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Epidemics and Policy: The Dismal Trade-off

Francesco Flaviano Russo

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University of Naples Federico II

University of Salerno

Bocconi University, Milan

CSEF - Centre for Studies in Economics and Finance
DEPARTMENT OF ECONOMICS – UNIVERSITY OF NAPLES
80126 NAPLES - ITALY
Tel. and fax +39 081 675372 – e-mail: csef@unina.it

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Abstract

I propose a stochastic SIR-Macro model to study the effects of alternative policies to cope with an epidemic. Lockdowns that order firms to close and that discontinues social activities slow down the epidemic progression at the cost of reducing GDP and increasing debt and, on average, decrease mortality. Testing strategies that identify and isolate a large number of infected but asymptomatics decrease mortality at a lower cost, but they are effective only if thorough. The more aggressive the pathogen, and the smaller the capacity of the health system, the bigger the gains from both policies. I also find that lockdowns work best in case of bigger average family size, diffused participation to the job market and bigger average workplace size.

JEL classification: E1, I1, H12.

Keywords: Lockdown, Testing, Pathogen, Pandemic

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* Università di Napoli Federico II and CSEF. Via Cinthia, Monte Sant'Angelo, 80126 Napoli. Tel: +39081675276. Email: francescoflaviano.russo@unina.it.

Table of contents

1 Introduction

The Covid-19[1] pandemic will most likely be the defining event of this century. An astonishing number of people died all over the World and those who recovered will probably face chronic health conditions and a worse quality of life for many years to come, while the policies implemented to cope with it resulted in major recessions and in a surge in public debt, with potentially severe long run effects, especially for low-income workers and highly-indebted countries. Recessions which are also fostered by feelings of uncertainty, fear and anxiety at best, will fade out slowly, further depressing investment and consumption. But the Covid-19 pandemic has not been the only large-scale infectious disease episode of the 21st century. Even abstracting from the influenza pandemic, that it a recurrent winter event in many countries, although not as deadly, and from the AIDS pandemic, that after three decades is still far from being over, there are at least four other major events with a global impact: the first SARS (2002-2004), originated in China and then exported mainly to other Asian Countries and to North America; the Swine Flu (2009-2010), that spread throughout the World from Mexico causing numerous deaths; the MERS (2012-2013), mostly concentrated in Middle Eastern countries; Ebola (2013-2016) that ravaged West Africa, with peaks in Guinea, Liberia and Sierra Leone. Sadly, it is also likely that the Covid-19 will not be the last epidemic, given the constant threats from zoonosis[2] or from antibiotic-resistant bacteria that are already killing thousand of people.

Faced with a very contagious pathogen that spreads rapidly, epidemiologists typically prescribe social distancing as a treatment[3], especially if infections could be asymptomatic, making it difficult to isolate the infected from the rest of the population. There are several advantages to this strategy: easing the pressure on the health system, which might not have enough capacity to properly treat all of the infected in case they are too many at the same time, with a consequent increase of the death rate; building additional capacity to treat patients, for instance new hospitals as in Wuhan, Milano and Madrid in the midst of the Covid-19 outbreak; buying time to develop a vaccine or a more effective pharmacological treatment,

[1] In this paper I will use Covid-19 to denote both the pathogen and the disease that it induces, although the pathogen correct name is SARS-Cov-2.

[2] A pathogen causing an infectious disease in animals that mutates to attack humans

[3] A so-called Non Pharmacological Intervention (NPI) to contain the infections without drugs.

which is especially valuable in case of new pathogens; waiting for the pathogen to mutate and to become less aggressive[4]. Social distancing can be reached in several ways: closing schools, universities, offices, plants, restaurants, bars, shops etc.; closing the national, regional of even city borders to avoid the geographical diffusion of the pathogen; forcing everyone to stay home as much as possible without meeting non-cohabiting family members and friends; in the limit, as in "Blindness" by Josè Saramago, confining away all of the infected and all of those who had contacts with them. These policies, that entail a variable mix of civil and economic liberties limitations, are commonly referred to as lockdowns.

The problem is that lockdowns are extremely costly. From a social and psychological perspective, segregating people from friends and extended family members will significantly impact on their well-being. Keeping children home without school will slow down their development with negative long run consequences and will impair the possibility of their parents, especially mothers, to work. From an economic perspective, closing economic activities will cause the GDP to plummet and will leave many workers without income and many entrepreneurs without cash-flow. Governments normally support the workers with transfers to avoid a precipitous decrease of their consumption and provide liquidity to the entrepreneurs in order to guarantee the continuity of their firms, but those policies require a huge amount of financial resources that, absent international aid or other forms of transfers, highly unlikely in case of an epidemic that hits simultaneously many countries, translate into additional public debt. Heavily indebted countries might find it hard to refinance their debt on the market, and their increased borrowing cost, with the consequent further increase in debt, will force them to implement restrictive fiscal policies in the future. Inflation is an alternative, assuming that an independent central bank, given the exceptional times, will be prone to accomodate the fiscal policy, but its cost could be even higher. Either way, the growth of the economy will most likely slow down and unemployment will most likely be persistently high for many years after. Moreover, some firms will be forced to shut down even in case of massive financial aid by the government, and the older among the unemployed will no longer be able to find another job. Investments and R&D will shrink and the government provision of essential public services might be called into question. Lockdowns are thus intergenerational redistributive policies:

[4]This is often the case for viruses whose goal, from a natural selection perspective, is not to kill most of their host organisms as this would impair their chances of reproducing and surviving.

assuming that they are effective at reducing the death toll of the epidemic, and therefore the cost for the current generation, they do so at a high cost that future generations will be called to pay. Lockdowns are also intragenerational redistributive policies, since not all firms can be closed, the most popular exceptions being the distribution and production chains of "essential" products such as food and drugs, and since not all individuals have enough savings to smooth consumption. Thus also the current generation bears a non-trivial cost.

However lockdowns are not the only policy alternative. Taiwan and Korea, in particular, showed that it is perfectly possible to stem the diffusion of a very aggressive pathogen such as the Covid-19 without forcefully closing economic activities or ordering people to stay home[5]. Their strategy consisted in a massive screening of the population to identify as many infected as possible, which is not an easy task in case of many asymptomatic infections. Once identified, the infected are quarantined, although normally not separated their families, and those who came into contact with them are identified, screened and, if positive, quarantined and so on. The large number of tests required for this Testing-Tracing-Quarantine (TTQ) policy (Alvarez, Argente and Lippi 2020) to work properly imply a substantial cost for the health system, which might also lack the required laboratory capacity to promptly process them. Moreover, since the contacts of the infected are often identified not only with the spontaneous declarations of the infected themselves, but also through rather invasive tracing technologies that use personal data collected from cell phones and credit cards, and that often make them public, albeit anonymously, there is a serious issue about privacy infringement which not many societies would tolerate. A further policy alternative can be to simply let the epidemic unfold without any intervention. The goal is to let as many people as possible develop specific antibodies against the pathogen in order to reach a sufficient level of protection against it (i.e. herd immunity). This is, for instance, what the Johnson government initially planned to do in the UK before the study by Ferguson et al. (2020) showed its potential death toll, eventually persuading them to change their minds.

The main goal of this work is to try to isolate and understand, from an economist angle, the main tradeoffs associated with the different policies to cope with a epidemic, including the possibility of doing nothing. Obviously the best policy will be conditional on the characteristics

[5]Many citizens spontaneously decided to reduce social activities and to wear individual protective devices regardless of any implemented policy in both countries as soon as the Covid-19 started to spread.

of the pathogen that is responsible for it. For instance, almost no government was ever tempted to close schools, shops and factories when the seasonal influenza is close to peak because its symptoms are typically mild and its death rate quite low, while almost all governments, with few exceptions, did so when faced with the Covid-19, significantly more contagious and aggressive.

I build a simple model that nests the workhorse stochastic Susceptible-Infected-Recovered (SIR) model of epidemics (Kermack and McKendrick 1954; Allen 2017) with an equilibrium macroeconomic model (a SIR-Macro model) in the spirit of Eichenbaum, Rebelo and Trabandt (2020) and Piguillem and Shi (2020). The model features a small economy whose members are all susceptible of infection by a new pathogen. All agents live in families. Some of them work and earn a fixed wage, while others are either pensioners or unemployed. All members of this economy engage in social activities that entail close contacts. The epidemic starts when some of those agents get the infection, say after a trip abroad. The infected agents spread the pathogen in their families, workplaces and through their social activities. I assume that the infected can be either asymptomatic or symptomatic and that the asymptomatics develop symptoms with a fixed probability. Those who develop symptoms do not go to work and participate to a very limited number of social activities, but they continue to meet all of their family members. Those who do not develop symptoms engage in their normal routine unless they are screened and identified, in which case they are quarantined. Both asymptomatics and symptomatics have a fixed probability to recover, which entails being protected for life against the pathogen. Death is a possible consequence of the infection, but only in case of symptoms. The health system is subject to a capacity constraint and the mortality is higher if the constraint is binding when too many individuals are infected at the same time, impairing the possibility of properly treating all. Firms are forced to temporarily close if the number of non-infected workers drops below a threshold. Closed firm, either because of an insufficient number of available workers or as an effect of the lockdown, reopen next period with a fixed probability. All agents are expected utility maximizers and can borrow at a fixed interest rate. The market for the single good produced in this economy clears in all periods. The government taxes wages and profits and transfers resources to all infected workers that do not go to work, as well as to all pensioners and to all of the unemployed, and balances its

budget before the start of the epidemic. The model highlights that the joint evolution path of the epidemiological and of the macroeconomic variables depends on the identity of the agents that get the infection at each point in time. For instance, if workers get the infection before the pensioners, there will be a larger output decrease at the beginning of the epidemic.

I use this framework to simulate the effect of alternative policies to cope with the epidemic, mostly lockdowns and testing strategies. Those policies have the potential to save lives, but they do so at the cost of decreasing aggregate output and increasing public debt. This is what I call the *dismal trade-off*. I propose a way to study this trade-off and to compare alternative policies, that entails computing the two *Dismal Ratios*[6] of each policy. Taking as benchmark the no-policy scenario with a government that lets the epidemic unfold without any intervention, the dismal ratios are the percentage of the population spared, if any, for each percentage point of output lost (Output Dismal Ratio or ODR) and for each percentage point of additional public debt (Debt Dismal Ratio or DDR). The first measure accounts for the burden put on the current generation, while the second for the burden on future generations.

I find that if the pathogen triggers asymptomatic infections and if testing strategies are feasible, in the sense that there is enough laboratory capacity to test a large number of individuals and if privacy-invading tracing technologies are allowed, they must be preferred. Lockdowns are second best alternatives, and have the potential of saving many lives, although at a high cost in terms of lost output and increased debt. Both lockdowns and testing strategies do not always result in less deaths with respect to the no-policy alternatives. On average, they do reduce the mortality rate, but, since they are mitigation policies that mostly reduce the pace at which the pathogen spreads, they might also result, in the end, in more deaths with respect to the no-policy alternative as the result of random variation, for instance in case a similar number of individuals get the infection over the course of the epidemic. I also found that the gains from lockdowns and testing strategies are higher in case of aggressive pathogens with a high mortality rate and in case of limited health system capacity, since they reduce the number of simultaneously infected individuals. The smaller the capacity of the health system, the more severe the lockdowns and the more extensive the testing strategies must be in order to smooth the contagion curve sufficiently to prevent the capacity constraint from becoming

[6]The terminology comes from analogy with the sacrifice ratio in monetary economics: the sacrifice in terms of output loss to bring inflation down.

binding.

The effects of a lockdown are crucially dependent on the family and industrial structure. I find that lockdowns are more effective: in case of smaller families, since family contacts are not an important source of contagion if many agents live alone or in families of two; in case of bigger firms, since they shutdown contagions from coworkers; in denser societies whose members engage in many social activities, since they account for most of the contagions; in case of more diffused participation to the labor market, because there are more people at risk of contracting the pathogen on the workplace. In terms of policy design, the simulation results show that testing policies, in order to be effective, must be able to isolate a big number of asymptomatics. Lockdowns, on the other hand, must be prolonged until the pathogen effective reproduction number (the number of new infections caused by each infected individual) drops below a very small threshold, although this means prolonging the epidemic. Moreover, severe lockdowns that close a large number of economic activities are not appropriate if the average firm size is small. Social-only lockdowns are less costly from a macroeconomic standpoint, but they might be insufficient in case of big average family and workplace size. In a two-regions extension of the baseline model, I also show that closing borders is essential regardless of the policy approach.

The rest of the paper is organized as follows. I discuss the very recent literature that merges epidemiological and economic models in section 2. Section 3 contains the model description, while in section 4 I fully explain the calibration and simulation details. Section 5 summarizes the main simulation results and compares different policies. Section 6 compares instead different lockdown and testing strategies that differ in their implementation details. In section 7, I discuss several model extensions (second waves of infection, schools, two countries and heterogenous social contacts). Section 8 concludes.

2 Related Literature

The literature on the economic effects of the epidemics started almost as soon as many governments implemented lockdowns to contrast the Covid-19 pandemic at the beginning of 2020. Most works incorporate an explicit epidemiological structure into a macro model (SIR-Macro

models) to study the effect of an epidemic and of the policies to cope with it. Examples include, among others, Atkeson (2020), Eichenbaum, Rebelo and Trabandt (2020), Piguillem and Shi (2020), Collard et al. (2020), Alvarez, Argente and Lippi (2020), Berger, Herkenhoff, and Mongey (2020) and Glover, Heathcote, Krueger and Rios-Rull (2020). I will not attempt to fully discuss this extensive literature, which would require a separate paper, but rather to highlight the differences between my work and the most related contributions.

The main difference with previous works is that my model is stochastic, in the sense that the evolution path of the epidemic is not fully determined given the initial stocks of infected, susceptible and immunes conditional on the basic reproduction number and on the implemented policy. Depending on the identity of the infected at each stage of the epidemic progression, several possible evolution paths are possible so that different policies can yield different results just as a consequence of random variation. For instance, lockdowns and testing strategies do not always result in a reduction of the death rate, especially in case the health system capacity constraint is not binding. Moreover, my framework is extremely flexible and can account quite easily for all sorts of agents, firms and family heterogeneity, making it suitable for many policy-relevant simulations.

My main result is similar to Piguillem and Shi (2020) and Berger, Herkenhoff, and Mongey (2020), who also show that testing policies are effective at smoothing the peak of the infection and at reducing both the mortality and the economic impact of the epidemic. Alvarez, Argente and Lippi (2020) find instead that testing policies are complementary to lockdowns, in the sense that they can be used to reduce its cost but not as a subsitute. Similarly, Dewatripont et al. (2020) recall that identifying asymptomatics is essential to ease the severity of lockdown and to contain its negative effect on output since, upon recovery, they can be allowed to come back to work.

In Eichenbaum, Rebelo and Trabandt (2020) the agents respond to the epidemic by decreasing their consumption because purchases expose them to the risk of infection. This additional source of contagions, which is absent from my analysis, determines a demand-side effect that amplifies the recessionary effect of the epidemic. A lockdown in their model is welfare improving because the agents do not internalize the increased risk of contagion to which other agents are exposed as an effect of their consumption and labor supply decision,

even if it induces a sharp recession. Krueger, Uhlig and Xie (2020) extend their model to heterogeneous goods and show that the consumption riallocation away from goods consumed in a social context, such as restaurant meals, can significantly slow down the progression of the epidemic, even absent any policy intervention. In both models, however, there is no scope for testing policies since all infected are symptomatics. Bethune and Korinek (2020) and Jones, Philippon and Venkateswaran (2020) also highlight the importance of contagion externalities for infected individuals that engage in social activities and show that lockdowns are a way to internalize them.

