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Pandemics, public policy, and Peltzman effects

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Abstract

Against the backdrop of Covid-19, we study the effectiveness of public policies typically employed to fight an epidemic. We extend the compartmental SIR model to explore the trade-offs which govern individual behavior. Our stylized model allows for a closed form analysis of vaccination and lockdown policies. We establish Peltzman effects: as policies lower the risk of infections, people become more socially active, which—in turn—undermines their effectiveness. We detect patterns in data for both US states and countries in Western Europe that are consistent with the notion that such effects are shaping actual infection dynamics to a considerable extent.

Keywords Pandemic · Behavioral adjustment · SIR model · Risk compensation · Lockdowns · Vaccinations

JEL Classification I12 · I18

1 Introduction

The Covid-19 pandemic spread across the globe in several waves during the years 2020–2022. Public policy responded quickly, first by imposing a series of lockdowns, later via a massive vaccination effort. The effect of these policies may be counteracted by a behavioral adjustment to their risk-reducing effect: People naturally expand their economic and social activity if they feel better protected, which—in and of itself—raises the number of new infections and thus the number of deaths. This type of

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adjustment has first been established in the context of safety regulation (Peltzman 1975). However, while such “Peltzman effects” were implicit in a series of numerical simulations presented during the pandemic, we still lack a clear comparative static analysis of vaccination and lockdown policies that focuses on these effects. In this paper, we aim to fill this void.

We develop a very stylized version of the Susceptible-Infected-Recovered (SIR) model, as frequently employed to study infection dynamics, allowing for (rational) adjustment of behavior (social activity) in response to both the state of the pandemic and the policy measures. We derive a number of results. First, the effect of vaccinations on infections and deaths is ambiguous. As vaccinations lower the risk of infection and death, individuals increase the level of social activity, which gives rise to more infections and deaths. We show that this offsets the direct effect of vaccinations if the degree of risk aversion is sufficiently low, potentially even making vaccination a welfare reducing policy—if implemented without any restrictions on social mobility, such as a lockdown policy.

Second, we demonstrate that this type of behavioral adjustment also arises for a lockdown policy uniformly imposed on a heterogeneous population where some individuals are more vulnerable to the disease than others. Being aware of their vulnerability, once the pandemic arrives more vulnerable individuals exercise a higher level of self-restraint. Consequently, a uniform lockdown is likely to constrain the behavior of less vulnerable individuals, thus lowering the infection risk for all, but may well be non-binding for the more vulnerable ones. If so, they have room to adjust and will increase their social activity in response to the lower risk. This unambiguously implies a higher case fatality and may even increase mortality. If the potential of a welfare reduction through vaccination may be avoided through an optimal lockdown policy, the potential of detrimental Peltzman effects in lockdown policies is in turn reduced by vaccination as this lowers the degree of heterogeneity in terms of vulnerability. The two policies should therefore not be seen as orthogonal efforts to fight a pandemic.

Finally, we present evidence on infection dynamics based on monthly observations for US states and countries of the European Union (EU), respectively. We find that both, current infections and deaths, are not much correlated with vaccination rates in previous months, consistent with the notion that behavioral adjustment largely offsets the direct impact of vaccinations. Turning to lockdowns, we distinguish between an early period prior to the availability of vaccinations and a later period when vaccinations had become available. Since vaccinations lower the degree of heterogeneity, which is key to the Peltzman effect in the lockdown policy, we must expect the lockdown stringency to be more effective in avoiding deaths during the second period. Our empirical results support this.

Relation to the literature. We share the focus of a large literature in economics which complements epidemiological studies of the Covid-19 pandemic by bringing to the fore people’s rational response to a changing environment, triggered by policy measures or otherwise. Recent contributions include Bobkova et al. (2024), d’Albis et al. (2024) and Fabbri et al. (2024). Rather than discussing the literature in detail, we refer readers to the recent survey by Bloom et al. (2022). Importantly, this literature largely relies on numerical simulations of calibrated models which makes it hard to

identify Peltzman effects. Instead, to pin down these effects we study what we consider the simplest possible economic extension of the SIR model, building on Farboodi et al. (2021) but providing closed-form analysis instead of simulation results. Perhaps the most important simplification that we make is to assume discrete time with three periods: a pre-policy period during which the epidemic emerges, the policy period at the beginning of which the policy is implemented and its effect unfolds, and a post-pandemic period. While admittedly stark, this simplification serves to highlight the intertemporal trade-off that decision makers, both individuals and the social planner, are confronted with, and it allows us to derive sharp theoretical predictions with clear intuition.

To be sure, we are not the first to offer analytical treatments of policies like vaccination and social distancing. Kremer (1996) and Geoffard and Philipson (1996) were the first to incorporate behavioral adjustment in their analysis of HIV-infections, and we compare our results to those of Kremer (1996) in somewhat more detail below. Toxvaerd (2019) uses a continuous-time framework to study the effect of a policy that permanently reduces the transmission parameter in the infection dynamics. Carnehl et al. (2021) use a similar model to examine how the peak of prevalence (number of infected persons) varies with the transmission parameter. He shows that in extreme cases a lower transmission parameter may even raise the peak prevalence during the pandemic as well as steepening (rather than flattening) “the curve” in early phases. However, a policy reducing the transmission parameter is different from rolling out vaccinations since it ignores the medical effect of vaccination (reducing the health hazard from the infection). Our analysis shows that this makes a big difference although the possibility of a welfare reduction still obtains.

A key element of our analysis is the heterogeneity among individuals in terms of vulnerability. The need to take account of this feature of pandemics is widely acknowledged. Toxvaerd (2019) briefly considers heterogeneity in terms of the infection risk but does not offer a full-fledged policy analysis for this case as we do for the lockdown-policy. Moreover, some of the simulation studies do allow for such heterogeneity (Brotherhood et al. 2021; Acemoglu et al. 2021; Makris 2024) but do not focus on behavioral implications for policy. In this paper, we show that imposing a uniform lockdown on a heterogeneous population is likely to cause a specific Peltzman-type effect that has so far not been acknowledged in the literature or the policy debate.

