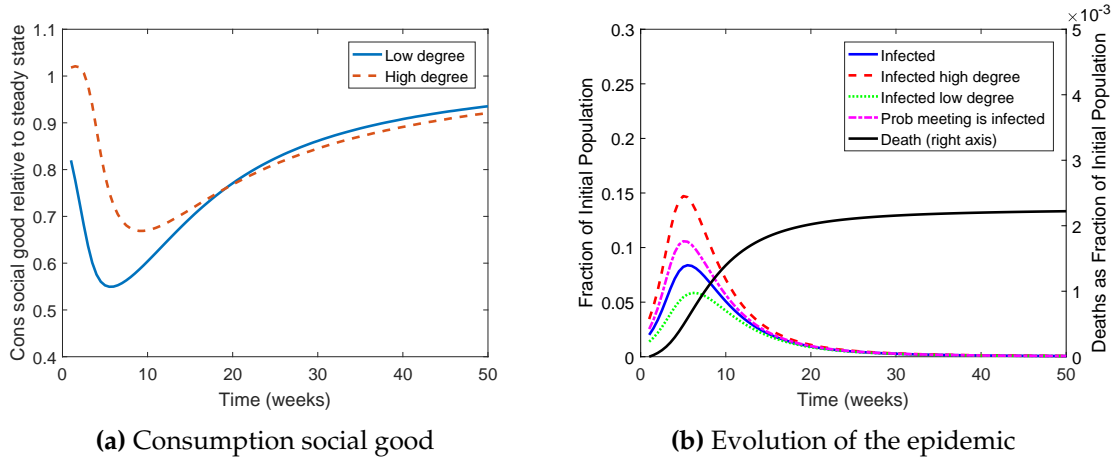
above 1 and the infectious share increases. As the probability that a meeting is infected rises, individuals lower their consumption of the social good further. After the epidemic has peaked consumption of the social good slowly goes back to its steady state level. In relative terms the high degree agents lower their social consumption to a smaller extent than the low degree agents. However, in absolute terms the change is comparable. The second panel of Figure 6 shows the degree. The degree falls quicker than social consumption because of the effect of aggregate consumption on the degree.[10] During the peak of the epidemic the degree falls by around 40% for the high degree agents and by 55% for the low degree agents. Thus, as the high degree agents lower their degree to a smaller extent than the the low degree agents, during the initial stage of the epidemic a larger share of the infected will be high degree agents compared to the SIR model.

Social Planner

I now consider a social planner that decides on how much of the social good is consumed by the high social and low social individuals such as to maximize welfare at time 0.[11] The first panel of Figure 7 shows the chosen consumption profiles and the second panel shows the evolution of the epidemic. When the disease arrives the planner decides to cut social consumption of the low social agents but does not adjust consumption of the high social agents. Then, during the first weeks it quickly lowers social consumption for both agents, after which it slowly increases consumption. Early on in the epidemic, the social planner chooses higher social consumption levels than in the competitive equilibrium. This leads to a relatively severe peak of infections. However, after some weeks, the planner lowers consumption of the social good more for the high degree agent than in the competitive

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[10]The 'gap' between the relative effect of the low degree agents and high degree agents is smaller for the degree than for consumption of the social good because of the intercept in the degree function (13).

[11]Welfare is the average utility at time 0, $\sum_\phi P(\phi)V(\phi)$, conditional on the initial infection rate, which is 2% here.