In Piguillem and Shi (2020), Farboodi, Jarosch and Shimer (2020), Collard et al. (2020), Garibaldi, Moen and Pissarides (2020), Bethune and Korinek (2020), Chang and Velasco (2020), Jones, Philippon and Venkateswaran (2020) and Toxvaerd (2020), agents also realistically react by taking individual precautionary measures that limit the spread of the pathogen, such as voluntarily decreasing the number of social interactions and their labor supply or wearing protective devices such as face masks for respiratory viruses. Th dynamic evolution of the pandemic is therefore endogenous and the policy maker interventions milder. In my model, I abstract from endogenous reactions of the agents which, at the cost of being less realistic, allows me to focus attention on the effects of the implemented policies per se. With respect to the endogenous response of the labor supply, this is equivalent to assuming that the agents do not have enough savings or borrowing ability to afford a labor supply reduction absent a lockdown with the associated monetary transfers.

Favero, Ichino and Rustichini (2020) build a much more detailed and realistic model than mine in terms of age and industrial structure and calibrate it to Italy for the Covid-19 case. Their task is to design the optimal, post-lockdown exit strategy to contain the output loss while keeping mortality low. They show that it is possible to achieve both goals letting all young workers in low-risk sectors and some of the young workers in high risk sectors return to work, while prolonging the quarantine for older ones. Their work shows the potential of fine-tuned public policies contingent on specific individual characteristics. With respect to their work, I offer a wider perspective, studying in particular alternatives to lockdowns such as testing policies that can achieve the same results. Glover et al. (2020) also consider a very detailed SIR-Macro model with heterogeneous health risk and multiple sectors to highlight

the redistributive effects of lockdowns and use it to evaluate US policies.

Collard et al. (2020) focus on the trade-off between current utility and future mortality that mitigation strategies such as a lockdowns involve. They show that the optimal policy entails a severe confinement followed by a gradual de-confinement until herd immunity is reached, the so-called "The Hammer and the Dance" (Pueyo 2020). Similarly to their work and to Favero, Ichino and Rustichini (2020), but differently from most other previous works (Piguillem and Shi 2020, Eichenbaum, Rebelo and Trabandt 2020, Alvarez, Argente and Lippi 2020, Glover et al. 2020, Hall Jones and Klenow 2020), I also focus on the trade-off between mortality and ouput loss, extending the discussion to public debt, rather than on finding the optimal policy for a given monetized value of life.

Chang and Velasco (2020) stress the complementarity between public health measureas and economic policy. For instance, expansionary policies implemented after the peak of infections, and announced at the beginning of the epidemic, can persuade agents to stay home without going to work when the contagions are close to peak. These effects of policy announcements are absent from my analysis.

Overall, I also try to offer a wider perspective on the effects of the policies designed to cope with an epidemic without focusing exclusively on the Covid-19 case, since, sadly, new epidemics might emerge in the near future, challenging again many governments.

There are also few works that studied the economic consequences of epidemics, and of diseases in general, before the Covid-19 2020 outbreak. Examples include, among others: Young (2005), who studies the long-run impact of the HIV epidemic in South Africa; Goenka, Liu and Nguyen (2014) that study the long-run consequences of epidemics on economic growth; Greenwood et al. (2019) that study the potential impact of several policy intervention to stem the HIV diffusion in Malawi.

3 The Model

I consider a closed economy where a total of N agents live and work before an epidemic strikes[7]. An agent i is represented by the vector $x_{ij,t}$, where t is the time period and where j indexes the firm where he works, with $j = 0$ in case he does not. The agents who do not work are either pensioners, unemployed or underage. In this baseline specification, I assume that underage agents do not attend schools or universities, but I will relax this assumption in section 7. n agent can be either: infected by the pathogen with symptoms that can be clearly ascribed to it; infected by the pathogen but asymptomatic; susceptible of infection; immune, after recovery from the infection; dead. For simplicity, I assume that all agents are susceptible in $t = 0$, which is the case for news pathogens against which no individual is naturally protected by its own antibodies and for which there is no vaccine available. The vector x is composed of five binary elements corresponding to the status of the individual with only one of the five equal to 1 while the others are zeros. Specifically $x_{ij,t} = \{f_{ij,t}, a_{ij,t}, s_{ij,t}, u_{ij,t}, d_{ij,t}\}$ where $f_{ij,t} = 1$ and $x_{ij,t} = [1, 0, 0, 0, 0]$, for an infected with symptoms, $a_{ij,t} = 1$ and $x_{ij,t} = [0, 1, 0, 0, 0]$ for an infected without symptoms, $s_{ij,t} = 1$ and $x_{ij,t} = [0, 0, 1, 0, 0]$ for a susceptible, $u_{ij,t} = 1$ and $x_{ij,t} = [0, 0, 0, 1, 0]$ for an immune and $d_{ij,t} = 1$ and $x_{ij,t} = [0, 0, 0, 0, 1]$ for a dead. The stocks of infected with symptoms is $F_t = \sum_{i=1}^{N} f_{ij,t}$, while the stock of infected without symptoms is $A_t = \sum_{i=1}^{N} a_{ij,t}$. The stocks of susceptibles, immunes and deads are instead, respectively, $S_t = \sum_{i=1}^{N} s_{ij,t}$, $U_t = \sum_{i=1}^{N} u_{ij,t}$ and $D_t = \sum_{i=1}^{N} d_{ij,t}$.

3.1 Families and Firms

The agents belong to I families, represented by the following collection of sets $\{n_1, n_2 \dots n_I\}$. The number of members of each family $h \in \{1, 2, \dots I\}$ is equal to the cardinality of each set n_h. Thus:

[7]For simplicity I will not model demographics independently from the epidemic. In other words, there will be no deaths in the population that are not due to the pathogen and there are no newborns that enter the economy. Thus, at time t, the total number of agents in the economy is simply equal to the initial population size N minus the total number of deaths. Since epidemics in the model will last at most for one year, this is not a strong assumption.

$$\sum_{h=1}^{I} |n_h| = N$$

Agents are associated to their respective families by the following function $N(i) = n_h$ if $i \in n_h$. The average family size will play an important role in the simulation: the bigger the families, the faster the spread of the pathogen and the less effective the lockdown effects.

There is a total of J firms in the economy, modeled by the collection of sets $\{m_1, m_2 \dots m_J\}$. Firms have $\hat{M}_j = |m_j|$ employees at full capacity, where $\hat{M}_j = 1$ stands for self employed agents. The total number of employed agents at full capacity is $\sum_{j=1}^{J} \hat{M}_j = L < N$. The firms employ the workers, which are all equally productive, to produce the unique undifferentiated product of this economy. I assume that the workers are assigned to the firms randomly and that it is possible for members of the same family to work together. This random assignment is an important source of variation for the final results. For instance, if agents from big families are assigned to big firms the pathogen will spread faster. Workers are paid the fixed wage w, which can thought of either the minimum wage prescribed by the law or the efficiency wage below which it is not optimal to pay the workers. The pensioners and the unemployed are paid a pension or subsidy equal to θw with $\theta \leq 1$. For simplicity, I assume that families with children receive the same amount θw for each underage, non-working member of the family. The product is sold at the market clearing price p_t. The firms are price takers and cannot decide how much to produce and how many workers to hire. They simply hire as much workers as they can, produce whatever they can and sell all the resulting output. I assume that symptomatic agents do not or cannot go to work. Asymptomatic agents, instead, can work only if they are not quarantined after a positive test result. I assume that it is not possible for the firms to replace sick workers with the unemployed, so the evolution of the epidemics impact on firms production. Similarly, firms cannot exchange workers. Moreover, I also assume that if the number of non-sick workers drops below a lower bound $\bar{M}_j$, the firm is forced to stop its operation. A firm that produces zero in a given period, either because of a lockdown policy that forces it to close or because there are not enough workers, reopens the next period with time-independent probability[8] λ. This assumption adds hysteresis to

[8]The parameter λ depends crucially on policy inetrvention. A government that transfers money to the closed firms or that extends a public guarantee for low interest rate financing increases the probability to reopen. I will not model explicitly the dependence of λ on public expenditure and debt

the model dynamic: not all firms have enough financial resources to pay the fixed costs while closed, so not all of them survive the epidemic. For simplicity, I assume that λ is not a function of how many periods the firm has been closed while, in reality, it is a decreasing function of it. The individual production function of firm j is therefore:

$$y_{j,t} = \mathbb{1}_{M_{j,t} > \bar{M}_j} \left[\Lambda + (1 - \Lambda) \mathbb{1}_{M_{j,t-1} \geq \bar{M}_j} \right] M_{j,t}^{\alpha} \tag{1}$$

where $\mathbb{1}$ is the indicator function and $\Lambda \sim Ber(\lambda)$ is a bernoulli random variable with parameter equal to the probability of reopening next period if closed λ. $M_{j,t}$ is the number of workers who can work at firm j in period t, either because they are susceptible, immunes or asymptomatic and not quarantined:

$$M_{j,t} = \hat{M}_{j,t} - \sum_{j \in m_j} f_{ij,t} - \sum_{j \in m_j} \kappa_{i,t} \, a_{ij,t} \tag{2}$$

where $k_{i,t} = 1$ if the asymptomatic worker i is quarantined in period t after a positive test result. Total production is simply the sum of all outputs from the J firms, while the potential output of the economy to the sum of all outputs at full capacity:

$$Y_t = \sum_{j=1}^{J} y_{j,t} \quad \leq \quad \hat{Y} = \sum_{j=1}^{J} \hat{M}_j^{\alpha} \tag{3}$$

I use he output gap $(Y_t - \hat{Y})/\hat{Y}$ as a measure of the recessionary effect of the epidemic and of the policies to contain it. Firms' profits are taxed at rate τ^y in case they are positive and there are no tax credits in case of negative profits. Profits are also not distributed as dividends: the firms simply retain them.

Agents choose consumption optimally given the expected future earnings. Infected agents who cannot work, either because symptomatics or asymptomatics and quarantined, earn the same income θw of the pensioners, unemployed and underage. I assume that the planning horizon is limited to the duration of the epidemic, that ends at time T, and that all agents, absent the epidemic, would have lived beyond time T. The optimization problem is therefore:

$$\max_{\{c_{ij,t+s}\}_{s=0}^{T-t}} E_t \sum_{s=0}^{T-t} \beta^s v(c_{ij,t+s}) \tag{4}$$

subject to the present vale budget constraint:

$$E_t \sum_{s=0}^{T-t} \frac{1}{(1+r)^s} p_{t+s}\, c_{ij,t+s} = w\,(1-\tau^w)\, E_t \sum_{s=0}^{T-t} \frac{1}{(1+r)^s} \left[\theta f_{ij,t+s} + (1-\kappa_{i,t}+\kappa_{i,t}\theta) a_{ij,t+s} + u_{ij,t+s}\right] \tag{5}$$

where τ^w is the tax rate on wages and transfers to pensioners, underage agents and infected who cannot work. I assume that there is perfect foresight and that agents form their expectations rationally.

3.2 The Government

The government balances its budget before the epidemic:

$$\tau^y \Pi_t + \tau^w w L_t + t\theta w(N - L_t) = \bar{G} + \theta w(N - L_t) \tag{6}$$

where L_t is the total number of workers in period t:

$$L_t = \sum_{j=1}^{J} M_{j,t}\, \mathbb{1}_{M_{j,t} > \bar{M}_{j,t}} \tag{7}$$

and Π_t is the sum of all firms' profits, if any:

$$\Pi_t = \sum_{j=1}^{J} \max\{(p_t y_{j,t} - w M_{j,t}) \mathbb{1}_{M_{j,t} > \bar{M}_{j,t}}\,, 0\} \tag{8}$$

$\bar{G}$, if positive, is the expenditure (say in the health sector) that the government finances with the difference between the tax proceedings and the transfers, while, if negative, is the extra income (say from natural resources) needed to finance the expenditures not covered by the proceedings. I assume, differently from Piguillem and Shi (2020) that the tests needed to identify the asymptomatics are costless, say because they are already part of what the health system normally does or because it is easy to divert resources normally allocated to the health system but used for other expenditures. I will discuss the consequences of costly tests and and deficit financing in section 5.3. These assumptions are such that the epidemic puts pressure on the government budget, because of a joint erosion of the tax base (less agents work, lower firms profits) and of an increase in transfers (less agents work). I assume that $\bar{G}$ does not change over time: it is not possible to build additional hospital capacity to treat the infected or to transfer additional resources to the firms to increase their probability to reopen. The consequence is that, when evaluating the impact of the epidemic on the government revenue, I

will have lower bounds for their actual impact, since many governments do in fact intervene to inject liquidity in the firms and to build additional health system capacity. The government does not pay any interest rate on the debt and it is free to borrow an unlimited amount of money on the market.

3.3 Pathogen Diffusion and Timing

Susceptible agents can get the pathogen when matched with an infected. I assume that symptomatic agents transmit the pathogen with probability π per match, while asymptomatic agents with probability $\bar{\pi} < \pi$. I assume, quite unrealistically, that the contagion probability per single match is independent from previous history: it does not matter if, before the current matching with an infected, the symptomatic already matched with other infected while, in reality, being exposed repeatedly to the same pathogen significantly increase the probability of getting the infection. Newly infected agents are asymptomatic and they cannot transmit the pathogen to others in the same period[9]. In the terminology by Allen (2017), all agents that get the pathogen are "exposed" for one period, meaning that they are infected but not infectious. Differently from Favero (2020) and Favero, Ichino and Rustichini (2020), I will not explicitly keep track of the exposed in the model, simply bunching them with the asymptomatics.

The timing is the following. At the beginning of the period all susceptibles match with family members and they have probability π to get the pathogen from each symptomatic family member and $\bar{\pi}$ from each asymptomatic family member. I assume that symptomatic agents are either home or, if hospitalized, that they are allowed to have visits from family members. The agents who work are then matched with all coworkers and they get the pathogen with probability $\bar{\pi}$ from each non-quarantined asymptomatic coworker. All agents than engage in social activities that result in matches with other members of the economy, say at the supermarket[10], in the metro, at school[11] or at the restaurant. I assume that all agents are matched,

[9] In the baseline simulation, the period will be equal to one week. The choice of a one-week latency or incubation period is in turn in line with the Covid-19 estimate of 5 days by Ferguson et al. (2020) and similar to Ebola, although bigger than Influenza (typically less than three days) and smaller than Measles (typically 10 days).

[10] In the model consumption activities are not separate contagion opportunities from social interactions. This is important because in a model with this separation there are demand effects that might exacerbate the recessionary effect of an epidemic as in Eichenbaum, Rebelo and Trabandt (2020): the agents simply decide to decrease consumption to limit their exposure to the pathogen.

[11] In this baseline simulation I do not consider schools separately from other social activities. In section 7.2

every period, with a fixed fraction of the agents who are not in his family or workplace, but that the identity of those agents (who exactly each individual meets) changes over time. I assume that a very small fraction of the symptomatics ψ participates to these social activities and that their identity changes randomly over time. The idea is that, even if symptomatics are easy to identify and to quarantine and even if most of them are sick and unable to move normally, there can still infect others, say the doctor that treats them or a random stranger on the way to hospital. Asymptomatics, instead, once identified and quarantined, are not allowed to go to work and to have social contacts until recovery and they cannot escape the quarantine. At the end of the period, the asymptomatics that were so at the beginning of the period can develop symptoms with probability ρ and both symptomatics and asymptomatics recover with exogenous probability[12] γ.