2 A simple framework

In this section, we set the stage by introducing a simple model in which self-interested individuals adjust to the pandemic without being forced to do so by government policy. The pandemic is described using the well-known SIR-model, which we enrich to account for social activity. For the sake of analytical tractability, we keep the model radically simple.

We start from a discrete-time version of the SIR-model, due to Kermack and McKendrick (1927). Using I_t and S_t to denote the stock of infectious and susceptible

persons, respectively, at the beginning of period t , the change in I_t is

$$\Delta I_t = \beta \frac{S_t I_t}{N_t} - \mu I_t - \rho I_t. \quad (1)$$

In this equation, N_t is the size of the population at the beginning of period t , and β reflects the average number of contacts per person during the period as well as the ease of virus transmission through these contacts. We henceforth call this the *transmission parameter*. A fraction ρ of those infected recovers and a fraction μ dies, whence the stock of the deceased changes according to μI_t . We assume that the parameters β , μ as well as ρ are time-invariant.¹ Assuming that the recovered, denoted by R , are no longer susceptible, we have $N_t = I_t + R_t + S_t$. We normalize $N = 1$ so that S and I are shares of the population.

We stipulate two modifications of the SIR-model. First, we collapse the entire pandemic into three periods: Period 0 is the initial period of the pandemic in which no policy intervention takes place. Period 1 is the *policy period* during which the pandemic evolves in line with Eq. (1), augmented by policy variables to be introduced below. And finally, period 2 refers to the *entire time after the policy period*. Note that the three periods need not be of equal length.

The second modification is to introduce social activity which generates utility but also influences virus transmission. We interpret the transmission parameter β as holding for a unit level of social activity, and—following Farboodi et al. (2021)—we multiply the stocks of susceptibles and infectious individuals by the respective average activity levels during the policy period, denoted by A_S and A_I . Implicitly, we assume that all individuals infected at the beginning of period 1 either die or recover during the period. With these modifications, Eq. (1) changes to

$$\Delta I = \beta A_S S A_I I, \quad (2)$$

where the period index has been dropped to simplify notation. As in Farboodi et al. (2021), we assume that individuals do not know their health status (susceptible or infectious), hence we may simplify by writing $A_I = A_S = A$.

We assume that individual wellbeing may be described by a periodic utility function $u(a)$, where a denotes the individual's level of social activity. This stands for production as well as consumption activities.² Marginal utility is positive but diminishing, $u' > 0$ and $u'' < 0$. Arguably, the wellbeing that an individual draws from an activity level a will also depend on other people's social activity. In the working paper version of this paper (Dietrich et al. 2023), we therefore stipulate a utility function $u(a, A)$, assuming that $u_A(\cdot) > 0$ as well as $u_{AA}(\cdot) < 0$ and $u_{aA}(\cdot) \geq 0$ (subscripts indicate

¹ Note, however, that all of these parameters depend on the length of the period.

² In an open economy, a may also involve cross-border contacts related to trade. Antràs et al. (2023) demonstrate that the SIR dynamics described above then involves a cross-border “epidemiological externality”. Moreover, if infections lead to a change in labor supply, then the dynamics of the pandemic will also lead to terms of trade effects.

partial derivatives).³ An atomistic individual will ignore the benefit that an increase in a will have on other people's (marginal) utility. This gives rise to an externality which, in and of itself, implies too little social activity taking place in a laissez-faire equilibrium. In what follows, however, we work with the simpler utility function $u(a)$ so as to squarely focus on the so-called infection externality. This externality arises because when deciding about a , an individual will naturally be concerned about the risk of becoming infected but will typically ignore what variations in a imply for the risk of others becoming infected.

To formalize this notion, we use $\delta(a)$ to indicate the probability of an individual dying in period 1, depending on the activity level a during that period. Assuming individuals know about Eq. (2), their perceived likelihood of becoming infected during period 1, *conditional* on being susceptible, is equal to $\beta a A I$. Note that atomistic individuals treat the average level of activity A as a constant when deciding about a . The term $A I$ thus captures the risk emanating from those who are infectious. By the law of large numbers, the probability of an individual being susceptible is equal to S , which we assume is known to the individual. Using $\delta_I > 0$ to measure the *excess* probability of dying from the disease, the perceived marginal effect of the individual's activity a on the likelihood of dying is

$$\delta'(a) = \delta_I \beta S A I. \quad (3)$$

This is the individual perspective. Taking the economy-wide perspective, the marginal effect of A on the probability of dying during period 1 is equal to $\delta_I \beta S I 2 A$. Compared with Eq. (3), the only difference is that A now stands for the activity level of both, susceptible and infectious individuals, which is why it gets multiplied by a factor of 2. This takes into account that an increase in A works through both, the group of susceptibles as well as the infectious, while Eq. (3) assumes that an individual is only concerned about itself becoming infected if susceptible, but not about infecting others if infectious. This is the infection externality.

Individuals decide about their activity levels at the beginning of period 1 so as to maximize expected lifetime utility. We assume that period-2 utility, as expected at the beginning of period-1, is affected by the period-1 activity level only through the probability that the individual survives to period 2. Thus an individual alive at the beginning of period 1 solves the following maximization problem:

$$\max_a u(a) + [1 - \delta(a)]\bar{u}_2, \quad \text{s.t. } \delta'(a) = \delta_I \beta S A I \quad (4)$$

where \bar{u}_2 is period-2 utility, conditional on surviving period 1. For simplicity, we abstract from discounting and normalize utility such that $\bar{u}_2 = 1$. Individuals thus face an intertemporal trade off: A higher activity level increases their current wellbeing but lowers their probability of surviving period 1, because the activity level determines the risk of becoming infected and thus the likelihood of dying from an infection.