(a) Consumption social good
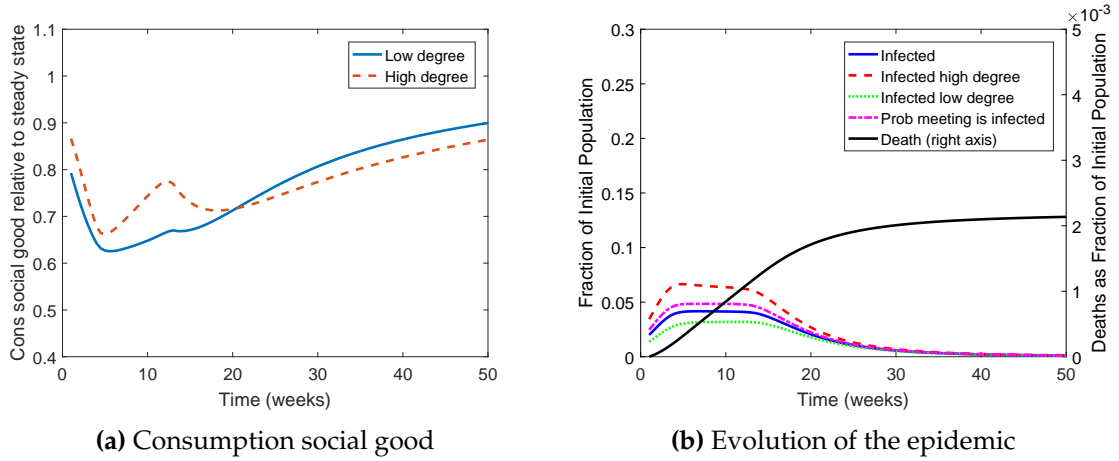
(b) Evolution of the epidemic

Figure 7: Social planner.

equilibrium. This lowers the reproduction number substantially such that the number of infectious individuals quickly drops. Thus, compared to the competitive equilibrium the social planner chooses an intense but short-lived peak. In the end this makes that 0.23% of the population dies compared with 0.26% in the competitive equilibrium.

That the social planner wants to subsidize social activity during the onset of the epidemic is because there is variation in the degree and because production faces decreasing returns to scale. As agents shift consumption from the social good to the regular good, aggregate output goes down due to decreasing returns to scale. In contrast, in a model with constant returns to scale the social planner decides to consume less of the social good than what is the case in the competitive equilibrium. The reason is that the returns to scale affect the trade-off between economic loss and the number of deaths. With constant returns to scale there is no economic cost from lowering social consumption and the planner limits social consumption so much that the reproduction number falls below one immediately such that the virus does not lead to an epidemic.[12] It is reasonable to assume that there is an economic cost to switching consumption at a large scale. Therefore, I stick with a decreasing returns to scale production function. Second, if all individuals would have the same degree I would not have obtained the result that the social planner wants to subsidize social consumption initially. The reason is that the planner prefers to have a high degree-weighted immunity. Thus, initially it chooses a high social consumption for the social individuals such that relatively many of them become infectious. Then, once this is the case it lowers their consumption substantially such that they do not infect too many others. That the planner does not want to limit social consumption much early on is similar to Alvarez et al. (2020) who find that it is optimal to impose a lockdown only after two weeks.

One reason that the planner chooses a severe but short-lived peak is that there is no maximum ICU capacity. Figure 7 shows that at the peak around 8% of the population is

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[12]With constant returns to scale there is of course still a cost in terms of utility when consumption of the social good falls below its steady state level.

(a) Consumption social good   **(b)** Evolution of the epidemic

Figure 8: Social planner with an ICU constraint at $I = 0.04$.

infectious. It is likely that at that point ICU capacity is reached and that once ICU capacity is reached the case fatality rate increases. Figure 8 shows the results when there is an ICU constraint such that once I exceeds 4% the death rate increases (in a quadratic way). The social planner lowers social consumption more severely with an ICU constraint. Especially consumption of the social agent is more affected compared with the planner without an ICU constraint. The ICU constraint makes that the epidemic stays at a plateau for a long period of time where I is around 4%.[13]

During the early phase of the epidemic, the planner does not want social consumption of the high social agent to drop by a larger extent than consumption of the low social agent. This is very different from what is achieved by a lockdown. A lockdown especially affects consumption of the high social agent. It is important to note that these results depend on parameter values. There are broadly speaking two types of strategies. One is to let the virus spread in a controlled way and to reach high levels of immunity eventually. This policy is optimal when the value of life is not too high, the case fatality rate is not too high and when a vaccine will not be available in the near future. This is the baseline calibration in this paper. When on the other hand there is a higher value of life or a higher case fatality rate the planner wants to suppress the virus as quickly as possible. In that case the planner wants to limit consumption of the social good more for the social agent than for the non-social agent (see Figure 11 in Appendix B). This trade-off also depends on the economic costs which are low here as labor can move freely between sectors.