The model, like many other SIR-Macro models (Favero, Ichino and Rustichini 2020 and Eichenbaum, Rebelo and Trabandt 2020 among others) features an health system capacity constraint: if the number of symptomatics patients, at any given time, exceeds the capacity of the health system to properly treat them, the mortality rate increases. More specifically, symptomatic agents at the beginning of the period die with exogenous probability δ in case the health system capacity constraint is not binding. If the constraint is instead binding, the death probability grows by a factor[13] ξ:

$$\delta_t = \delta \left[1 + \xi \mathbb{1}_{(F_t/N)>g}\right] \tag{9}$$

where g is the indicator of the health system capacity, say the availability of hospital beds.

3.4 Dynamics

The model dynamic is summarized by five equations that describe the evolution of the status of each individual. The first describes the transition to the status of infected and symptomatic:

I will instead explicitly model schools.

[12]Differently from Favero, Ichino and Rustchini (2020), I do not model explicitly the different demand of health services by different patients depending on the severity of the symptoms, which are all equal in my model, so that the probability to recover is constant.

[13]Eichenbaum, Rebelo and Trabandt (2020) model instead the death rate as an increasing and convex function of the number of infected,

$$f_{ij,t+1} = (1-\Delta_t)(1-\Gamma) f_{ij,t} + P(1-\Gamma) a_{ij,t} \tag{10}$$

where $\Delta_t \sim Ber(\delta_t)$ is a bernoulli random variable equal to one with probability equal to the death probability δ_t, $\Gamma \sim Ber(\gamma)$ is a bernoulli random variable equal to one with probability equal to the recovery probability γ and $P \sim Ber(\rho)$ is a bernoulli random variable with probability equal to the probability of developing symptoms ρ. Symptomatics agents in t are still symptomatic in $t+1$ if they do not recover and if they do not die, while asymptomatics agents become symptomatics if they do not recover and if they develop symptoms. The second equation describes the transition to the status of infected and asymptomatic:

$$a_{ij,\,t+1} = (1-\Gamma)(1-P) a_{ij,t} + H_{ij,t} s_{ij,\,t} \tag{11}$$

where $H_{ij\,t}$ a bernoulli random variable equal to one in case of infection in period t, defined as follows:

$$H_{\,ij,t} = \begin{cases} 1 & \text{prob} \quad 1-(1-\pi)^{\bar{F}_{i,t}} (1-\bar{\pi})^{\bar{A}_{ij,t}} \\ 0 & \text{otherwise} \end{cases} \tag{12}$$

$\bar{F}_{i,t} = F_{-i,t} + \eta^f_{i,t}\, \psi(F_t - F_{-i,t})$ is the number of symptomatics with whom each susceptible is matched, equal to the number of symptomatics in his family $F_{-i,t}$

$$F_{-i,t} = \sum_{\{\tilde{i} \in N(i)\,;\, \tilde{i} \neq i\}} f_{\tilde{i}j,t} \tag{13}$$

plus a fraction $\eta^f_{i,t}$ of all symptomatics who are not in his family and who are able to participate in social activities $\psi(F_t - F_{-i,t})$. The number of asymptomatics with whom he is matched is instead $\bar{A}_{ij,t} = A_{-i,t} + A_{-j,t} + \eta^a_{i,t} A_{-i\,-j,t}$, and it is equal to the number of asymptomatics in his family $A_{-i,t}$

$$A_{-i,t} = \sum_{\{\tilde{i} \in N(i)\,;\, \tilde{i} \neq i\}} a_{\tilde{i}j,t} \tag{14}$$

plus the number of non-quarantined asymptomatics in the firm m_j where he works:

$$A_{-j,t} = \sum_{\{\tilde{j} \in m_j\,;\, \tilde{j} \neq j\}} (1-k_{i,t})\, a_{i\tilde{j},t} \tag{15}$$

plus a fraction $\eta^a_{i,t}$ of all non-quarantined asymptomatics who are not in his family or workplace

$$A_{-i-j,t} = \sum_{\{j \notin m_j \,;\, i \notin N(i)\}} (1 - k_{i,t})\, a_{ij,t} \tag{16}$$

A susceptible agent i is randomly matched, in period t, with a fraction η^f_{it} of the symptomatics that participate in social activities and with a fraction η^a_{it} of the non-quarantined asymptomatics. I assume that he is also randomly matched with a fraction η^s_{it} of the susceptible who are not in his family and workplace $S_{-i-j,t}$ and with a fraction η^U_{it} of the immunes who are not in his family or workplace $U_{-i-j,t}$. The total number of individuals with whom he is matched in period t is $\eta_{i,t}N = \eta^f_{it}\psi(F_t - F_{-i,t}) + \eta^a_{it}A_{-i-j,t} + \eta^s_{it}S_{-i-j,t} + \eta^u_{it}U_{-i-j,t}$. I assume that this total number of random matches does not change over time and across individuals and I define $\eta = \eta_{i,t}$ as the density of the economy. Dense economies (high η) are either characterized by very social agents, for instance that regularly meet friends, or are densely populated, so that public transportation shops and, in general, public places, are more crowded, offering more contagion opportunities. I will relax this assumption in section 7, allowing for heterogeneous social contacts across individuals although not over time[14].

The third dynamic equation is for the transition into the status of immune, which is absorbing, since I assume that recovering entails the development of antibodies that protect for life from future infections:

$$u_{\,ij,t+1} = \Gamma f_{\,ij,t} + \Gamma a_{\,ij,t} + u_{\,ij,t} \tag{17}$$

In this simple formulation I do not model the possibility of developing antibodies for susceptible without getting the diseases and recovering, as it would be possible if a vaccine were available. The fourth dynamic relationship describes the persistence in the status of susceptible:

$$s_{\,ij,t+1} = s_{\,ij,t}(1 - H_{ij\,t}) \tag{18}$$

[14]The number of social contacts might actually be endogenous to the progression of the epidemic, and therefore time-changing, if the agents optimally choose to reduce the contagion risk avoiding to meet others (Eichenbaum, Rebelo and Trabandt 2020). I will abstract from this feature.

which happens in case susceptibles do not contract the infection. The last dynamic relationship is for the transition into the status of dead which is, obviously, absorbing:

$$d_{ij,t+1} = \Delta_t(1-\Gamma)f_{ij,t} + d_{ij,t} \tag{19}$$

since, by assumption, only symptomatics who do not recover can die. An important statistic to monitor the progression of the epidemic is the effective reproduction number R equal to the number of new infections per each infected individual:

$$R_t = \frac{1}{F_t + A_t}\sum_{i=1}^{N} H_{ij,t} \tag{20}$$

A high effective reproduction number means that pathogen spreads quickly. Containing the epidemics entails bringing the effective reproduction number down to a small level. The model does not allow a closed form solution but can be simulated. In the next section I discuss the solution procedure in detail.

4 Model Solution

I calibrate the model to Italy for a generic pathogen, without focusing exclusively on Covid-19 to make the analysis as general as possible. That said, some of the parameters will refer to Covid-19 or, to be precise, to what is known about Covid-19, given the great uncertainty around many of its distinctive features. Table 1 summarizes all parameter values used for the baseline simulation. I discuss extensively the robustness of the results to several alternatives in section 7. It is important to stress that the scope of my analysis is not to have a detailed framework to do forecasting on the epidemic dynamics in a particular country or for a particular pathogen as Favero, Ichino and Rustichini (2020) do for Italy. Those models need to be much more detailed in terms of age, industrial structure and social interactions in order to produce credible numbers. My model is just a stylized conceptual framework that highlights the basic trade-offs behind different policy alternatives to cope with an epidemic.

Table 1: **Prameters**

Parameter	Description	Value
δ	Death probability	0.025
ξ	Death prob multiplier if binding capacity constraint	2
g	Health system capacity	0.2
ρ	Probability of symptomms	0.25
π	Contagion probability, symptomatics	0.108
$\bar{\pi}$	Contagion probability, asymptomatics	0.108
γ	Recovery probability	0.5
η	Density of the economy	0.028
β	Discounting	1
r	Interest rate	0
θ	Fraction of the wage to non-workers	0.8
ψ	Fraction of symptomatics in social activities	0.1
K	Percentage of quarantined asymptomatics	0
α	Returns to scale	1
λ	Probability to reopen a closed firm	0.99
W	Labor force participation	0.6
z	Fraction of workers below which a firm does not open	0.2
τ^w	Tax rate on labor income and transfers	0.3
τ^y	Tax rate on profits	0.3

4.1 Parameters and Calibration

I simulate an economy with $I = 500$ families[15] at the weekly frequency. To set the family composition, I use data from the Italian Institute of Statistics (ISTAT) relative to 2019. According to the data, 31% of the families are composed by just one member, 27% by two members, 20% by three members, 16% by four members and 6% by 5 or more. I cap the last group to 5 members and create families according to this size distribution. The resulting number of agents in the economy N is slightly more than 1200, which entails an average family size of $\bar{n} = N/I \approx 2.5$. The labor force participation or employment rate is $W = L/N = 0.6$ consistently with ISTAT data.

For the size distribution of firms I use again data from ISTAT. First I fix the fraction of small firms. 95% of the Italian firms in 2017 had less than 9 employees and 44% of the workers were employed in firms with 9 employees or less. Scaling down to the dimension of the simulated economy, I fix the number of small firms with one employee $|m_j| = 1$ to 0.44. As for

[15] A bigger number of families delivers essentially the same results but at the cost of significantly increasing the computational time.

the other firm sizes, I draw them from a uniform distribution between zero and a maximum value O. The number of firms J and the maximum firm size O are then calibrated so that the total number of workers in the firms of size bigger than one is equal to 0.56% and such that the average firm size is equal to 3.87 employees, consistently with ISTAT data. The resulting values are a maximum size $O = 12$ employees (around 4% of the workers) and $J = 64$ firms. This last number implies the presence of 1 firm every 7.8 agents, in line with the number of 1 firm every 10 individuals from ISTAT. Matching this last value entails having a slightly bigger firm size that will accelerate the diffusion of the pathogen. I analyze the robustness to alternative values in section 7.

The lower bound number of workers below which a firm cannot produce is $\bar{M}_j = \lfloor zM_j \rfloor$ with $z = 0.2$. Thus a firm with 10 employees cannot open if less than 3 of them show up to work. This limit is deliberately very low to avoid increasing the long run costs of the no-policy alternative too much. The production function has constant returns to scale $\alpha = 1$. The probability λ to reopen a closed firm, either because of the lockdown or because there are not enough workers is 0.99 per week. This means that if a lockdown lasts for 2 months, on average only 92.3% of the closed firms will reopen when it ends. I chose such an high value for the probability to reopen to avoid increasing the long-run cost of the lockdown too much. It is equivalent to assuming a massive public intervention, for instance in the form of liquidity assistance, that prevents closed firms with little cash flow and zero earnings to avoid becoming insolvent.

I assume that $\psi = 0.1$, which means that 90% of the symptomatics are randomly excluded from social interactions, and that identity of those symptomatics varies over simulation runs. I also assume that the asymptomatics are not quarantined in the baseline, no-policy, scenario, so $\kappa_{i,t} = 0 \forall \{i, t\}$, which means that all asymptomatics go to work and participate to social activities. I calibrate the density parameter η to have, given the family and firm structure and given the population size, the average number of contacts per individual used by the the Istituto Superiore di Sanità (ISS) (2020) for the covid-19 projections. Averaging their figures over all age classes, with weights equal to the actual population size in 2019 from ISTAT, I obtained 18.5 average contacts per individual. In the model, the total number of contacts SC_{ij} for agent i working in firm j is equal to his family size minus 1 plus the number of

coworkers plus the number of random matches: $SC_{ij} = |n_i| - 1 + |m_j| - \mathbb{1}_{|m_j|>0} + \eta N$. I calibrate η so that $(1/N)\sum_{i=1}^{N} SC_{ij} = 18.5$. The resulting value is $\eta = 0.011$ and the resulting number of random matches $0.011 * N$. Then I randomly extract without replacement, for each susceptible agent i working in firm j and for each period t, a subset $e_{i,t}$ of $\lceil 0.011 * N \rceil$ elements from the set $N_{-i-j,t}$ of all agents who are not dead at time t and who are not in family i and firm j. Then I obtain $\eta^f_{i,t}$ counting the number of symptomatics that engage in social activities in the set $e_{i,t}$ and dividing it by the total number of symptomatics that engage in social activities who are not in family i: $\eta^f_{i,t} = \frac{1}{F_t - F_{-i,t}} \sum_{i \in e_{i,t}} f_{ij,t}$. The fraction $\eta^a_{i,t}$ is instead equal to the number of non-quarantined asymptomatics in the set $e_{i,t}$ divided by the total number of non-quarantined asymptomatics at time t who are not in family i and firm j: $\eta^a_{i,t} = \frac{1}{A_{-i-j,t}} \sum_{i \in e_{i,t}} (1 - \kappa_{i,t}) a_{ij,t}$. Similarly, $\eta^s_{i,t}$ and $\eta^u_{i,t}$ are, respectively equal to the number of susceptibles in set $e_{i,t}$ divided by the total number of susceptibles at time t who are not in family i and firm j, $\eta^s_{i,t} = \frac{1}{S_{-i-j,t}} \sum_{i \in e_{i,t}} s_{ij,t}$, and to the number of immunes in set $e_{i,t}$ divided by the total number of immunes at time t who are not in family i and firm j, $\eta^u_{i,t} = \frac{1}{U_{-i-j,t}} \sum_{i \in e_{i,t}} u_{ij,t}$.

I simulate a very aggressive pathogen that is difficult to treat. The reason is to have enough variation in the death rate under different policy alternatives, since it would be mostly very close to zero or actually zero with milder pathogens. Moreover, if the pathogen is not aggressive or if it is easy to treat, there is actually no actual gain from lockdowns or testing strategies, making the whole exercise not meaningful. The baseline death probability is $\delta = 0.025$ per week. To make the simulation more realistic, I also add an age structure to the model, assuming that a fixed fraction of the population has a higher death probability, say because they are old or because they have preexisting health conditions that make the infection more problematic. I assume, conservatively, that 17% of the population has a 50% higher death probability both in case of binding health system capacity constraint or not, where 17% is the fraction of individuals above 70 in Italy. In the model, some of those agents are pensioners ($j = 0$), but some others work ($j \neq 0$), which entails assuming that there is an equal number of healthy olds and sick youngs. The multiplier in case of health system stress (binding capacity constraint) is $\xi = 2$, similarly to what Eichenbaum, Rebelo and Trabandt (2020) assume for the multiplier at the peak of the epidemic. For the baseline simulation, I consider a very

high capacity oh the health system $g = 0.2$ that implies a non-binding capacity constraint for all possible epidemic trajectory, but I discuss extensively the results under different capacity levels, which, anticipating the results, are crucial in determining the opportunity of a lockdown. In the simulation, I draw a death probability for each symptomatic infected independently, to emphasize that the way the epidemics unfolds is also the result of random variation, which is also the reason why it is important to simulate the model many times. The alternative of killing a fixed fraction of the symptomatics results in a death rate that tracks the behavior of the symptomatics. I assume that one quarter of the asymptomatics develop symptoms, $\rho = 0.25$ and half of both the symptomatics and the asymptomatics recover $\gamma = 0.5$. As for the death rate, I draw the probabilities to recover and to develop symptoms independently. The average death rate in the population upon infection with a non-binding health system capacity constraint is therefore equal $\delta^{avg} = 0.025 * 0.25 * (0.83 + 0.17 * 1.5) = 0.68\%$, which is in line with Eichenbaum, Rebelo and Trabandt (2020) for the Covid-19 case and equal to the death probability estimated and used by the ISS in Italy for the covid-19 projections. Give this average death rate, the probability to remain infected for one period a non-binding health system capacity constraint is equal to $(1 - \pi) * [1 - \rho + \rho(1 - \delta^{avg})] = 0.676$. The consequence is a quite high median duration of the infection without any policy intervention of 6 weeks, that does also translate in a high peak of the infection.