³ This latter assumption reflects a behavioral feature emphasized in pandemic-related analysis by McAdams et al. (2023), namely that the “(marginal) benefit of activity increases as others are more active.” Our working paper assumes the degree of complementarity to be weak, so that $u_{aa}(A, A) + u_{aA}(A, A) < 0$.

The *individually* optimal level of activity, denoted by a^* , satisfies the first order condition

$$u'(a^*) = \delta_I \beta S A I. \quad (5)$$

The second order condition is satisfied from $u'' < 0$. Given homogeneity of agents, the laissez-faire equilibrium is determined by this condition, with $a^* = A^*$. Under the assumptions made about the utility function, such an equilibrium exists.

The *social* optimum is determined by the following maximization problem:

$$\max_A u(A) + 1 - \delta(A) \quad \text{s.t.} \quad \delta'(A) = \delta_I 2\beta S A I. \quad (6)$$

The first order condition for the socially optimal activity level, denoted by \tilde{A} , is:

$$u'(\tilde{A}) = \delta_I 2\beta \tilde{A} S I. \quad (7)$$

Comparing this with Condition (5) above, we recognize that the laissez-faire equilibrium has too much social activity, $A^* > \tilde{A}$, which is intuitive given the infection externality described above.

3 Vaccination

In this section, we explore the effect of vaccination on the laissez-faire equilibrium. For simplicity, we assume that the entire population is vaccinated at the beginning of the policy period. Decisions are again made at the beginning of period one, but after the policy is implemented. Vaccination has two *direct* effects. First, it provides medical protection in lowering δ_I , the excess death probability caused by an infection. And secondly, it provides epidemiological protection in lowering the likelihood of susceptible individuals becoming infected as well as the likelihood of infectious individuals to transmit the virus, thus lowering β . We assume both effects of vaccination to be common knowledge. Moreover, it is plausible to assume that the medical protection effect does not apply to individuals who were infected already prior to the start of the vaccination program.

We define $\sigma := -[u''(A^*)/u'(A^*)]A^* > 0$, which we refer to as the coefficient of relative risk aversion. Using $a^* = A^*$ in (5) and writing the equation in log-changes, we obtain $-\sigma \Delta_V \log A^* = \Delta \log \delta_I + \Delta \log \beta + \Delta_V \log A^*$, where Δ_V indicates a vaccination-induced change from the initial equilibrium. This implies

$$\Delta_V \log A^* = -\eta (\Delta \log \delta_I + \Delta \log \beta) > 0. \quad (8)$$

In this equation, we define $\eta := 1/(1 + \sigma)$, a parameter which lies between zero and unity, with a lower value of η indicating a higher degree of risk aversion. The inequality in Eq. (8) conveys a simple but important insight: For a finite degree of risk aversion, vaccination will generally raise the level of social activity, for the simple

reason that individuals now perceive a lower risk associated with this social activity. We refer to this as the Peltzman (1975) effect.

The Peltzman effect has repercussions for the dynamics of the pandemic. The level of A affects the evolution of infections according to Eq. (2), which we may rewrite as $\Delta I = \beta(A^*)^2 SI$. Taking logs and differentiating, we obtain $\log \Delta I = \Delta \log \beta + 2\Delta \log A^*$. This suggests that the effect of vaccination on new infections is ambiguous. Epidemiological protection, $\Delta \log \beta < 0$, clearly lowers the number of new infections. But then there is the behavioral adjustment $\Delta_V \log A^* > 0$, driven by both the epidemiological and the medical protection, which raises the number of new infections. Substituting from Eq. (8), we obtain

$$\Delta_V \log \Delta I = (1 - 2\eta)\Delta \log \beta - 2\eta\Delta \log \delta_I. \quad (9)$$

The same logic applies to the number of deaths D , which evolves according to $\Delta D = \delta_I \Delta I$, or $\log \Delta D = \log \delta_I + \log \Delta I$. Substituting from above, vaccination affects the infection-induced number of new deaths according to

$$\Delta_V \log \Delta D = (1 - 2\eta)(\Delta \log \beta + \Delta \log \delta_I). \quad (10)$$

Proposition 1 (The effect of vaccination on new infections and deaths)

- a) Vaccination reduces the deaths caused by the pandemic if and only if $\sigma > 1$.
- b) The necessary and sufficient condition for vaccination to reduce the number of new infections is $\sigma > 1 + 2 \frac{\Delta \log \delta_I}{\Delta \log \beta}$.

Proof Part a) follows immediately from Eq. (10), given that $\Delta \log \beta < 0$ as well as $\Delta \log \delta_I < 0$. In turn, the condition for part b) is $\frac{1-2\eta}{2\eta} > \frac{\Delta \log \delta_I}{\Delta \log \beta} > 0$, which may be written as $\eta \left(1 + \frac{\Delta \log \delta_I}{\Delta \log \beta}\right) < 1/2$, which implies $\sigma > 1 + 2 \frac{\Delta \log \delta_I}{\Delta \log \beta}$. \square

The intuition for this proposition is straightforward. The strength of the Peltzman effect is higher with a lower degree of risk aversion σ . For high enough values of risk aversion, $\sigma > 1$, the effect is not strong enough to make vaccination ineffective regarding mortality (part a). However, the condition for vaccination to reduce the number of new infections is stronger (part b), because the strength of the Peltzman effect is determined by $\Delta \log \beta + \Delta \log \delta_I$, while the number of new infections gets reduced only on account of $\Delta \log \beta$. A pervasive feature of the vaccinations for Covid-19 that became available at the start of 2021 was a relatively strong medical effect, coupled with a limited epidemiological effect, meaning a high value of $\frac{\Delta \log \delta_I}{\Delta \log \beta}$. In light of this, Proposition 1 offers an explanation for why large-scale vaccination did not halt infection dynamics.