Constrained Planner

It might be challenging to implement the above social planner solution in practice. Therefore, I consider two versions of a constrained planner. In each version the planner has access to one of two policy tools. The first is a planner that can decide on a time-varying

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[13]It is difficult to see from the graph, but I slightly exceeds the capacity constraint early on.

maximum, κ_t, on social consumption

$$c_t^s(\phi) \le \kappa_t\,. \tag{16}$$

This affects the agent with a high ϕ more severely. An example of such a policy would be a lockdown in which people are only allowed to go out for essential reasons.

I contrast this with a planner that puts a "tax" $\tau \ge 0$ on the social good such that the budget constraint becomes[14]

$$c_t^r(\phi) + (1 + \tau_t)P_t^s c_t^s(\phi) \le B_t\,. \tag{17}$$

An example of such a policy would be to put a constraint on how many people can be at the same time in a restaurant. All else equal, this would raise the price of the social good.

Both τ_t and κ_t are allowed to be time-varying but I assume that this time variation is limited. Initially, the planner can change the policy every two weeks, then every three weeks, and after 21 weeks the intervals become longer.[15] I do this for computational reasons but also out of realism. If a government would change its policy continuously this would be confusing to the people. Indeed, there are several governments that in the spring of 2020 decided on a step by step exit strategy from the lockdown, with a few weeks spacing between each step. The policy intervals are set to be longer during the final phase of the epidemic since when the epidemic is slowly fading away there is no need to adjust policy as frequently as during the early stage of the epidemic.

Figure 9 shows the results when a maximum on social consumption is imposed. The top panel shows how the epidemic evolves over time and the bottom panel shows how consumption of the social good evolves over time for both agents. The black line is the policy constraint, κ, and the dashed lines show the steady state consumption level. The first eight weeks of the epidemic the planner decides to do nothing. Only after the peak has been reached does the planner decide to impose a constraint on social consumption. The reason is that the constraint would only affect the social agents, while the social planner results imply that the planner does not want to constrain consumption of the social agent too much initially. 0.25% of the population will die eventually of the virus with this policy.

Figure 10 shows the results when there is a tax on social consumption. Now the black line shows the tax, τ, on social consumption. Initially, the tax starts at a low level and then gradually increases. But also this planner decides to impose a stricter policy only after the epidemic has peaked. The tax is with 40% at its maximum after twenty weeks, after which it slowly declines. During the second year there is still a tax of 7% in place (not shown). 0.22% of the population eventually dies because of the virus. This is less than in the case

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[14]I assume that the tax is lump-sum rebated to the household, which is hidden in the budget B_t.

[15]To be precise, there are first six intervals of two weeks, then three intervals of three weeks, then two intervals of five week, one interval of eight weeks, one interval of ten weeks, and during the second year the policy is assumed to be constant over time.
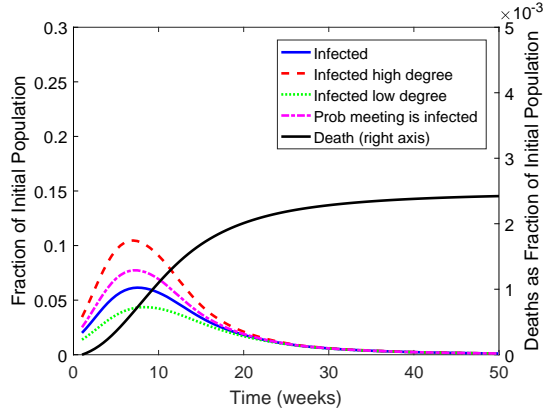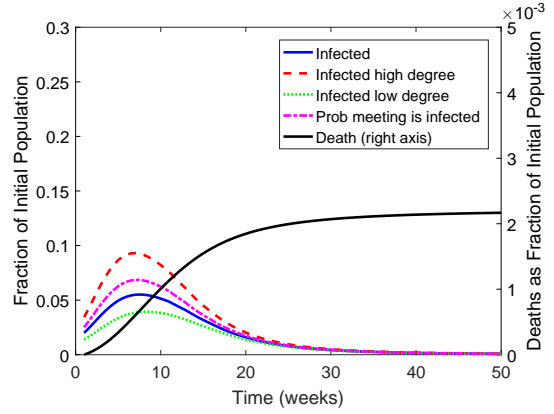
Figure 9: Maximum on social consumption

Figure 10: Tax on social consumption

of a maximum on consumption as the tax also lowers consumption of the low social agent.