I assume that the transmission probability is the same for both symptomatics and asymptomatics. I calibrate the value to have a basic reproduction number R_0 equal to 2 in line with the Covid-19 figures in Chowdhury et al. (2020) and similarly to Influenza and Ebola, but bigger than MERS (around 0.5) and SARS (close to 1) and significantly smaller than HIV (around 3) and Measles (around 15). Given the 18.5 average contacts per individual, I set $\pi = \bar{\pi} = 2/18.5 = 0.1081$.

The interest rate r in the baseline simulation is equal zero, so all agents are allowed to borrow without cost, and there is no discounting $\beta = 0$. The government transfer $\theta = 0.8$ of the wage to pensioners, minors, symptomatics, quarantined asymptomatics and to all workers of the closed firms as an effect of the lockdown. The fixed wage w is normalized to 1 without loss of generality. Wages, transfers and profits are all taxed at the 30% rate ($\tau^w = \tau^y = 0.3$).

4.2 Policies

I simulate the response of the economy to the pathogen shock conditionally on 3 different policies. The first consists of letting the pathogen spread without any restriction. The second is a lockdown that imposes social distancing on a large scale, closing a certain number of firms ($M_k = 0$ for some $k \in \{1, 2, \dots J\}$) and reducing the density η (number of social interactions-random matchings). The third entails instead an extensive testing to identify and quarantine the asymptomatics. This last policy makes sense only for pathogens that, in a non-negligible percentage of cases deliver asymptomatic infections, as in the Covid-19 case, but not for mostly symptomatics infections such as smallpox. With respect to the covid-19 pandemic, the first approach is close to what the Swedish government decided to do or to what the British government initially thought of doing before looking at the catastrophic scenarios in Ferguson et al. (2020); the testing strategy is instead associated to South Korea and Taiwan, while the lockdown to most other countries including, among others, the US, France, Germany and Italy, albeit at different degrees. I assume that all policies are equally costless to implement. In case of the lockdown, the assumption is that there are no psychological costs associated to the reduction of social activities and that it is possible to enforce it without, say, deploying more police officers or military personel. In case of the testing policy, the assumption is that screening and tracking agents is feasible at a negligible cost regardless of the scale of the operation, which is indeed a very strong assumption. For instance, the unavailability of a sufficient number of testing devices and specialized professionals and laboratories to handle them was one of the main challenges faced by many countries in the midst of the Covid-19 outbreak. In addition, I assume that the government does not raises taxes to cover the transfers to the sick and to the workers who are forced home by the lockdown, but rather that it lets the debt increase.

The lockdown policy starts 4 weeks after the pathogen shock, runs for a minimum of 8 weeks and then it ends only if the two-weeks effective reproduction number of the pathogen drops below 0.5. The choice of the starting date is arbitrary but reasonable: it takes time for the pathogen to spread at a sufficient degree to be considered a treat to public health and it takes some time for the government to implement a policy. In section 6, I analyze the robustness of the results to alternative starting dates. There are two reasons why I impose the

minimum length. The first is that it is realistic, both because of the time needed to collect and process the epidemiological data and because of the delay between information acquisition and political decisions. The second is that contagions might go up as soon as the lockdown ends and, without a minimum length, there is the risk of opening too soon, when the effective reproduction number is small without a sufficient number of immunes to stem a second way of infections. The choice of 8 as minimum weeks constraint might actually seem high, especially if compared to the optimal lockdowns in Piguillem and Shi (2020). However, as I show in section 6, shorter minimum weeks constraints are often not binding, in the sense that it takes longer to bring the effective reproduction number below the threshold. Anyway in section 6 I analyze extensively the consequences of different minimum lengths. The choice of 0.5 as a threshold for the effective reproduction number is arbitrary, although realistic based on what many governments did for the Covid-19 pandemic, but the main results of the analysis proved to be very robust to alternative values betweem 0.3 and 0.7. Moreover, given the high costs of the lockdown, threshold values below 0.3 seem to high, while values above 0.7 trigger second round of infection in many more cases, making the lockdown not effective. Alvarez, Argente and Lippi (2020) and Farboodi, Jarosch and Shimer (2020) show that the optimal response to the epidemic involves a slow relaxation of social distancing. In my simulation I assume instead that all economic and social activities come back to normal as soon as the effective reproduction number drops below the threshold.

The baseline lockdown policy consists of closing 50% of the firms and reducing η by three quarters, so the number of remaining social interactions is $\psi/4$. The choice of which firm to close is obviously crucial: for the same proportion of closed firms, in case the closed firms are smaller than the open there is a milder effect with respect to the case where big firms are closed. Typically governments opt to close firms whose production is not considered essential or strategic, keeping open, for instance, all economic activities related to the food production and supply chain, to the health system. Since there are both big and small firms in those sectors (big supermarkets and cornershops, biotech companies and small pharmacies), I assume that firms are closed at random but proportionally on size. In greater detail, I choose the parameter ε such that the following expression delivers the target value of 50% closed firms:

$$\frac{1}{J}\sum_{l=1}^{\tilde{l}}\sum_{j=1}^{J} \quad \lceil \varepsilon \mathbb{1}_{|m_j|=l} \rceil = 0.5 \tag{21}$$

where $\tilde{l} = \max |m_j| \, j \in \{1, 2, \dots J\}$ is the maximum firm size. Then a random fraction of ε firms of size size $|m_j|$ is closed, rounding to the next positive integer (in case the product of ε and the total number of firms of given size in not an integer). With this rule, a lot of big firms are closed even in case of mild shutdowns, which is reasonable since big firms offer more contagion opportunities. Moreover, closing 50% of the firms entails having 52% of the workers still at work during the lockdown, which is slightly smaller than the 60% estimated by Barbieri, Basso and Scicchitano (2020) for Italy and used by Favero, Ichino and Rustichini (2020).

The testing policy consists instead of an extensive screening that can successfully and instantaneously isolate K percent of the asymptomatics every period. Thus:

$$K = \frac{\sum_{i=1}^{N} k_{i,t} a_{ij,t}}{\sum_{i=1}^{N} a_{i,t}} \tag{22}$$

with $K = 0.5$ as a baseline. I assume that the testing policy starts 4 weeks after the pathogen shock exactly as the lockdown and, for simplicity, I also assume that it is immediately possible and costless to isolate the target value of asymptomatics. Both assumptions are indeed extreme and hardly realistic. In section 5.3 I will discuss the robustness of the results case of a positive marginal cost for the test, assuming that they are financed in deficit and, therefore, that they do not impact on current GDP. In section 6 I will instead discuss the results in case it is feasible to isolate only a smaller fraction of the asymptomatics.

4.3 Simulations

I start each simulations with the infection of 2 random agents that develop symptoms, upon infection, with probability ρ. For instance, they could have been just back from a trip abroad and not isolated and quarantined, or they could have been in close contact with the animals that were intermediate carriers of a pathogen that just mutated to attack humans (zoonosis). I will refer to this initial two infections as the pathogen shock. I simulate the evolution of

the economy under different policies 500 times[16] and discard the cases where there is only a limited circulation of the pathogen, for instance because the first infected agents recover before transmitting the pathogen to a sufficient degree to start the epidemic. Such cases are however very rare. In other words, given the model calibration and given the structure of the economy, the minimum critical mass, in the words by Piguillem and Shi (2020), is very small.

Two remarks before proceeding. First, in the model the agents do not respond to the initial spread of the pathogen and to the implemented policies, differently from some previous contributions (Piguillem and Shi 2020, Farboodi, Jarosch and Shimer 2020, Collard et al. 2020, Garibaldi, Moen and Pissarides 2020, Bethune and Korinek 2020, Chang and Velasco 2020, Jones, Philippon and Venkateswaran 2020, among others). For instance, workers cannot choose to stay home if the firm that employs him is open even if doing so increases the contagion risk thereby decreasing his expected future wealth. One possible way to rationalize this assumption is the absence of monetary transfers to non-sick works who do not show up at work coupled with the absence of sufficient savings and/or borrowing capacity to support consumption. Second, both lockdowns and testing policies involve a mandatory quarantine for the symptomatics, which might be problematic to enforce in case of mild symptoms, requiring a considerable effort. This is equivalent to assume that the symptoms are so severe that this enforcement is not costly, as it is the case, for instance, for Ebola.

5 Results

I organize the discussion of the main simulation results in four distinct subsections. In subsection 5.1 I discuss the impulse response to the pathogen shock. In subsection 5.2 I compare instead the performance of the three main policies looking at the distribution of their main outcomes over different simulation runs. In subsection 5.3 I focus instead on the dismal trade-offs. Subsection 5.4 finally discusses the robustness of the results to alternative parameters and assumptions. One potential shortcoming of my simulation exercise is that I compare lockdown and testing strategies for given implementation details without computing the optimal, welfare-maximizing ones. The problem is that welfare maximizing policies are contingent on

[16]More simulations result essentially in the same results. In other words, the amount of random variation that can impact on the final results is not too big.

the value of life used to compute them. The higher this value, the bigger the gains from policies that reduce mortality even at a high output cost. I prefer instead to focus on the trade-offs.

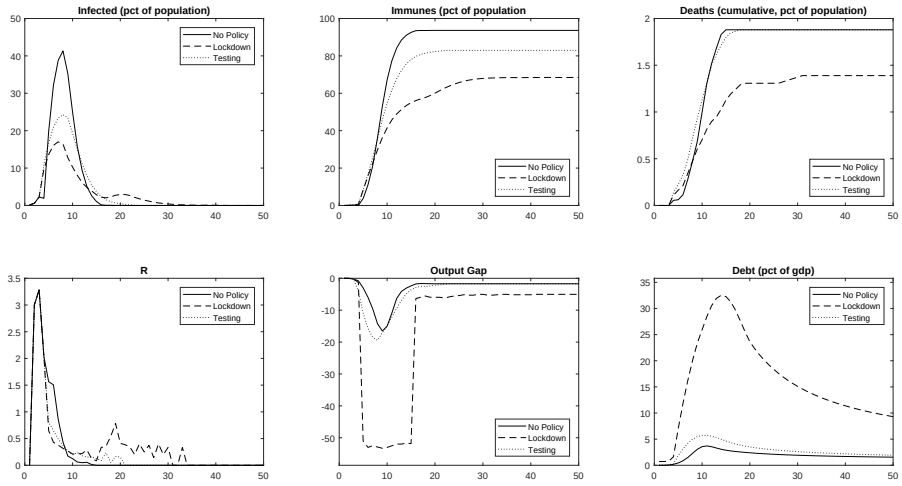
5.1 Impulse Responses

Figure 1 plots one of the possible impulse responses to the initial pathogen shock under the three baseline policies. Given the high contagion rate and given the structure of the economy and the timing of the model, the epidemic unfolds rather quickly and it ends after 17 weeks in case of no-policy intervention, 22 weeks in case of the testing and 40 weeks in case of a lockdown. The lockdown policy lasts for 12 weeks, which means that, after the minimum 8 weeks, it takes an additional four weeks to reduce the two-weeks effective reproduction number below 0.5. In the no-policy scenario, slightly more than 40% of the population is infected at the peak, which happens 9 weeks after the shock. The lockdown is effective at smoothing the infection peak, thereby easing the pressure on the health system. The peak in case of lockdown is around 18% of the population at the 8th week after the shock, which is less than half with respect to the no-policy scenario. There is a slight increase of contagions as soon as the lockdown ends, which however fades away quite rapidly. The testing policy results are in the middle: a peak of around 24% of the population at the 9th week and a fast decrease afterwards. At the end of the epidemic, roughly two thirds of the population is immune in the lockdown case, while slightly more than 80% in the testing case and around 95% in the no-policy case. Deaths at the end of the epidemic are much lower in case of a lockdown, around 1.25% of the population versus the 1.8% of the no-policy and testing alternatives. The cost of the lockdown policy is an output decrease and a debt increase. The output gap drops deeply when the lockdown starts, both because many firms are closed by the government and because of many symptomatic workers in the firms that remain open[17]. The no-policy and testing alternative also entail a non-negligible output loss, especially around the peak of the infection, since the symptomatics (in the no-policy case) and the quarantined asymptomatics (in the testing case) cannot work. After the epidemic, the output gap is very close to zero in

[17]The rule that I use to close firms, proportionally on size, implies that many of the big firms are closed. Therefore, with $\alpha = 1$ the output loss in case of a lockdown is always above the fraction of closed firms even absent any other symptomatic individual in the economy that cannot go to work.

the no policy and testing scenarios, since the high value of the probability to reopen a firm $\lambda = 0.99$ implies that most firms do not close and since the death rate is, after all, quite small. In case of a lockdown, there are some firms that do not reopen despite the high value of λ due to the prolonged period of inactivity, and the output gap stabilizes at around -4%. The debt also increases very quickly in the lockdown case as an effect of both the decreased tax proceedings, due to the high number of closed firms with zero taxable profits, and to the high number of temporarily unemployed workers whose income is supported by the government. The debt tends to revert as soon as the lockdown ends, but, at the end of the epidemic, it is still very much above the level in the no-policy and testing scenarios, around 10% of GDP versus less than 2%.

Figure 1: **Impulse Response to the Pathogen Shock. Example 1.**

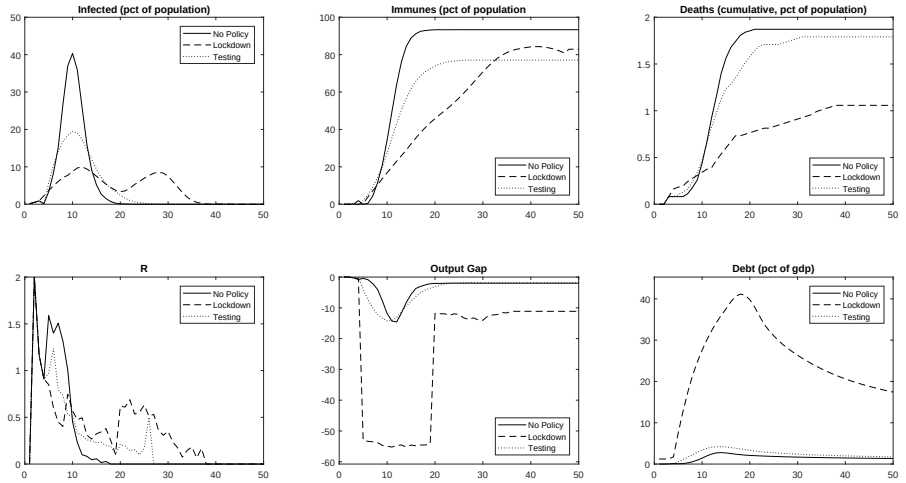


Impulse response to the pathogen shock consisting of two randomly infected agents. Upper left panel: total number of infected agents (sum of symptomatics and asymptomatics) as a percentage of the pre-epidemic population size N. Upper central panel: total number of immunes as a percentage of N. Upper right panel: total number of deaths as a percentage of N. Lower left panel: one-week effective reproduction number defined in equation 20. Lower central panel: output gap. Lower right panel: Debt to Gdp ratio, where the debt at time t (sum of all budget deficits or surpluses from 0 to t.)