To see how vaccination affects welfare, we recall that the laissez-faire equilibrium involves an infection externality working towards too much social activity. In this case, since vaccination unambiguously raises the equilibrium level of this activity, it has a negative first-order effect on welfare, which may dominate the direct benefit from higher protection. In other words, vaccination raises welfare only if the Peltzman effect is not too strong. Indeed, if this effect is muted so that vaccination still

reduces the number of new infections (see Proposition 1 above), then vaccination will unambiguously increase welfare.

Proposition 2 (Vaccination and welfare)

Rolling out vaccination in the laissez-faire equilibrium affects aggregate welfare as follows:

- a) *In general, the welfare effect of vaccination is ambiguous; it will be negative, if the Peltzman-type behavioral adjustment is strong enough.*
- b) *If the net effect of vaccination on the rate of new infections is zero or negative, then its welfare effect is unambiguously positive.*

Proof The formal proof is given in the appendix. □

Lest the reader is left with a pessimistic impression about the effect of vaccination, we add an important remark. Vaccination necessarily improves welfare if introduced, not in the laissez-faire equilibrium (the case in Proposition 2), but in the social optimum. Clearly, the *direct* effects of vaccination, that is, the medical protection and the epidemiological protection, are both unambiguously welfare increasing. In the social optimum, the *indirect* effect from behavioral adjustment is zero due to the first order condition; see Eq. (7) in connection with the constraint in Eq. (6). This is an instance of the Envelope theorem. This result holds an important message for policy: the full welfare potential of vaccination can only be achieved if accompanied by a policy that corrects for the infection externality, say through mandatory social distancing, or lockdowns. In other words, implementing a (complete) vaccination policy does not remove the need of a policy of (optimal) social distancing.

Earlier results in the literature have mostly focused on an isolated epidemiological protection, i.e., the reduction in the transmission parameter β , without allowing for a simultaneous change in δ_I . These results often have a distinct Peltzman flavor. For instance, in his seminal HIV-study, Kremer (1996) demonstrates that an increase in the number of partners—the analogue to our level of social activity—caused by a lower transmission rate may increase the HIV-prevalence, provided that the pertinent behavioral elasticity is strong enough. A similar result is noted regarding social activity in a COVID-19-type environment in Carnehl et al. (2021). Kremer (1996) explicitly notes the possibility that vaccination may be a welfare reducing policy but does not pin down conditions under which this is the case.

4 Social distancing with heterogeneous individuals

The infection externality present in a laissez-faire equilibrium suggests that constraining people's social behavior is a natural way to achieve the social optimum. In this section, we demonstrate that behavioral adjustment comes back with a vengeance and may hamper the effectiveness of such lockdowns once we consider a heterogeneous population and assume—in line with actual practice—that a lockdown is imposed uniformly on the entire population.

4.1 Heterogeneity and social activity under laissez-faire

We now allow for individuals to be differently vulnerable in terms of the risk of dying from Covid. For simplicity, we assume but two groups of individuals and call the less vulnerable the “young”, and the more vulnerable the “old”. These two groups will be referred to by a subscript index $g = y, o$. When an individual belonging to group g becomes infected, her probability of dying increases by δ_{Ig} , and our assumption is that $\delta_{Io} > \delta_{Iy}$.

We assume that, other things equal, individuals do not differ in terms of their likelihood of transmitting or contracting the virus. Importantly, however, since vulnerability determines individual behaviour, the activity levels of the young and the old will generally be different. The activity level of an individual belonging to group g is denoted by a_g while A_g denotes the average within the group. Tracking the group-specific evolution of infections, we use i_g to denote the rate of infectious individuals within group g and I_o to denote the share of infectious individuals from group g in the entire population. Denoting the overall share of the old in the population by ω , we have $I_o = \omega i_o$ and $I_y = (1 - \omega) i_y$. Analogous relationships hold for the the group-specific shares of susceptible individuals, denoted by s_g and S_g , respectively.

With this notation, Eq. (2) naturally extends to

$$\Delta i_g = \beta A_g s_g (A_o I_o + A_y I_y). \quad (11)$$

The term $(A_o I_o + A_y I_y)$ captures the overall “infection base”, i.e., the risk to susceptibles that emanates jointly from the infectious young and the infectious old. The key assumption here is that in their social activity people mingle across groups. To simplify our analysis, we assume that the share of susceptibles within group o is the same as that within group y , writing s for $s_o = s_y$.

What does Eq. (11) imply for behavior? As in the baseline model, we assume that individuals do not know their health status but do know their vulnerability status g . Individuals must rely on Eq. (11) as well as common knowledge to form expectations. The probability of an individual of group g becoming infected, *conditional* on being susceptible, is equal to $\beta a_g (A_o I_o + A_y I_y)$. Invoking the law of large numbers, the individual sets the probability of being susceptible equal to s , which we assume to be public knowledge. Consequently, the individual’s perceived probability of dying during period 1 is $\delta_g(a_g) = \delta_{Ig} \beta a_g s_g (A_o I_o + A_y I_y)$, and the marginal effect of a_g on this probability is

$$\delta'_g(a_g) = \delta_{Ig} \beta s (A_o I_o + A_y I_y) > 0. \quad (12)$$

Note that all terms on the right-hand side of this equation relate to the group level, as opposed to the individual level. Hence, when deciding about a_g , individuals treat the entire right-hand side as a constant.