Thus, both planners choose to let the virus run almost uncontrolled during the first weeks. The reason is that this builds up immunity quickly. If the planner would have chosen a high tax early on it would need to continue having a high tax for a long period of time.[16] Also Eichenbaum et al. (2020) find that it is optimal to first gradually increase and then to gradually decrease the containment policy.

Welfare is higher with the tax, but welfare differences are generally small. The agent is willing to give up 0.002% of lifetime income in order to move from the policy that imposes a maximum on consumption to the policy that imposes a tax.

III Conclusions

I study the effects of policy during an epidemic when there is heterogeneity in the degree across agents. The main results are that when designing policies one should take into account how the policy affects degree-weighted immunity versus average immunity, and, hence, that it might not be optimal to lower the degree of the high degree agent to the same extent as the degree of the low degree agent.

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[16] One reason why governments chose strict containment policies relatively early on was to buy time to increase health care capacity which is not in the model here.

It is important to stress that the quantitative results presented here are mainly an illustration as the results depend on parameter values that come with great uncertainty. Also, the economic model is limited as, for instance, there is no transmission of the virus in the workplace and no heterogeneity in the case fatality rate (e.g., due to heterogeneity in age). Nonetheless, the model shows that even when the high degree agents are not less likely to die than the low degree agents it is still optimal to not constrain the high degree agents too much.

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Appendix A Model Details

The Bellman equation of the household problem is as follows (where for notational simplicity I have omitted that state and choice variables depend on ϕ)

$$V_\phi(S_t, I_t, t) = \max_{\{c_t^s, c_t^r\}} u_\phi(c_t^s, c_t^r) + \beta(1 - \lambda_D \tilde{I}_t) V_\phi(S_{t+1}, I_{t+1}, t+1) \tag{18}$$

$$\text{s.t. } c_t^r + (1 + \tau_t) P_t^s c_t^s = B_t$$

$$S_{t+1} = (1 - p \cdot f_t \cdot d_t(c_t^s)) S_t$$

$$I_{t+1} = p \cdot f_t \cdot d(c_t^s) \cdot S_t - (\lambda_R + \lambda_D) I_t.$$

The aggregate state t comprises the price of the social good P_t^s, income per capita B_t which depends on the population alive (because production is decreasing returns to scale), and f_t which depends on how socially active the infectious are. Also the degree function $d_t(c_t^s)$ depends on the aggregate state as it depends on the level of aggregate consumption of the social good C_t^s. $\tilde{I}_t$ is the probability that a (living) agent is infected, $\tilde{I}_t = \frac{I_t}{L_t(\phi)}$, where $L_t(\phi)$ is the probability of being alive. Thus, conditional on being alive at time t, $(1 - \lambda_D \tilde{I}_t)$ is the probability that an agent is alive next period. If the agent dies it receives the value zero. τ_t is the tax on the social good that can be set by the liberal planner. In the competitive equilibrium $\tau_t = 0$.

I normalize the productivity A_r of the regular good to 1 and choose the regular good as the numeraire. I set α_j equal across sectors. This leads to the following optimization problem of the firm

$$\max_{l_r} l_r^\alpha - w l_r, \tag{19}$$

$$\max_{l_s} P_s A_s l_s^\alpha - w l_s. \tag{20}$$

A competitive firm chooses inputs such that the valued marginal product is equal to the wage, which yields after rewriting that

$$l_r = (P_s A_s)^{1/(\alpha-1)} l_s. \tag{21}$$

Combining this with labor market clearing, $l_s + l_r = L$, where L is the population that is alive, gives

$$l_s = \frac{1}{(P_s A_s)^{1/(\alpha-1)} + 1} L, \tag{22}$$

$$l_r = \frac{(P_s A_s)^{1/(\alpha-1)}}{(P_s A_s)^{1/(\alpha-1)} + 1} L. \tag{23}$$

I set A_s such that P_s is equal to 1 in the steady state.