The impulse response in figure 1 is actually just one of the possible responses of the economy. For instance, contagions might actually go up a lot when the lockdown ends with a second peak of infections. This is more likely if the lockdown is very effective and fast at reducing the effective reproduction number but such that the number of immunes is not low

enough. Figure 2 plots the impulse responses for such a case. In general, on the important features of the model is that it highlights the huge variability of results that different policies might yield. In the next section I study extensively this variability.

Figure 2: **Impulse Response to the Pathogen Shock. Example 2.**



Impulse response to the pathogen shock consisting of two randomly infected agents. Upper left panel: total number of infected agents (sum of symptomatics and asymptomatics) as a percentage of the pre-epidemic population size N. Upper central panel: total number of immunes as a percentage of N. Upper right panel: total number of deaths as a percentage of N. Lower left panel: one-week effective reproduction number defined in equation 20. Lower central panel: output gap. Lower right panel: Debt to Gdp ratio, where the debt at time t (sum of all budget deficits or surpluses from 0 to t.)

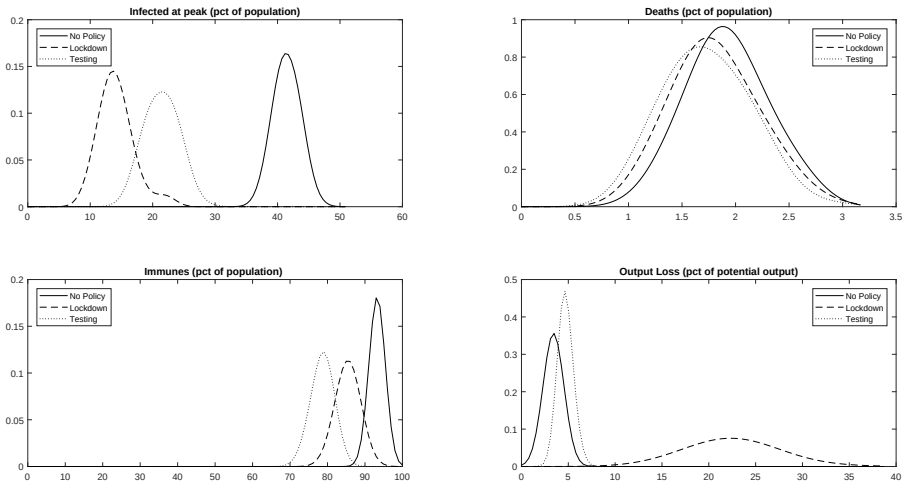
5.2 Policy Comparison

To account for the variability of responses of the economy to the pathogen shock, I simulate the model 200 times[18] and discard the simulations with no epidemic. This is actually an unlikely but possible event: if the two initially infected agents do not work and live in small families and if they recover quickly, they might not be able to spread the pathogen sufficiently to trigger the epidemic. Figure 3 plots the empirical distributions over the simulation runs of the percentage of infected at peak, of the death rate at the end of the epidemic, of the percentage of immunes at the end of the epidemic and of the cumulative output loss during

[18]More simulation runs result in essentially the same results for the distributions of the main variables of interest.

the epidemic as a percentage of potential output[19]. The summary statistics are summarized in table 2, together with two additional variables: the debt to gdp ratio at the end of the epidemic and inflation, defined as the percentage difference between the price at the end of the epidemic and the price at the beginning.

Figure 3: **Policy Comparison**



Empirical probability density functions of the response to a pathogen shock over simulation runs conditional on three baseline policies. Upper left panel: total number of infected agents (sum of symptomatics and asymptomatics) at the peak of the infection as a percentage of the pre-epidemic population size N. Upper right panel: total number of deaths at the end of the epidemic as a percentage of N. Lower left panel: total number of immunes at the end of the epidemic as a percentage of N. Lower right panel: Cumulative output loss during the epidemic as a percentage of potential GDP.

Lockdowns make epidemics last longer: a median of 37 weeks (95% confidence interval [32;47]) as compared to the 22 [20;27] weeks of the no-policy alternative and 28 [23;35] of the testing policy. This duration is defined as the first period with zero infected in the economy. The main advantage of this slower unfolding of the epidemic is the possibility to properly treat all patients and, in case of a new pathogen such as the Covid-19, to figure out the best medical treatment. As a further evidence of the lower pressure on the health system, the median percentage of the population with the pathogen at the peak of the infection is equal to 13.8% [10%;19.5%] with a lockdown as compared to 41.8% [38.9%;44.5%] in the no-policy scenario and to 21.3% [17.6%;25.5%] of the testing case. In all three policy scenarios, the peaks in the

[19]The output loss can be annualized simply adding the output gap at the end of the epidemic multiplied by 52 minus the duration of the epidemic in weeks.

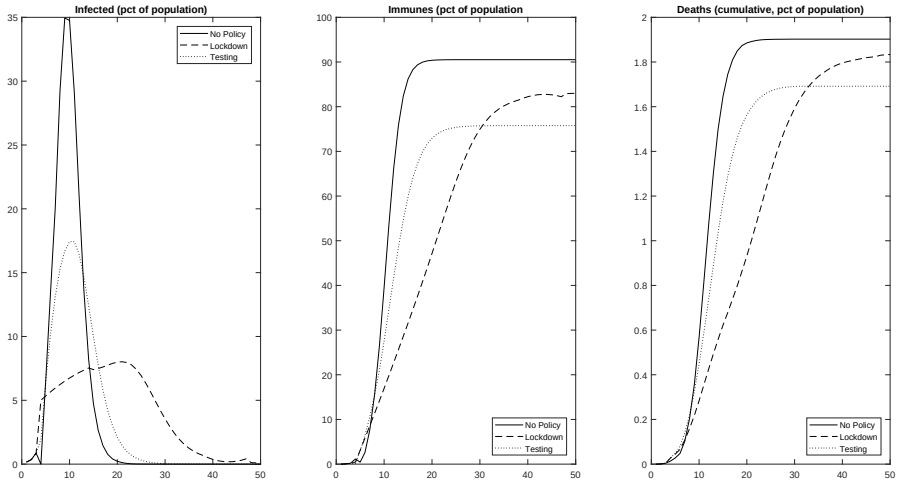
contagion curves are actually quite high as a consequence of the high probability to remain infected for another week in the baseline simulation, equal to 67.6%. Another important effect of the the lockdowns is that they reduces the total number of infected agents, resulting both in a lower fraction of immunes at the end of the epidemic, which is not a desirable outcome, and in a lower death rate, which is perhaps there main task. The median fraction of immunes at the end of the epidemic with a lockdown is in fact 85.3% [80.9%;88.5%] versus 93.3% [91.7%;94.5%] in the no-policy scenario. In section 7 I analyze if this lower fraction of immunes exposes the economy to the risk of a second way of infections and I explore the possible consequences. The death rate is instead 1.95% [1.38%;2.68%] in the no-policy scenario and 1.84% [1.22%;2.47%] in case of a lockdown. The testing policy, reducing conagions from asymptomatics, reduces the number of infected even more than a lockdown, resulting in a median of 78.5% [74.4%;81.8%] of immunes at the end of the epidemic and in a median 1.71% [1.13%;2.37%] death rate. For all three policies, the standard deviations of the death rate are quite high, resulting in large confidence intervals. In other words, lockdowns and testing strategies are highly uncertain policies and they do not necessarily reduce the number of deaths in all circumstances. For instance, lockdowns and testing strategies can result in more deaths if, among the lower number of infected, there is a higher share of high-risk individuals, which is more likely in case they live in big families or work in big firms or both. Similarly, if there are a lot of asymptomatics in the big firms before the lockdown starts, there will be an increase in infection and deaths regardless of the lockdown, especially if those agents are in the firms that are not closed.

Figure 4 plots the average impulse responses for the total fractions of infected, immunes and deads[20] over all simulation runs conditional on the three policies, obtained averaging over simulation runs (with an epidemic). As already stressed, lockdowns and testing policies reduce, on average, the number of deaths and, overall, the number of infected, and alleviate the pressure on the health system by smoothing the infection peak.

The main side effect of lockdowns is their high cost, both for the current and for the future generations. The median output loss induced by a lockdown is 22.1% [17.4%;28.7%], almost

[20]I do not plot output and debt because, given the different duration of lockdowns, there is an average smooth response after the 8th week due to open firms in a subset of the simulations only, which is not informative. The effective reproduction number, on the other hand, is too volatile across simulations and so the average is not informative.

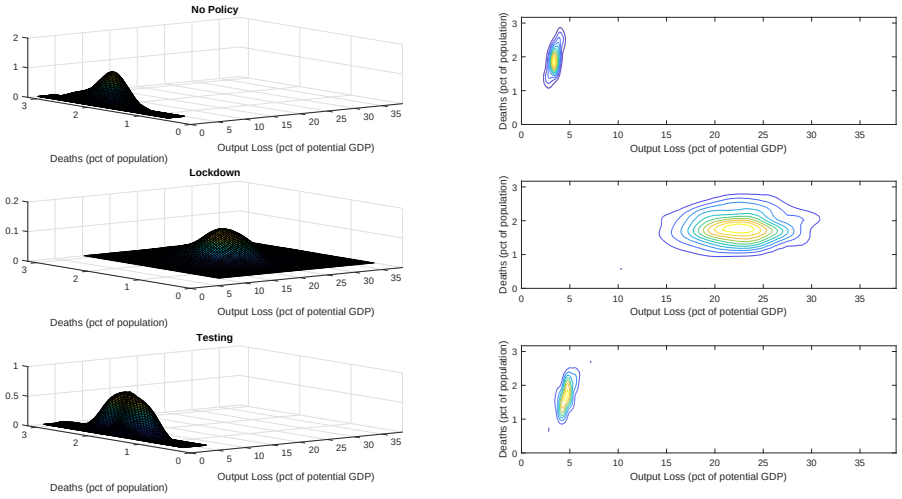
Figure 4: **Impulse Response to the Pathogen Shock, Average**



Average impulse response over 500 simulation runs to the pathogen shock consisting of two randomly infected agents. Left panel: total number of infected agents (sum of symptomatics and asymptomatics) as a percentage of the pre-epidemic population size N. Central panel: total number of immunes as a percentage of N. Right panel: total number of deaths as a percentage of N.

7 times as big as the median loss in the no-policy scenario, equal to 3.5% [2.7%;4.3%] and almost 5 times as big as the loss associated to the testing policy, equal to 4.7% [3.8%;5.7%] (but remember that this loss is computing assuming that tracing and testing asymptomatics is costless). The median debt to gdp ratio increase at the end of the epidemic, which is a measure of the cost for future generations, is instead equal to 15.3% [10.7%;22.5%] in case of lockdown while only 1.5% [1.2%;1.8%] in case of no policy and 2% [1.7%;2.5%] in the testing scenario. Since the government supports income with very generous transfers (80% of the wage) if production and, thus jobs are discontinued, an additional cost of the epidemic is inflation: the median price increase at the end of the epidemic is 2.1% [1.2%;3.1%] in the no-policy case. In case of testing, the inflation cost is lower, a median increase of 1.9% [1.2%;2.7%], as an effect of two contrasting forces: on the one hand, more of the asymptomatics are identified and temporarily suspended from their jobs, but there are less infected agents overall and, therefore, less of the symptomatics who do not go to work. The biggest inflation cost is however associated with lockdowns, that determine a median price increase of 7.8% [3.8%;13%].

Figure 5: **Output Loss and Mortality**



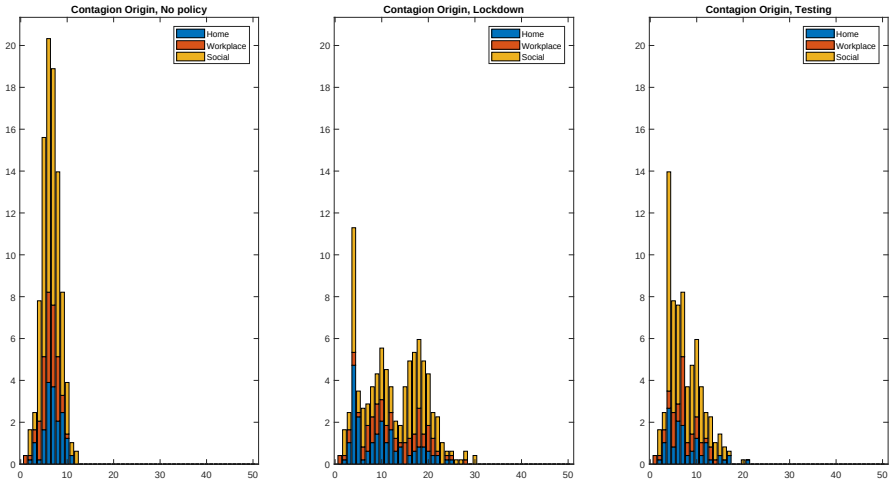
Empirical probability density functions of the simulation results for the three baseline policies (see text for details): no policy (solid line), lockdown (dashed line) and testing (dotted line).

All in all, a lockdown yields, on average, a mortality reduction at a high cost in terms of foregone output and increased debt. The testing strategy, if feasible, appears instead to have a better performance. Figure 5 shows the joint distribution of the death rate and of the output loss for the three policy alternatives. Clearly the output loss determined by a lockdown is very high and variable, while the range of mortality rates is not very different in the three alternatives. In other words, as already stressed, lockdowns do not necessarily reduce mortality: they mainly slow down the progression of the epidemic. In the next subsection, I dig deeper into the relationship between reduced mortality and reduced output and between reduced mortality and increased debt computing the dismal ratios.

To better understand how the policies work, figure 6 plots the breakdown of new contagions due to contacts within the family, at the workplace and due to social activities for one of the possible epidemic trajectories. Since firms are, on average, small (many of them have just one worker) and since the labor market participation is quite low in this calibrated economy, workplace contacts account, on average, for roughly 20% of all contagions[21] in the no-policy scenario. Most of the contagions, around 60% on average, are instead due to social contacts

[21]This number is similar to the 17% of transmissions on the workplace used by Eichenbaum, Rebelo and Trabandt (2020) for the Covid-19 case in the US

Figure 6: **Breakdown of New Contagions, Example**



Breakdown of new contagions, in percentage of the population, due to family, workplace and social contacts conditional on the three baseline policies (see text for details): no policy (left panel), lockdown (central panel) and testing (right panel).

and the remaining 20% to family contacts[22]. Lockdowns and testing strategies reduce the total number of contagions and increase the percentages of contagions due to contacts within the family. In the lockdown case, this is because some firms are closed and some social activities discontinued. In the testing case, because quarantined asymptomatics cannot spread the pathogen on the workplace and in social interactions.