The optimal level of activity weighs the marginal utility against the expected utility loss from a higher probability of dying, which is equal to $\delta'_g(a_g) \bar{u}_{2g}$, where \bar{u}_{2g} denotes the period-2 (i.e., post-pandemic) utility of an individual belonging to the vulnerability-group g , conditional on surviving period 1. In view of Eq. (12), we now

assume that our vulnerability assumption $\delta_{Io} > \delta_{Iy}$ extends to $\delta_{Io}\bar{u}_{2o} > \delta_{Iy}\bar{u}_{2y}$, and we simplify writing by using $v_g := \delta_{Ig}\bar{u}_{2g}$. This gives rise the following first-order conditions determining optimal individual activity levels a_g^* , conditional on group-specific averages A_g :

$$u'(a_g^*) = v_g \beta s (A_o I_o + A_y I_y), \quad g = y, o. \quad (13)$$

The second-order conditions are satisfied from the assumption of diminishing marginal utility. Equilibrium requires that $a_g = A_g$ for $g = y, o$ and is therefore determined by

$$u'(A_o) = v_o \beta s (A_o I_o + A_y I_y), \quad \text{and} \quad (14)$$

$$u'(A_y) = v_y \beta s (A_o I_o + A_y I_y). \quad (15)$$

This is a two-equation system that jointly determines the equilibrium average activity levels for each group, which we denote by A_y^* and A_o^* . Equation (14) describes the “best-response” for the average activity of the old to alternative average activity levels of the young, and conversely for Eq. (15).⁴

Given that the old are more vulnerable, we expect them to be less active than the young. This, indeed, follows from Eqs. (14) and (15). Divide one by the other to obtain

$$\frac{u'(A_o^*)}{u'(A_y^*)} = \frac{v_o}{v_y}. \quad (16)$$

Since $v_o > v_y$, the right-hand side is larger than 1, and diminishing marginal utility then implies $A_o^* < A_y^*$. This has an important consequence for a lockdown policy. If uniformly enforced for both, the young and the old, such a policy will naturally be binding for the young but may well be non-binding for the old. The old will then have room to become more active even under lockdown conditions. And this is, indeed, what they will want to do since they perceive a safer environment from a reduced activity level of the young as enforced by the lockdown. In the next subsection, we shall provide a comparative static analysis of the laissez-faire equilibrium highlighting this type of adjustment and what it means for the evolution of infections and deaths from the pandemic.

To pave the ground for this comparative static analysis, we derive the slope of Eq. (14), which describes adjustment of the old to changes in activity of the young. Towards that end, we define $\sigma_o := -[u''_o(A_o^*)/u'_o(A_o^*)] A_o^* > 0$, which is the analogue to the elasticity σ for the homogeneous case. Differentiating Eq. (14) gives $u'' dA_o = v_o \beta s (I_o dA_o + I_y dA_y)$. Collecting terms, moving to log-changes, and approximating $d \log A_o \approx \Delta \log A_o$ and $d \log A_y \approx \Delta \log A_y$, we obtain the following response of the old to a change in the activity of the young:

$$\Delta \log A_o = -\eta_o \Delta \log A_y, \quad (17)$$

⁴ This is a slight abuse of the term best-response function, since decisions are not being made collectively by the groups, but by individuals holding group averages constant.

In this equation, $\zeta_o := A_o I_o / (A_o I_o + A_y I_y)$ measures the share of infectious old in the overall “infection base”. In turn, $\eta_o := (1 - \zeta_o) / (\zeta_o + \sigma_o)$ is the analogue for the old to η in the simpler model without heterogeneity. Clearly, $\eta_o > 1$: the two groups’ social activities are strategic substitutes.

Note that η_o , like η in the homogeneous case above, is falling in σ_o , the coefficient of risk aversion. But unlike η , η_o is not bounded from above by unity. Equation (17) is very intuitive—and key to our subsequent analysis: When the young reduce their social activity, in compliance with the lockdown policy, the old perceive a less risky environment and will therefore want to increase their activity—the more so, the less risk averse they are and the lower the share of the *young* in the “infection base”. But they will only be able to adjust in line with Eq. (17) if the lockdown, while restrictive for the young, is sufficiently mild, as we detail in the next section.

4.2 The effectiveness of lockdown policies

Suppose a lockdown policy imposes a uniform maximum level of activity $\bar{a} < A_y^*$ on the entire population. In percentage terms, the young will be forced to change their level of activity by $(\bar{a}/A_y^* - 1) < 0$. Obviously, for the lockdown to be non-binding for the old we must have $\bar{a} > A_o^*$. However, this is not sufficient for the old to be able to adjust according to Eq. (17) above. To see this, we use A_o^ℓ to denote the *desired* activity level for the old once the young are less active in compliance with the lockdown. This level can be determined from Eq. (17), simply by replacing $\Delta \log A_y = \log A_o^\ell - \log A_o^* = A_o^\ell/A_o^* - 1$ and $\Delta \log A_y = \bar{a}/A_y^* - 1$. It is easy to verify that

$$A_o^\ell = -\eta_o \bar{a} \alpha^* + (1 + \eta_o) A_o^*,$$

where $\alpha^* := A_o^*/A_y^* < 1$. Setting $A_o^\ell = \bar{a}$ gives us an equation which determines a lower bound a_ℓ for the value of \bar{a} , such that any lockdown $\bar{a} > a_\ell$ allows the old to move along their best response function according to Eq. (17):

$$a_\ell = \frac{\eta_o + 1}{\alpha^* \eta_o + 1} A_o^* = \frac{\alpha^* (\eta_o + 1)}{\alpha^* \eta_o + 1} A_y^*. \quad (18)$$

Note that $\frac{\eta_o + 1}{\alpha^* \eta_o + 1} > 1$, hence $a_\ell > A_o^*$. A lockdown level \bar{a} in the upper vicinity of A_o^* may seem like non-binding for the old, but if their degree of risk aversion is sufficiently low (high value of η_o) and if the gap between A_y^* and A_o^* is sufficiently large (low value of α^*), then the old will hit the lockdown constraint when trying to adjust according to Eq. (17). This will be true if \bar{a} , while larger than A_o^* , is lower than a_ℓ , and in this case it cannot be said that the lockdown is non-binding for the old.