Income per capita equals the sum of the wage, profits of the firm and the tax that is

rebated lump-sum:

$$B = \frac{l_r^\alpha + (1 + \tau)P_s A_s l_s^\alpha}{L} \tag{24}$$

$$= \frac{(P_s A_s)^{\alpha/(\alpha-1)} + (1 + \tau)P_s A_s}{\left((P_s A_s)^{1/(\alpha-1)} + 1\right)^\alpha} L^{\alpha-1} \tag{25}$$

To solve this optimization problem, I consider a horizon of 100 weeks and assume that at the final period the disease is gone (either because there is a perfect cure, a vaccine or the epidemic has run its course). This means that during the final period the agent is no longer worried about meeting infectious individuals and therefore consumes its steady state levels (i.e., $c_T^r = (1 - \phi)B_T$ and $c_T^s = \phi B_T/P_T^s$) with its associated steady state value. Then, conditional on the time path for P_t^s, B_t, f_t and C_t^s I use (18) to iterate backwards and calculate the consumption policy function. I do this for each ϕ separately. Given the consumption policy functions of the different ϕ individuals and an initial seed of infectious agents I simulate the economy and the epidemic forward. I check whether markets clear and whether the evolution of the epidemic is consistent with f_t and B_t. If not, I update the guess of the aggregate state and iterate until convergence.
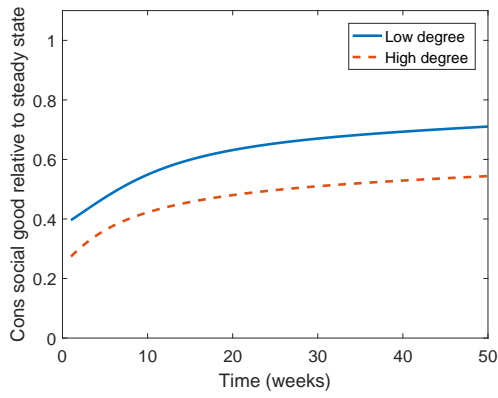
For the constrained planners I solve the above optimization problem conditional on a time path for τ and κ, respectively. I search over these time paths until I have found a maximum for the weighted value function at time zero evaluated at the initial seed of infectious agents.

The (unconstrained) social planner solves the optimization problem taking both agents into account at the same time. Therefore, there are five state variables: S and I for each type and the time state t. As this gets computational challenging I take a shortcut. I specify a time path of consumption of the social good for both types and calculate the resulting evolution of the epidemic and the associated discounted sum of utility. I search over this time path of consumption in order to find a maximum. I have used different initial values for this vector and they lead to the same results.
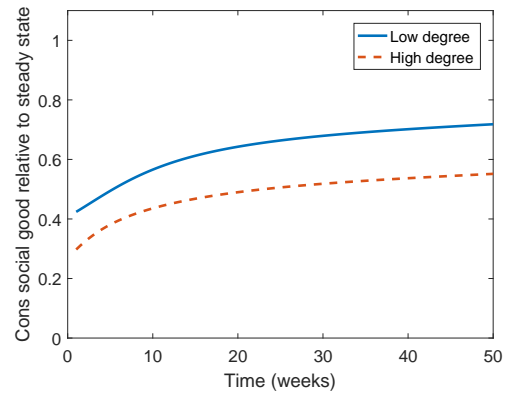
Value of Life

As explained in the main body of the text I target a value of life of 1 million US dollar, which corresponds roughly to 16 times annual GDP per capita in the US. In the steady state of the model, the flow utility is $\log(B) + \underline{u}$ such that the steady state value is $\frac{\log(B)+\underline{u}}{1-\beta}$. The model value of life in prices equals $\frac{\log(B)+\underline{u}}{(1-\beta)MU}$, where MU is the marginal utility of income increasing by 1 which with log utility equals $1/B$. Thus, I set $\underline{u}$ such that $B\frac{\log(B)+\underline{u}}{(1-\beta)}$ equals 16 times annual income.

Appendix B Additional Figures

(a) Value of life 3 million US dollars

(b) 2% of infected dies.

Figure 11: Consumption social planner - robustness.