5.3 Dismal Ratios

To summarize the trade-offs between lives saved and output lost and between lives saved and debt increase, I construct two *Dismal Ratios*. The first is the Output Dismal Ratio (ODR), defined as the percentage of the population spared as an effect of the policy, if any, for each extra percentage point of output lost:

$$ODR = \frac{\frac{1}{N}\left(D_T^{nop} - D_T^{pol}\right)\mathbb{1}_{D_T^{nop} > D_T^{pol}}}{\frac{1}{TY}\left(\sum_{t=1}^{T} Y_t^{nop} - \sum_{t=1}^{T} Y_t^{pol}\right)} \tag{23}$$

[22]With respect to the estimates of the importance of different contagions sources for respiratory diseases in Ferguson et al. (2006), I have less contagions within the family (30% in Ferguson et al. 2006) and more from social activities (33% in Ferguson et al. 2006, but excluding schools which I count in this category).

where the superscript *nop* denotes the no-policy scenario and the superscript $pol \in \{lock; test\}$ lockdown or testing policies. The second is the Debt Dismal Ratio (DDR), defined as the percentage of the population spared as an effect of the policy, if any, for each additional unit of debt as a percentage of GDP:

$$DDR = \frac{\frac{1}{N}\left(D_T^{nop} - D_T^{pol}\right)\mathbb{1}_{D_T^{nop} > D_T^{pol}}}{B_T^{pol} - B_T^{nop}} \tag{24}$$

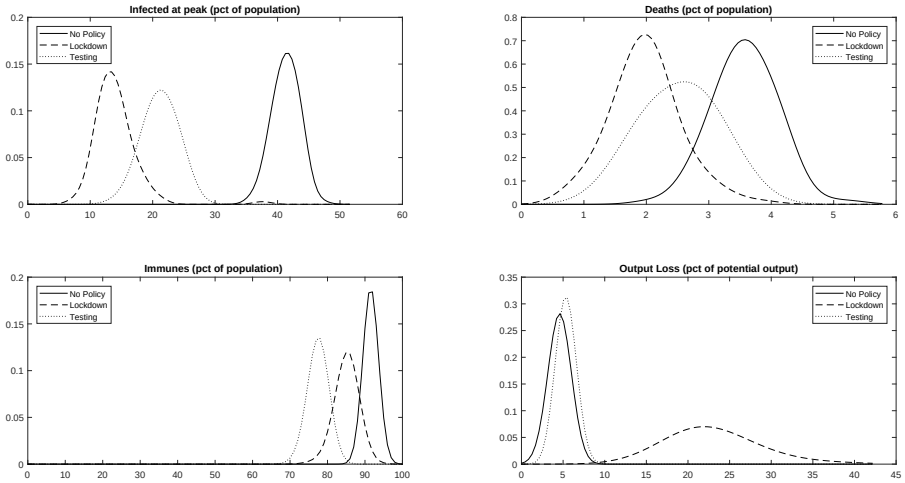
where B_T^{nop} and B_T^{pol} are the debt to gdp ratios, at the end of the epidemic, respectively without any policy implemented and with a policy. Debt itself is computed at the sum of all deficits accumulated from 0 to T, which are the difference between the right hand side and the left hand side of equation 6. The dismal ratios can be also interpreted as measures of the effectiveness of a policy. A zero dismal ratio means that the policy resulted in more deaths with respect to the no-policy case regardless of its performance in terms of output and debt. A negative dismal ratio means instead that there is no trade-off: life can be saved without any cost. A positive dismal ratio means instead that there is a trade-off: saving lives requires an output reduction or debt increase. Importantly, having a small dismal ratio does not mean that the policy is not worth implementing. Borrowing the words of an article that appeared on The Economist (2020) at the time when many governments, including Italy, Spain, France, India and the US announced their lockdowns, computing the dismal ratios it is not the same as "putting a price tag on human life". Saving lives is in fact what governments should pursue. The point is figuring out what is the best way to do it, and by best way I mean without putting an excessive burden on current and future generations. This is also the reason why I focus on the trade-off between lives saved and output lost as in Favero, Ichino and Rustichini (2020), without explicitly computing the optimal welfare-maximizing policy as in most previous contributions (Alvarez, Argente and Lippi 2020; Piguillem and Shi 2020; Farboodi, Jarosch and Shimer 2020, among others): the optimal policies will be contingent on the monetized value of life used to compute them, which is arguably not a desirable choice rule.

The two dismal ratios are unique at each simulation run and depend on how the epidemic unfolds. The first three rows of table 3 report their medians over the simulation runs conditional on the implemented policy, together with the median duration, death rate, output loss

and debt to gdp ratio. The last column of table 3 reports a crucial information to evaluate a policy: the percentage of zero dismal ratios in the simulation runs, which is the fraction of the simulations where lockdowns or testing policies did not result in less deaths with respect to the no-policy alternative. The median ODR of a lockdown is 0.0049 [0; 0.0556]. For a population of 60 millions, this means that a lockdown saves a median of 2940 lives for each unit of output lost. The median DDR of a lockdown is instead 0.0067 [0;0.0752], which translates, for the same population, in 4020 lives saved for each additional percentage point of the debt to gdp ratio. The problem, however, is that in almost 46% of the simulations the lockdown does not translate into a lower number of deaths. Once again, the simulation results show that lockdowns are risky policies, that do not always reduced mortality but that always put a considerable burden on the economy. The low median mortality and the small median output lost of the testing policy imply instead a median ODR of 0.2112 [0; 2.0245] and a median DDR of 0.4074 [0;3.5938]. The median number of lives saved by the testing policy are thus 126720 for each unit of output lost and 244440 for each additional unit of debt. Testing policies, however, do not reduce the mortality in 34% of the cases, which is less than lockdowns but still quite high. Thus testing policies are also risky, although less than lockdowns, and potentially more effective.

Lockdowns and testing policies, however, are very valuable in case the health system capacity constraint becomes binding, as it is likely to be the case at the peak of infections. In the baseline simulations that I just discussed, I assumed that it is feasible to properly treat all patients regardless of their number or, in other words, that there is enough capacity in the health system to potentially treat simultaneously the entire population. This might be a good assumption for a mild influenza or for a bacteria that responds to antibiotics, but not, for instance, for the Covid-19 that required hospitalization and intensive care treatments for a high percentage of the infected. Since this assumption is unlikely to be true for some pathogens, I simulated again the model assuming a smaller health system capacity, $g = 0.05$. The densities of the outcomes over simualtion runs are summarized in figure 7. In this case the lockdown and testing policies avoid the sharp peak of the infection and, by keeping the death rate at its baseline level, save a lot many lives more. This simulation illustrates clearly why lockdowns are praised upon. I assume that $\xi = 2$, which means that the mortality for

Figure 7: **Policy Comparison. Binding Health System Capacity Constraint.**

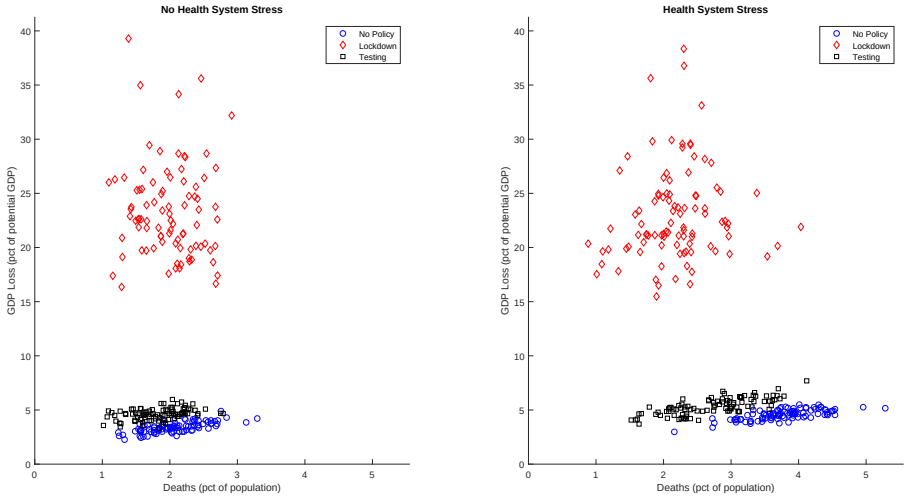


Empirical probability density functions of the response to a pathogen shock over simulation runs conditional on three baseline policies with binding health system capacity constraint $g = 0.05$. Upper left panel: total number of infected agents (sum of symptomatics and asymptomatics) at the peak of the infection as a percentage of the pre-epidemic population size N. Upper right panel: total number of deaths at the end of the epidemic as a percentage of N. Lower left panel: total number of immunes at the end of the epidemic as a percentage of N. Lower right panel: Cumulative output loss during the epidemic as a percentage of potential GDP.

the symptomatics doubles in case of binding health system capacity constraint. The meadian death rate with $g = 0.05$ in case of no-policy implemented is 3.59% [2.77%;4.22%], while it is 1.96% [1.06%;2.84%] in case of a lockdown, with almost the same output loss as in case of non-binding capacity constraint. The resulting median ODR and DDR of a lockdown are therefore much higher, respectively 0.0895 [0.0274;0.1913] and 0.1219 [0.0352; 0.2676]. Perhaps more importantly, lockdowns in this economy reduced do not reduce mortality in just 2.7% of the simulations. The testing strategy, however, is still associated to bigger gains: a median ODR of 0.6906 [-13;16] and a median DDR of 2.7466 [-6;23], with zeros in 11% of the simulations.

Figure 8 summarizes the outcomes of the three policies in the death rate-output loss space for the two cases of unlimited health system capacity (left panel) and in case of $g = 0.05$ (right panel). Lockdowns outcomes are more volatile and imply bigger output losses but, in case of binding health system capacity constraint, they also imply a lower death rate. There is actually a problem in case there is very little capacity in the health system: it is possible

Figure 8: **Output Loss and Mortality With and Without Health System Stress**



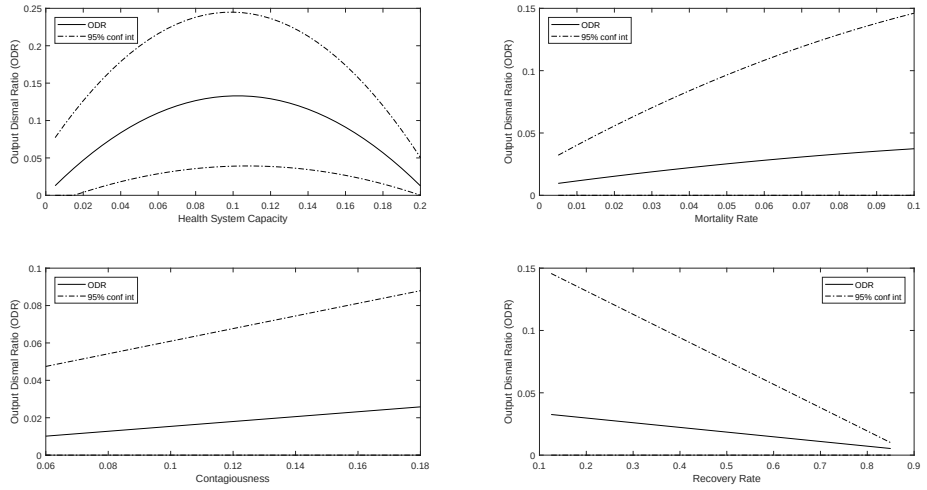
Scatter plot for 100 randomly choosen simulation results for the three baseline policies: no policy (circles), lockdown (diamonds) and testing (squares). Left panel: non binding health system capacity constraint. Right panel: binding health system capacity constraint ($g = 0.05$).

that the lockdown and testing strategies are not enough to avoid the stress and the capacity constraint becomes binding even with a lockdown. In the benchmark simulation, this happens with $g = 0.01$. In this case the median ODR and DDR of a lockdown are, respectively, 0.0063 [0;0.0838] and 0.0089 [0;0.1183], with zeros in 47% of the simulation. Not very differently from the baseline case. This evidence does not suggest, however, that lockdowns are not appropriate in case very small capacity of the health system, but rather that they must be more severe in order to further smooth the peak of the infection.

The gains from lockdowns and testing policies are crucially dependent not only on the health system capacity but on the full set of characteristics of the pathogen. Figure 9 plots[23] the ODR of a lockdown as a function of the health system capacity (upper left panel), of the mortality rate (upper right panel) of the contagiousness of the pathogen (lower left panel) and of the recovery rate (lower right panel). I omitted the relationship with the probability of developing symptoms because increasing this probability is equivalent to increasing the death rate. I also omitted the DDR pictures because they are very similar. As already stressed, the

[23]The lines in the plots are predictions based on quadratic regression of the medians and 95% confidence interval bounds over the simulation runs.

Figure 9: **Output Dismal Ratio (ODR) for Different Pathogens**



Median Output Dismal Ratio (ODR) of a lockdown as a function of health system capacity (upper left panel), mortality rate (upper right panel), contagiousness (lower left panel) and recovery rate (lower right panel). Predictions based on quadratic regressions.

gains from a lockdown are maximized in case of intermediate capacity of the health system, since the capacity constraint becomes binding even with a lockdown in case of very small capacity and since it never becomes binding even without any policy intervention in case of very high capacity. The gains from a lockdown also increase with the mortality rate and with the contagiousness of the pathogen, but they are decreasing in the recovery probability. All in all, the evidence summarize in figure 9 suggests that lockdowns are more appropriate for aggressive pathogens such as Covid-19 or Ebola unless the recovery rate is high as in the case of Measles or Chickenpox. However the confidence intervals almost always include zero, with the only exception of intermediate capacity of the health system, which means that there is the possibility that a lockdown will not save any lives.

Testing policies obviously make sense only for pathogens that yield asymptomatic infections for a non-negligible number of individuals. The gains from testing strategies are also higher at intermediate levels of capacity of the health system and if the mortality is high. However if the pathogen is very contagious, the testing strategy is not enough to contain the infections, resulting in almost the same number of deaths as in the no-policy case with

a small ODR. Similarly, if the recovery rate is small there are a lot of infected at the same time and the testing strategy that identifies a fixed number of them is not enough to contain the infections, with a resulting high death rate and small ODR. Testing policies are therefore less effective in case of very contagious diseases or if the infected are slow to recover. In all cases, however, the ODR and DDR of the testing policy are above the ones of a lockdown. The problem of this testing policies, however, is that they are not always feasible (not enough capacity to test).

One caveat is that the above computations overstate the benefits of the testing policy because the tests themselves do not have any cost. This is not a problem for the ODR computation because I can assume deficit-financing. But this means that the true DDR is lower than my computations suggest. To gauge the magnitude of this difference, let's suppose that a single test costs 10 euros and, rather unrealistically, that the cost is the same regardless of how many tests are processed[24]. Yearly GDP per capita in Italy is roughly 34500 euros, so 10 euros are equivalent to 1.5% of the weekly GDP per capita. Suppose that, in order to discover the target 50% of all asymptomatics, the government needs to administer tests to 10% of the population every period. The total cost amounts to 0.15% of nominal GDP which, for 20 weeks, is 3% of weekly GDP. The median debt to gdp ratio with testing at the end of the epidemic is 2%. Thus the correction factor to have a DDR that includes the tests cost is 2/5. In the baseline simulation, the median DDR of the testing policy is 0.4074. Multiplying it by 2/5 gives 0.1629 which is still far above the DDR of the lockdown. Even assuming that a test costs 50 euros, with a correction factor of 2/17, I have a DDR of 0.047.

5.4 Robustness

In this section I analyze the robustness of the main simulation results to alternative parameter values and assumptions. The results are summarized in table 3.

In the baseline simulation, the density parameter η is calibrated in order to match the estimated average number of social contacts in Italy. However there are areas in the country where this density is arguably much higher, especially metropolitan areas, with the consequence of

[24]Piguillem and Shi (2020) assume, more realistically, that the cost of the tests grows quadratically with their number at a rate that makes it too costly to test all agents in the same period.

a different epidemic unfolding. Shutting down social contacts might be highly beneficial in this context. I simulated the model again setting $\eta = 0.022$, twice as big as in the baseline simulation. Contagions are much faster in this case and, as results, the epidemics last, on average, less with respect to the baseline. Without any policy intervention, the mortality is also higher, since more agents get the infection, and so are the output loss and debt increase. Lockdowns are much more effective in denser sociaties: the ODR and DDR are, respctively, 0.0170 and 0.0226 with zeros in just 37% of the simulations. Testing strategies, on the other hand, are less effective in this context, since isolating 50% of the asymptomatics does not decrease the contagions from social contacts sufficiently enough. A more through testing is necessary in order to do so, but that is often not feasible. In section 7 I will also discuss a model extension to heterogeneous social participation.