The situation is illustrated by Fig. 1, based on a simple parameterization of the utility function.⁵ The equilibrium levels of A_o and A_y are measured against the vertical axis

⁵ We chose the functional form $u(a) = \ln a - a + 1$, following Farboodi et al. (2021). This specification satisfies all assumptions made above. Our calibration assumes that at the beginning of the policy period 2%

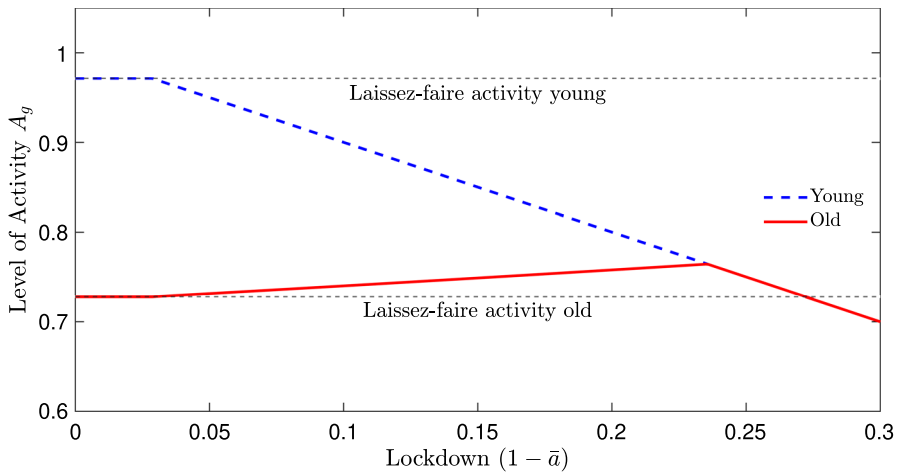


Fig. 1 Quantitative illustration of how a lockdown impacts activity levels. The vertical axis measures activity level of the young (dashed line) and the old (solid line) against lockdown intensity ($1 - \bar{a}$), measured along horizontal axis. For model parameterizations, see footnote 5

for a given lockdown stringency, $1 - \bar{a}$, depicted on the horizontal axis. The dashed blue line represents activity of the young, with a flat segment for low lockdown-stringency levels $a_\ell > A_y^*$, where the lockdown is non-binding for the young because they, too, show some social restraint under laissez faire. This changes once $1 - \bar{a}$ becomes larger than $1 - A_y^*$: The activity level of the young then falls with a slope equal to unity. The solid red line displays the activity level for the old. Beyond a certain point, the activity levels of both groups are equalized: The two lines intersect once the lockdown stringency reaches $1 - a_\ell$. For any lockdown that reduces the activity level further (stringency above $1 - a_\ell$), both A_o and A_y decline with a slope equal to unity.

This Peltzman-type adjustment by the old affects the degree to which the young will, in the end, feel restricted by the lockdown. Ex ante, one might measure the degree of restrictiveness by the ratio A_y^*/\bar{a} . Ex post, however, this overestimates the extent to which the young feel constrained. The reason is that, with the old now being more active than A_o^* , the young no longer want to choose the activity level A_y^* , but a lower level instead.

The behavioral interdependency between the more vulnerable and the less vulnerable has implications for the effectiveness of a lockdown policy. In the remainder of this section we first explore the policy's effect on new infections. Subsequently, we examine what this means for the case fatality, that is, the number of deaths per newly infected person, as well as for mortality, the number of deaths per person. And finally, we briefly explore the welfare implications.

of the population in each group are infected: $i_o = i_y = 0.02$. The share of old in the population is assumed as $\omega = 18\%$, consistent with US data on the share of population older than 65. The expected lifetime utility at the beginning of period 2 for the young is $\bar{u}_{2,y} = 1000$ and $\bar{u}_{2,o} = 100$ for the old. The probability of an infected individual to die is $\delta_{I_y} = 0.002$ for the young, and $\delta_{I_o} = 0.252$ for the old. This is consistent with estimates by the CDC (Centers for Disease Control and Prevention) of a 126 times larger mortality rate if infected for those 65 years or older. The parameter β is set to 1.

The following proposition looks at new infections, where i_g denotes the rate of new infections within group g and the operator Δ_L denotes the effect of the lockdown.

Proposition 3 (Lockdown and new infections)

A lockdown $\bar{a} \in (a_\ell, A_y^*)$

- a) *unambiguously lowers the rate of new infections among the young, and it*
- b) *raises the rate of new infections among the old, if and only if $\eta_o > \frac{1-\zeta_o}{1+\zeta_o}$.*
- c) *It is unambiguously true that $\Delta_L \log \Delta i_o > \Delta_L \log \Delta i_y$.*

Proof The proof is provided in the appendix. \square

The intuition of this proposition is relatively straightforward. New infections among the young are lowered as the lockdown forces them to become less active. This works through both infected as well as susceptible young individuals. We may call this the direct effect. At the same time the old now pose more of a threat to the young because of their Peltzman-type reaction, but this indirect effect works only through those among the old who are in fact infectious. For this reason, the direct effect always dominates, as stated in part a) of the proposition.

As to part b), new infections among the old are lowered through the direct effect of the lockdown only to the extent that the young are in fact infectious. In contrast, the harmful Peltzman effect operates through the entire group of the old. Those who are susceptible increase their exposure to infections, while those who are infectious increase the risk of infecting others. Within the group of the old, therefore, the Peltzman effect strikes twice, and if it is strong enough (high value of η_o , or low risk aversion of the old), then it may dominate the direct effect of the lockdown.⁶ Note the role of ζ_o , the share of the old in the “infection base”. Intuitively, the larger this share, the lower the critical value that η_o needs to surpass for the lockdown to cause an increase in the infection rate among the old. Remember that a high value of η_o means that the old are less risk averse. Arguably, ζ_o is higher with a higher share of more vulnerable individuals in the population as a whole.

Regarding part c) of the proposition, if the Peltzman effect does in fact cause the lockdown to increase the rate of new infections among the old, then there is the distinct possibility of the aggregate rate of new infections increasing as well, provided that the share of old in the population is large enough. But even if the rate of new infections falls within both groups, the Peltzman effect implies that it falls more (in log terms) within the group of the young than the group of the old.

This implies that the lockdown increases the share of the old in the entire group of newly infected individuals. And since the old are more vulnerable, the result is that a larger overall share of infected individuals will die from the disease. In other words, the case fatality increases.

However, a higher case fatality need not make the lockdown a detrimental policy. If it is coupled with a strong reduction in the rate of new infections, then the lockdown may still reduce the number of people who die from the infection. Indeed, this will necessarily be the case, if the lockdown reduces the number of new infections among

⁶ It should be noted here that we have assumed the share of susceptibles (infectious) to be the same across groups.

both, the young and the old. But if it causes a higher rate of new infections among the old, a distinct possibility according to part b) of Proposition 3, then the result may be a higher higher number of deaths caused by the pandemic. The condition under which this occurs is found in part b) of the following proposition.

Proposition 4 (Lockdown and pandemic-induced deaths)

A lockdown $\bar{a} \in (a_\ell, A_y^*)$ has the following effects on the number of deaths caused by the pandemic:

- a) It unambiguously increases the case fatality rate.
- b) It increases the mortality, if and only if $-\frac{\omega \Delta_L \log \Delta i_o}{(1 - \omega) \Delta_L \log \Delta i_y} > \frac{\delta_{Iy}}{\delta_{Io}} \frac{A_y^*}{A_o^*}$.

Proof The proof is provided in the appendix. \square

The case described in part b), admittedly, is an extreme case that hinges on a large relative size of the more vulnerable group in society, $\omega/(1 - \omega)$, and on a high vulnerability of this group relative to the other, δ_{Io}/δ_{Iy} . Note, too, that the log-changes in the group-specific new infection rates appearing in the condition for part b) are driven by ζ_o the activity-weighted share of the old in the overall number of new infections, as well as the degree of risk aversion.

What does the Peltzman-type adjustment of the old mean for the welfare effect of a lockdown policy? To see this, we go back to Eqs. (14) and (15), which describe the laissez-faire equilibrium. Each group ignores the infection externality that we have described in detail in Sect. 2. Importantly, this externality applies, not just within each group, but also across groups. And because the old are more vulnerable, $v_o > v_y$, the cross-group externality is larger for the activity of the young. The reason is that the old have a larger marginal utility due to a lower activity level. For the exact same reason the within-group externality is larger for the activity of the old. It is clear that in a laissez-faire equilibrium both types of individuals are too active, compared to the social optimum, but due to the aforementioned asymmetry, we cannot say which of the two groups exhibits the larger excess activity.

An optimal policy of social distancing would choose different target levels of activity for each group. Also, the marginal effect of a lockdown that is binding for both groups, starting from the laissez-faire equilibrium, would clearly be welfare increasing. However, the same cannot be said for a uniform lockdown which is non-binding. The reason is that such a policy has two opposing first-order welfare effects, given that both groups are too active, yet respond in opposite directions to the policy. The enforced reduction in the activity of the young has a positive first order welfare effect, both for the young themselves and for the old. And the Peltzman-type reaction of the old has a negative first order welfare effect, again affecting both groups. Without knowing details about the social welfare function, one cannot rule out that the Peltzman effect renders a uniform lockdown welfare decreasing.

The way in which the Peltzman effect works out for fatality and mortality, namely through a vicious composition effect among the infected (higher share of more vulnerable individuals), is reminiscent of an effect noted in the HIV-context by Kremer (1996). Heterogeneity in his case means people choosing different levels of activity

for the same HIV-prevalence. A Peltzman-type reaction of heterogeneous individuals to a change in transmission may then change the composition of the pool of available partners. More specifically, if high-activity people respond to higher prevalence by lowering their activity by less, proportionally, than low-activity people, then the activity-weighted HIV-prevalence increases, which in turn raises the number of new infections. Kremer (1996) argues that this type of asymmetric reaction among heterogeneous individuals is very likely. Indeed, he even notes the possibility of high-activity people increasing their activity as a result of higher HIV-prevalence. Obviously, this asymmetry raises the specter of multiple equilibria, which is at the core of his analysis of heterogeneity. While similar to some extent, the mechanism in Kremer (1996) is different in crucial ways. Most importantly, heterogeneity in our case relates to the health hazard, not to behavior as such. Moreover, the mechanisms that we highlight do not entail the possibility of multiple equilibria.

The above analysis of heterogeneous individuals can also be applied to cross-border contacts between different countries. Antràs et al. (2023) analyse the implications of such “epidemiological integration” where contacts between differently healthy countries are related to international trade. Our analysis suggests that in such an environment national policies will have cross-border externalities not just of the epidemiological sort, but also through Peltzman effects: If one country’s social distancing policy reduces the infection risk that this country poses for individuals of other countries, then these individuals will face an incentive to increase their levels of activity, their own country’s policy permitting.

5 Evidence

We now confront the predictions of the theory with data for US states, on the one hand, and a group of countries in Western Europe on the other.⁷ These are two fairly homogeneous sets of countries. Still, each US state or European country has experienced its distinct dynamics of the pandemic and has implemented its own policy measures, which makes them particularly suitable for our empirical analysis.

We rely on data from March 2020 until December 2021. We use data for infections and mortality (Dong et al. 2020), vaccination (Mathieu et al. 2021) and testing (Hasell et al. 2020). To measure the stringency of lockdowns, we use the “Containment and Health Index” compiled by Hale et al. (2021). Monthly infections I_t , deaths D_t and tests T_t are measured per 100,000 people. The monthly case fatality rate is denoted by

⁷ Specifically, our sample includes Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Netherlands, Norway, Portugal, Spain, Sweden and the United Kingdom.