With an average family size of 2.5, there are a lot of contagions, in the baseline simulation, due to family contacts. Lockdown do not discontinue those contacts and are, therefore less effective. I simulated the model again assuming a higher percentage of singles (50% instead of 31%) and reducing proportionally the percentages of families with more than one member (22% with two members, 15% with three, 11% with 4 and 2% with 5). The median ODR and DDR of the lockdown are, respectively, 0.0105 and 0.0158, about twice as much as in the baseline simulation, with zeros in 40% of the cases. Testing strategies, on the other hand, are less effective with smaller families because, for the same population size, there are a lot of contagions from social contacts that are not prevented.

Lockdowns are also more effective in case of bigger firms, since they significantly reduce contagions on the job. I simulated the model halving the percentage of small firms with 1 employee and redistributing proportionally the remaining firms' shares. Given the lockdown rule that closes firms proportionally to size, the output loss in this case is not much different. However mortality is lower and, as a result, the ODR and DDR are bigger (0.0084 and 0.0114), with zeros in 37.5% of the simulations. Testing strategies are also more effective with bigger firms, since they reduce contagions from asymptomatics on the workplace with only a marginal production disruption.

The participation to the labor market is quite low in benchmark simulation, which is one of the reasons why contagions from social interaction are more frequent than contagions on

the job. With a 75% participation, there is a sharper mortality reduction due to the lockdown, with higher ODR and DDR (0.0129 and 0.0159) and zeros in 42.5% of the simulations. With such an high participation to the labor market, isolating 50% of the asymptomatics is not enough to reduce contagions on the job significantly, so testing strategies are less effective.

In the benchmark simulation, I assumed that 17% of the population has a higher death rate, either because of old age or because of other pathologies, where the 17% was set in order to match the fraction of Italians aged 70 or more. The gains from a lockdown are actually higher in case of an older population since, for the same output loss and debt increase, there is a steeper mortality decrease, resulting in higher ODR and DDR (0.0098 and 0.0132) with zeros in 41% of the simulations. With this higher average death rate, testing strategies are less effective at reducing mortality, with lower ODR and DDR with respect to benchmark.

Summarizing, lockdowns are more effective with older agents, smaller families, bigger firms, dense societies and high participation to the labor market. In all cases, however the ODR and DDR of the lockdown are below the ones of the testing policy, which is therefore preferable. Even adding a moderate and fixed cost of the tests as in section 5.3 would yield the same results, with the only exception of the DDR for dense economies with a high test cost.

6 Lockdown Design and Testing Efficacy

The lockdown rules that I used in the baseline simulation are just one of the many possibilities. The scope of this section is to analyze extensively the effects of the details of the lockdown rules on the dismal ratios. Table 4 summarizes the results. In all cases I will compare the results with the benchmark lockdown characterized by a minimum of 8 weeks, by the forced closing of 50% of the firms, by the discontinuing of 75% of the social activities and that ends when the two-weeks effective reproduction number drops below 0.5.

Lockdowns can be more or less severe. The severity can be measured either by the minimum number of weeks, by the percentage of firms closed and of social contacts discontinued, or by the stopping rule, i.e. the level of the effective reproduction number below which the economy must drop in order to come back to normal. I start exploring the consequences of a different minimum number of weeks. If these number is very high, there is the risk of having a lockdown

which is longer and more costly than needed to bring the effective reproduction number down. If it is too small, the risk is opening the economy when there is not a sufficient number of immunes, exposing the economy to the risk of a second wave. Lockdowns with a 4 weeks minimum do not last less which respect to be benchmark. The reason is that, in almost all cases, 4 weeks are not enough to bring the (2-weeks) effective reproduction number below the target 0.5. In fact the median epidemic duration, mortality, output loss and debt increase of those lockdowns is indeed very similar to the benchmark. The resulting ODR and DDR are also very close to benchmark, respectively 0.0089 [0;0.0759] and 0.0120 [0;0.1025], with zeros in 41.5% of the simulations. Lockdowns that last for a minimum of 12 weeks are instead longer, and make also the epidemic last longer, with a median duration of 42 weeks. These longer epidemics are in turn associated with a more pronounced median output loss and debt increase with respect to benchmark and a with lower death rate. The resulting ODR and DDR are higher than benchmark, respectively 0.0123 [0;0.635] and 0.0157 [0;0.0838] with zeros in 37% of the cases. Thus a shorter minimum lockdown period does not seem to be binding in most cases, while a longer one significantly increases the epidemic duration, although with gains in terms of reduced mortality and improved dismal ratios. The problem, however, is that longer lockdowns are not easy to enforce.

A more stringent stopping rule that re-opens the economy if the two-weeks effective reproduction number drops below 0.25 results in longer lockdowns and longer lasting epidemics (median duration 49 weeks), with a bigger median output loss (26%) and a bigger median debt to gdp ratio (20%). However they are very effective at containing the number of infections and at reducing the death rate down to 1.71% at the median. The resulting ODR is 0.0117 [0;0.0452] and the resulting DDR is 0.0128 [0;0.0589], with zeros in just 30% of the simulations. The additional cost of this more severe lockdown is a smaller number of immunes at the end of the epidemic (median 80%) to protect from the risk of a second wave. A less stringent stopping rule that re-opens the economy if the two-weeks effective reproduction number drops below 0.75 gives instead results in line with the benchmark, although more volatile as a consequence of the more frequent occurring of second peaks of infection due to a premature reopening. The median output loss is smaller under this less stringent stopping rule (16.2%) and the median debt increase less pronounced (9.7%), while the death rate only

slightly higher (1.89%). However in 47% of the simulation there is no actual gain in terms of reduced mortality. The conclusion is that, if feasible, it is better and safer to re-open the economy when the effective reproduction number is sufficiently low, although this means prolonging the lockdown and epidemic. Taking the reasoning to the extreme, I also explored the possibility of re-opening the economy when the two-weeks effective reproduction number drops at zero. The median epidemic duration in this case is 54 weeks, which compared to the 22 of the no-policy case and 37 of the regular lockdown seems really a lot. The median output loss is also considerably high (31%) in this case and so is the debt increase (25%). But this is the cost to pay to bring he mortality rate down, to a median of 1.46%, with only 22% of the simulation resulting in more deaths. The resulting ODR and DDR are higher, respectively 0.0221 [0;0.0593] and 0.0223 [0;0.0709]. Despite being more effective, these extreme lockdowns appear to be hardly feasible for many governments, that might find it hard to leverage the necessary resources to finance it on the market, not to mention the difficulty of enforcement and the potential adverse consequences on the individual health and well being due to a prolonged interruption of social contacts. Moreover, since a quite small number of agents get the infection in this extreme lockdown scenarios (51% median), there is a high risk of a second wave of infections with additional deaths. A longer lasting epidemic might also foster feelings of uncertainty that might translate in a consumption and investment reduction with longer lasting consequences, and the firms that remain close for such long periods might find it harder to reopen. In short, prolonging lockdowns too much it is not desirable.

The severity of the lockdown depends also on the number of firms closed and on the percentage of discontinued social contacts. A more severe lockdown that forces 75% of the firms to close, for the same number of social contacts, entails a higher median output loss with respect to the benchmark lockdown and a higher median debt increase. This lockdown is very fast at bringing the effective reproduction number below the threshold and, on average, the lockdowns last for less. The problem is however that the economy opens to soon, when there is not a sufficient number of immunes, and there is often a second peak of contagions. The result is a death rate which is actually higher than the benchmark lockdown. Thus severe lockdowns must be combined with more stringent stopping rules in order to be effective. But this will actually prolong them and, as already noted, they will be more difficult to enforce.

Viceversa, a less severe lockdown that forces 25% of the firms to close lasts on average for longer and there are second peaks less often, resulting in lower death rate with respect to the benchmark lockdown. The very same reasoning applies to mild social lockdowns versus severe social lockdowns for the same number of closed firms. At the extreme, discontinuing social contacts only has the obvious advantage of a very small cost in terms of median output loss (3%) and debt increase (1%), and entail a very small median mortality rate (1.72%). In just 37% of the cases there is no reduction in mortality and the corresponding ODR and DDR are negative. Viceversa, closing firms only results in a median death rate which is not very different from the no-policy benchmark and in 57% of the cases there is no mortality reduction. As stressed in section 5.2, In this economy there are a lot of small firms and labor force participation is low, thus social contacts are the most important source of contagions. In such conditions closing firms does not give good results. The problem is that discontinuing social contacts might have important side-effects on the individual well-being and I do not have a good framework to assess those costs.

Another crucial detail of the lockdown strategy is when to start it. I consider two alternative scenarios: the first is an earlier start, at the third week instead of the fourth. Since it takes time to collect data and to take a decision, I preferred not to consider earlier starting dates. The second scenario entails instead a late start, at the 6th week, still before the peak of the no-policy scenario in all simulations. Starting earlier gives and advantage in terms of reduced mortality, but it takes longer to bring the effective reproduction number below the threshold, resulting in longer lockdowns and longer epidemics (median duration 43 weeks) and in a more pronounced median output loss and debt increase, respectively 26% and 19%. The resulting ODR and DDR are however bigger than benchmark, respectively, 0.0090 [0;0.0451] and 0.0108 [0; 0.0619] with zeros in 40% of the cases. Thus it is better to start early. Starting late results instead in a slightly higher median mortality rate and a bigger median output loss and debt increase. However there is a smaller number of simulations with a zero gain in terms of mortality and the ODR and DDR are, as a consequence, in line with the benchmark. Thus starting late is not a big issues, conditionally on doing so before the infection peak.

The last two rows of table refdismshut summarize instead the results of two alternative testing strategies who are able to isolate, respectively, 25% and 75% of the symptomatics.

Isolating fewer asymptomatics results in a higher peak of infected and in a higher median death rate (1.95%), but still smaller than the no-policy alternative. The median output loss is however slightly smaller than the baseline testing strategy, 4.4%, However in 55% of the cases there is no actual gain in terms of less deaths. Thus the testing strategy, to be effective, must be able to isolate a sufficient number of asymptomatics. If it is feasible to test a large number of agents and correctly identify 75% of the asymptomatics, there is a sharp decrease in the median death rate (1.14%). The median output loss is actually lower with respect to the benchmark testing (3.9%) because there are fewer agents that get the infection and, thus, fewer asymptomatics that do not go to work, as well as less deaths. The result is a very high ODR, 0.5617 and a very high DDR, 0.8903 and in just 5% of the cases the morality rate is higher than the no-policy scenario. The problem is that isolating such a large fraction of the population of asymptomatic is a daunting task that most government cannot complete. But these results are indeed useful to show the potential of such policies.

Favero, Ichino and Rustichini (2020) proposed to let younger workers come back to work as an exit-strategy for the post Covid-19 lockdown in Italy. The rationale is that younger individuals are less susceptible of developing severe symptoms and dying and, therefore, having them back to work will not increase the death rate while allowing the output gap to close. Following their lead, I considered an alternative approach to the lockdown that order only high-risk workers, with higher death probability upon infection, to stay home. Since I have a small fraction of high risk individuals in since some of them do not participate to the labor force, I end up with an outcome that is not very different from the benchmark no-policy scenario. In fact in 50% of the cases there is no reduction in the mortality rate. This alternative approach to the lockdown yields better results in case of many high risk workers.

In conclusion, the simulation results show that testing strategies, if feasible, are the best way to cope with an epidemic. However they must be able to isolate a sufficient number of asymptomatics to be effective. Lockdowns are the second best alternative, and it is best to prolong them until the effective reproduction number is sufficiently low, even if this means a longer epidemic. If the average firm size is small, there are not much gains in severe lockdowns that close many economic activities. Social lockdowns only might actually reach the same results at a lower cost in terms of output and debt, although perhaps a higher individual cost.

7 Extensions

In this section I summarize the results from several model extensions. In subsection 7.1 I assess the risk and consequences of second waves of infections, simply shocking the economy again after the first wave of the epidemic ends. In subsection 7.2 I assume that underage, non-working agents go schools, with additional contagion opportunities. In subsection 7.3 I extend the framework to two countries and analyze the effects of closing borders. Subsection 7.4 explores instead the consequence of heterogenous participation to social activities. Subsection 7.5 studies instead an alternative policy approach that reduces the contagion probability.

7.1 2nd Waves?

All policies that reduce the number of infected reduce also the number of immunes at the end of the epidemic. A small number of immunes, in turn, opens up to the possibility of a second wave of contagions, triggered for instance by another agent that contracts the pathogen abroad during a trip, and the resulting in additional deaths and output loss that must be acknowledged in order to properly evaluate the effects of the policies. However it is also possible that a treatment or vaccine is developed before the second pathogen shock hits, which is in fact one of the reasons why taking time with a lockdown might be a good idea. Suppose however that this is not the case and, therefore, that second waves are a potential threat. To evaluate the vulnerability to a second wave of contagions, I performed the following exercise: for all simulation run and conditional on the three baseline policies, I infected two randomly chosen agents who, at the end of the epidemic, were still susceptible. I repeated this random infections 100 times and counted the percentage of times that the two new randomly infected triggered a second round of infections, where the second round is defined as a number of infected bigger than 6 (three times the stocks of new infected) 4 periods after the new infections. Over 100 runs of the baseline simulation, I median percentage of second waves is 0 for all policies. In fact only for two simulation runs there are 2% of the second run infections that trigger a second wave in the lockdown case. Thus, regardless of the implemented policy, there is essentially no risk of a second wave in the baseline simulations. In case of a more severe lockdown that stops when the two-weeks effective reproduction number drops below

0.1, the median percentage of second waves is also zero for all policies. The only difference is that for 17 simulation rounds there are 4% of the second run infections that trigger a second round in the lockdown case. Thus the risk is extremely small also in case of a severe lockdown.

7.2 Schools

In the baseline simulation I did not model schools explicitly: underage agents are treated like pensioners and unemployed, who can only get the pathogen from family or social interactions. Putting it differently, schools bunched with other social activities, with the consequent impossibility to evaluate separately the effect of a lockdown that closes schools. I extended the model assuming that a fixed fraction of the agents who do not work and who live in families of three or more go to daycare, to school or attend a university. For simplicity I abstract from single and divorced parents[25]. This fraction is in turn calibrated to match the percentage of the Italian total population that attends daycare, schools and universities. According to ISTAT data, in Italy 38.5% of the agents aged between 19 and 25 attend a university and 25% of the kids aged less than 3 go to daycare. Assuming that all kids aged between 3 and 18 attend a school, I obtain a total fraction of 18% of the population in daycares, schools and university. The average school size in Italy according to ISTAT is 223 kids or 2.2% of all school age kids. I set the number of schools in the model in order to have the same percentage. Then I also re-calibrate the parameter η to have the average target number of contacts per individual, including schools, of 18.5. To simulate the model with schools, I assume that symptomatics and quarantined asymptomatics do not attend and considered schools as an additional source of potential contagions together with families, workplaces and social activities. A lockdown in this economy entails, in addition to the baseline lockdown, the closing of all schools with a switch to e-learning platforms. The results are almost identical to the benchmark simulation. A lockdown that closes schools only does not impact on production but, given the small average school size, does not significantly slow down contagions and, thus, does not decrease the death rate significantly. The median ODR and DDR of such policies are zeros and in more

[25]Alternatively, single parents are assumed to live with one or two grandparents or with another partner. In case of divorced parents, the assumption is that the two divorced parents and the kid make up, in the model, for a single family unit, which is reasonable since, in most cases, both parents are allowed to see the children, albeit with restrictions.

than 50% of the simulations there is no actual gain in terms of reduced mortality. However in those simulations where the mortality rate is lower, there are actually several negative ODR and DDR, since the output gap is often above the no-policy level due to a lower number of infected. Putting it differently, schools do not produce anything in the short run, so closing them yields a costless mortality reduction. This is the reason why they have been the first to close in many countries, including Italy, Spain and France, in response to the Covid-19 outbreak, even before the full lockdowns. The problem, however is that, although the gdp cost of closing them is essentially zero in the short-run, it is non-zero in the long-run if distance learning yields worse results than traditional learning, for instance because of a productivity reduction of the new workers on the job market.

7.3 Two Countries

In the baseline simulation I considered a closed economy whose residents do not have any contacts with foreigners. However epidemics are often imported from abroad, which is in fact the main reason why I justify the pathogen shocks as originated by some agents who come back infected from a trip abroad. To evaluate the importance of external links for the dynamics of the epidemic and for the effects of alternative policies, I extended the model to a two-countries framework. More specifically, I assumed that the social activities encompass also matches with members of a foreign economy, for instance in the form of trips, which are therefore an additional source of contagions. I assume that both symptomatics and quarantined asymptomatics do not engage in this activities, for instance because they are not allowed to leave the country. A lockdown, in these economies, entails also closing the national borders, resulting in a complete discontinuing of social interactions with foreigners. For simplicity, I assumed the two economies are identical. Contagions from contacts with foreigners will be more frequent in more integrated economies and closing the borders will be more effective in those cases. I modeled the integration between the two economies as dependent by a density parameter similar to η, equal to the percentage of the foreign population with whom each resident is matched every period. Then I simulated this two-countries model assuming very integrated economies, with this density parameter equal to $\eta/2$. The simulation works as follows: the initial pathogen shock hits the foreign country and it is then imported in the home

country via social contacts. Four weeks after the beginning of the epidemic in the foreign country, both the home and the foreign country implement a policy, which is the same for both. This is actually an early start for the home country with respect to be baseline simulation, but the Taiwan example at the beginning of 2020 in response to the Covid-19 outbreak in China shows that it is not an unreasonable assumption. With this additional source of contagions, absent a re-calibration of η, there is a smaller median duration of the epidemic and a higher median percentage of infected at peak for all three policies and in both countries. The death rate is also higher in all scenarios and so is the output loss. However lockdowns are more effective since they shutdown an additional source of contagions. The resulting ODR and DDR in the home country are, respectively 0.0111 and 0.0168, with zeros in 43% of the simulations. testing strategies with open borders are instead less effective with respect to the benchmark simulation. Closing borders while keeping social economic activities unchanged has the advantage of reducing contagions without any output costs, and result, in the home country, in a negative median ODR. The conclusion is that closing borders must be a priority. This two identical countries extension can be also further generalized to more countries and further calibrated to different values. Moreover, it can be also calibrated to several regions in order to evaluate the opportunity of closing only a subset if the regional borders. But such exercises are beyond the scope of this work.

7.4 Heterogenous Participation to Social Activities

In the baseline model, all agents engage in the same number of social activities while, in reality, there are individuals who are more social and, that, regularly meeting more people, might accelerate the diffusion of the pathogen if infected and asymptomatic. I extended the model to account for heterogeneous participation to social activities relaxing the assumption of $\eta_{i,t} = \eta \quad \forall\{i,t\}$. More specifically, while still keeping the time invariance assumptions, so that social activities are still exogenous with respect to the progression of the epidemic, I assume a Gamma distribution for the social contacts and I calibrate the two parameters in order to match the mean and variance of the distribution of social contacts by age groups used by the ISS to simulate the Covid-19 progression in Italy, respectively 18.5 and 26. The results are almost indistinguishable from the baseline. Maybe a selective quarantine for those

highly-social individuals, regardless of their infections status, will yield good results in terms of infection containment at a small output cost, but it would not be legal in most countries.

7.5 Reduced Contagion Probability

An additional policy approach to the epidemic entails recommending or imposing good practices that reduce the contagion probability. For instance, wearing masks and washing hands for respiratory transmittable pathogens or wearing condoms for sexually transmittable diseases. In the models by Piguillem and Shi (2020), Farboodi, Jarosch and Shimer (2020) and Collard et al. (2020), this is actually what rational agents do regardless of what the government imposes. A policy that, after the fourth week of the epidemic, is able to reduce the contagion probability from 10% to 8% for both symptomatics and asymptomatics delivers negative median ODR and DDR, but which are zero in 43% of the cases. Reducing these contagions probabilities to 5% results instead in negative median ODR and DDR with zeros in 16% of the simulations. Good practices that reduce the contagion probability should be a priority.

8 Conclusion

Coping with an epidemic asks for difficult calls. Social distancing slows down the epidemic and can save lives, but at the cost of discontinuing social and economic activities, with an increase of public debt in case of income support for the unemployed and for the entrepreneurs whose businesses are closed. My simulation results show that lockdowns do indeed save lives on average, although not always, but at a high cost for the current and future generations. In case of asymptomatic infections, testing strategies, if feasible, yield better result, as they have the potential to reduce the mortality of the epidemic without disrupting economic activities. But, in order to work properly, they must involve a through testing of the population which is not always feasible.

The simple model that I proposed is very flexible and with few, more realistic, extensions can be fruitfully used to study the importance of several aspect from which, in this work, I abstract. One of those is heterogenoeus workers productivity, which will drive the output

response to the epidemic. If the high-productivity workers are hit first, there will be a deep production drop at early stages of the epidemic and, if the lockdown is implemented when some of the high productivity workers already recovered, it will have a lower cost. Selective lockdowns that force home the low-productivity workers will be obviously less costly, but, as in the case of the highly-social individuals just discussed, they will most likely be illegal.

A further overlooked aspect in my analysis is the output effect of discontinuing social activities. In my framework workplaces are more akin to manufacturing plants than restaurants or bars. If meeting friends is forbidden as part of a lockdown, there will also be less demand for the latter independently from the fact that they are open, with an impact on the GDP. Sweden after the Covid-19 outbreak is a good example in this sense. The consequence is that my model overstates the output cost of a lockdown. More generally, if people respond to the epidemic by taking actions to protect themselves independently from government intervention, as in Piguillem and Shi (2020), it is possible to have a significant output loss even without a social lockdown. On top of that, the uncertainty on the future that characterize the earlier stages of diffusion of the pathogen might also result in a consumption and investment decrease that might reduce GDP as in Eichenbaum, Rebelo and Trabandt (2020). In order to properly evaluate the importance of all these effects, it is necessary to build a more detailed and realistic model than mine.

As highlighted throughout the paper, lockdowns involve a delicate balance of interests, first and foremost regarding who will bear the current and future cost the extra expenditures that are necessary to make it bearable and acceptable. As such, they involve complicated negotiations among partisan and/or opportunistic politicians, who will obviously very cautious when implementing a recessionary policy, perhaps also resulting in a war-of-attrition type of situation. However there is actually an important political advantage associated with lockdowns, which is also among the reasons why they have been so popular as a response to the Covid-19 pandemic: no elected politician wants to be blamed for the death of his citizens. In other words, if there are many deaths even with a lockdown in place, there is always the possibility to claim that there could have been many more without; in case of few deaths, conversely, it is quite easy to claim a success. A win-win choice. The Italian case is actually a good example of a government that gained consensus during the Covid-19 pandemic, while

the widespread criticism to the essentially non-interventionist strategy of the Swedish and Brazilian governments against the Covid-19 shows the opposite side. A complete comparative evaluation of alternative policies cannot abstract from the their political convenience. I live such an analysis to future work.

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Table 2: **Policy Comparison**

	No policy				**Lockdown**				**Testing**			
	med	std	95% conf int		med	std	95% conf int		med	std	95% conf int	
Duration	22	2.077	20	27	37	4.734	32	47	28	3.939	23	35
Infpeak	41.792	1.759	38.916	44.498	13.854	3.131	10.041	19.506	21.283	2.456	17.590	25.513
Deaths	1.954	0.389	1.382	2.683	1.841	0.398	1.221	2.469	1.709	0.380	1.134	2.369
Immunes	93.301	0.827	91.667	94.490	85.322	2.379	80.991	88.525	78.457	2.245	74.424	81.811
YLoss	3.486	0.464	2.712	4.287	22.156	3.521	17.414	28.729	4.700	0.577	3.837	5.651
Debt	1.497	0.165	1.223	1.769	15.308	3.679	10.724	22.538	2.098	0.244	1.708	2.504
Inflation	2.115	0.542	1.203	3.087	7.794	2.821	3.841	13.049	1.873	0.500	1.187	2.714

Notes: Duration is the length of the epidemic defined as the first period with zero infected. Infpeak is the maximum percentage of infected agents (sum of symptomatics and asymptomatics) in a given period during the epidemic. Deaths is the total number of deaths at the end of the epidemic as a percentage of the pre-epidemic population size. Immunes is the total number of immunes at the end of the epidemic as a percentage of the pre-epidemic population size. YLoss is the cumulative output loss over the epidemic as a percentage of potential GDP in the same time span. Debt is the Debt to GDP ratio at the end of the epidemic. Inflation is the percentage difference between the price at the end of the epidemic and the price at the beginning. Med stands for median over all simulation runs. std stands for standard deviation over all simulation runs. 95% conf int is the 95% confidence interval over all simulation run (5% of the simulations resulted in a lower value than the lower bound and 5% in a bigger value than the upper bound).

Table 3: **Policy Comparison, Robustness**

	Policy	Duration	Deaths	YLoss	Debt	ODR	DDR	No Gain
Baseline	No Policy	22	1.9544	3.4857	1.4972			
	Lockdown	37	1.8410	22.1561	15.3078	0.0049	0.0067	45.7
	Testing	28	1.7087	4.7005	2.0975	0.2112	0.4074	34.0
Highdens	No Policy	19	2.1053	4.2025	1.8604			
	Lockdown	30	1.8791	20.0272	13.0270	0.0170	0.0226	36.8
	Testing	20	1.9721	6.3152	2.9321	0.0920	0.1594	37.9
Smallfam	No Policy	21	2.0257	3.7689	1.6688			
	Lockdown	34	1.8798	20.9140	13.8709	0.0105	0.0158	39.8
	Testing	25	1.8206	5.5130	2.5358	0.0847	0.1607	39.8
Bigfirms	No Policy	23	1.9624	3.3850	1.4360			
	Lockdown	40	1.8669	22.3905	15.9534	0.0084	0.0114	37.5
	Testing	30	1.5523	4.3092	1.8998	0.4824	0.9757	22.5
Lab75	No Policy	21	2.0375	3.7024	1.4600			
	Lockdown	34	1.7843	21.1242	14.3892	0.0129	0.0159	42.3
	Testing	25	1.7915	5.1972	2.3338	0.1061	0.2387	35.1
Older	No Policy	22	2.1259	3.5775	1.5112			
	Lockdown	38	1.8791	22.2619	14.9563	0.0098	0.0132	40.7
	testing	28	1.8730	4.9107	2.0942	0.1102	0.3591	39.5

Notes: Duration is the length of the epidemic defined as the first period with zero infected. Deaths is the total number of deaths at the end of the epidemic as a percentage of the pre-epidemic population size. YLoss is the cumulative output loss over the epidemic as a percentage of potential GDP in the same time span. Debt is the Debt to GDP ratio at the end of the epidemic. ODR is the Output Dismal Ratio defined in equation 23. DDR is the Debt Dismal Ratio defined in equation 24. The table reports the median outcomes over all simulation runs conditional on three policy approaches, no policy, lockdown and testing, for different scenarios (first column). No gain pct is the percentage of simulation runs with zero ODR and DDR, so with lockdowns or testing strategies resulting in a higher death rate with respect to the no-policy alternative. The Scenarios are the following: Baseline is the benchmark simulation; Highdens entails doubling the density η; Smallfam entails a 50% share of singles; Bigfirms entail halving the share of firms with 1 employee; Lab75 entails $W = 0.75$ or 75% labor force participation; Older entails doubling fraction of the population with higher death rate upon infection.

Table 4: **Lockdown Design and Testing Efficacy**

	Duration	Mortality	YLoss	Debt	ODR	DDR	No Gain
No policy	22	1.9544	3.4857	1.4972			
Lockdown, bench	37	1.8410	22.1561	15.3078	0.0049	0.0067	45.7
Lockdown, min 4 weeks	39	1.8062	21.4228	14.6039	0.0089	0.0120	41.5
Lockdown, min 12 weeks	42	1.7945	23.2124	16.5509	0.0123	0.0157	37.2
Lockdown, thresh 0.75	33	1.8914	16.2127	9.7426	0.0065	0.0091	46.7
Lockdown, thresh 0.25	49	1.7087	26.4205	19.9464	0.0117	0.0128	30.7
Lockdown, thresh 0	54	1.4646	31.4121	25.7163	0.0221	0.0223	22.3
Lockdown, firms mild	37	1.7129	13.2070	7.4737	0.0339	0.0549	37.6
Lockdown, firms severe	37	1.9704	29.2497	22.5149	0.0000	0.0000	54.1
Lockdown, social mild	32	1.8745	19.6246	12.6348	0.0088	0.0119	46.1
Lockdown, social severe	36	1.8868	21.6179	14.7671	0.0000	0.0000	50.6
Lockdown, firms only	23	2.0458	18.0390	10.9092	0.0000	0.0000	57.7
Lockdown, social only	35	1.7213	3.0543	1.1134	-0.3859	-0.4073	37.1
Lockdown, early	43	1.7974	26.0673	19.4113	0.0090	0.0108	40.5
Lockdown, late	45	1.8714	25.0393	18.1755	0.0063	0.0077	39.3
Testing, bench	28	1.7087	4.7005	2.0975	0.2112	0.4074	34.1
Testing, 0.25	23	1.8946	4.4000	1.9900	0.0000	0.0000	55.6
Testing, 0.75	37	1.1410	3.9357	1.6076	0.5617	0.8903	4.9

Notes: Duration is the length of the epidemic defined as the first period with zero infected. Deaths is the total number of deaths at the end of the epidemic as a percentage of the pre-epidemic population size. YLoss is the cumulative output loss over the epidemic as a percentage of potential GDP in the same time span. Debt is the Debt to GDP ratio at the end of the epidemic. ODR is the Output Dismal Ratio defined in equation 23. DDR is the Debt Dismal Ratio defined in equation 24. The table reports the median outcomes over all simulation runs conditional on policy (first column). No gain pct is the percentage of simulation runs with zero ODR and DDR, so with lockdowns or testing strategies resulting in a higher death rate with respect to the no-policy alternative. Policies: No policy means no policy intervention; Lockdown, bench is the baseline lockdown with 8 weeks minimum requirement, 50% of firms closed, 75% of social activities discontinued and end when the two-weeks effective reproduction number drops below 0.5; Lockdown, 4 weeks and Lockdown, 12 weeks are lockdowns with 4 and 12 weeks minimum requirement; Lockdown, thresh 0.75, Lockdown, thresh 0.25 and Lockdown, thresh 0 are lockdowns that end when the two-weeks effective reproduction number drops below 0.75, 0.25 or at 0. is a lockdown that ends when the two-weeks effective reproduction number drops below 0.25; Lockdown, firms mild and Lockdown, firms severe are lockdowns that close 25% and 75% of the firms; Lockdown, social mild and Lockdown, social severe are lockdowns that discontinue 50% and 87.5% of the social activities; Lockdown, firms only is a lockdown that does not discontinue social activities; Lockdown, social only is a lockdown that does not close firms; Lockdown, early and Lockdaown, late start the third and sixth week after the pathogen shock; Testing, bench, Testing, 0.25 and Testing, 0.75 are testing strategies that are able to isolate and quarantine 50%, 25% and 75% of the asymptomatics every period;