CFR_{*t*}. To account for the average time between infection and death due to COVID-19 we lead the death count by 2 weeks as we compile the CFR_{*t*} (Pachetti et al. 2020).⁸

In what follows, we relate different outcome variables, x_{it} , for a given country/state i and in a given month t to public policy measures implemented in the previous month. We consider three outcome variables, each measured in logs: new infections, Covid-related deaths and the case-fatality rate. In terms of policy measures we focus on the vaccination rate, v_{it-1} , and the lockdown stringency, s_{it-1} . Importantly, we interact the effect of lockdown stringency with an indicator variable, V_{it} , which assumes a value of zero for all observations in 2020, that is, prior to the start of vaccinations in early 2021. In this way we account of the fact that, according to our model, the Peltzman effect in the reaction to the lockdown first and foremost depends on whether there is heterogeneity in the population in terms of vulnerability. Since the vaccinations were initially targeted mostly at the more vulnerable, an important effect of the vaccination was to reduce the degree of heterogeneity of the population. We thus expect the Peltzman effect to show up in our results primarily for the no-vaccination regime in 2020.

Formally, we estimate the following equation with an error term u_{it} :

$$x_{it} = \gamma_1(1 - V_{it-1})s_{it-1} + \gamma_2 V_{it-1}s_{it-1} + \xi v_{it-1} + \beta X_{it} + \theta_i + u_{it}. \quad (20)$$

In this expression, θ_i are fixed effects for countries/states and X_{it} is a vector of control variables which includes two lags of the dependent variable and the number of tests in the current as well as in the two previous months, and the lag of the indicator variable. Our main interest lies in the parameters γ and ξ , which we estimate using OLS, reporting robust standard errors.

Table 1 shows the results, in the left panel for US states, in the right panel for the countries in Western Europe.⁹ The first line reports the estimated coefficients γ which speaks to a possible effect of the lockdown in the pre-vaccination period: stronger lockdowns are associated with fewer infections and deaths in the following month. However, the association is weaker in the case of deaths, where it is not even significant for European countries. Most importantly, in column three, we find that more stringent lockdowns are associated with *increasing* case fatality rates, particularly in Europe. This is consistent with the prediction of our model that a lockdown, if imposed uniformly across a heterogeneous population, triggers a Peltzman effect which causes a higher case fatality.¹⁰ The second line shows the estimate for the same relationship once vaccination has become available. In this case the association between lock-

⁸ Formally, we use:

$$\text{CFR}_t = \frac{\sum_{d=1}^N \text{Deaths}_{d+14}}{\sum_{d=1}^N \text{Infections}_d}, \quad (19)$$

where Deaths_d (Infections_d) denotes the number of new deaths (infections) on day d of month t . N is the number of days in a month.

⁹ Table 2 in the Appendix shows that results are robust when we estimate the specification with the dependent variables in levels, instead of logs.

¹⁰ See also early evidence by Cao et al. (2020). They find that stricter lockdown measures are associated with higher case-fatality rates in high-income countries.

Table 1 Effect of public policy measures

	US states			Western Europe		
	Infections _{<i>t</i>}	Deaths _{<i>t</i>}	CFR _{<i>t</i>}	Infections _{<i>t</i>}	Deaths _{<i>t</i>}	CFR _{<i>t</i>}
γ_1	−1.025*** (−3.87)	−0.726** (−2.75)	0.630** (−2.97)	−1.565** (−3.31)	−0.448 (−0.73)	1.251** (−2.64)
γ_2	−1.275*** (−4.70)	−1.010*** (−4.48)	0.152 (−0.65)	−1.815*** (−3.48)	−1.120* (−2.39)	0.769* (−1.97)
ξ	0.415* (−2.51)	0.321* (−2.14)	−0.237 (−1.49)	−0.377 (−1.47)	−0.418 (−1.76)	0.181 (−1.06)
State FE	yes	yes	yes	yes	yes	yes
N	988	975	975	282	277	277
r ²	0.812	0.790	0.500	0.795	0.773	0.707

Parameter estimates for model (20); monthly observations 2020:03–2021:12; containment index is the log of stringency index, dependent variables are measured per 100 thousand population for Deaths and Infections; robust standard errors used; *t* statistics in parentheses, based on robust standard errors; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

down stringency and CFR becomes much weaker, again as our model suggests it will. Note, too, that with the Peltzman effect arguably subdued due to vaccination (lower degree of heterogeneity), the negative association between the lockdown stringency and infections as well as deaths is stronger in line two than in line one.

The third line of the table reports the estimate for the relationship between the vaccination rate, v_{it-1} , which varies on the interval $[0, 1]$. Here again we detect a striking pattern. Contrary to common-sense expectation, the vaccination rate in the previous months is not strongly associated with lower infections or deaths in the current months. In Western Europe there is no significant relationship at all, while in the US it is even positive—and marginally significant. Such a pattern, if studied through the lens of our model, does not suggest that vaccinations are not effective. Indeed, it is *because* they are highly effective that they trigger a Peltzman effect, which may limit or even offset their direct medical effect.¹¹

6 Conclusion

In this paper, we analyze Peltzman effects in the context of the COVID-19 pandemic: self-interested individuals will respond to policies aimed at reducing the health hazard from an epidemic by becoming less cautious in their economic and social behavior. We investigate how such behavior may erode the effectiveness of vaccination and social distancing (lockdown). Using a stylized SIR-model that allows for behavioral adjustment, we identify conditions under which a Peltzman effect is responsible for a policy of rapid and complete vaccination to cause an *increase* in the number of new infections. We emphasize, however, that this does not necessarily make vaccination

¹¹ Consistent with this interpretation, Guo et al. (2021) provide cross-country evidence suggesting that vaccination increases social mobility.

a welfare-reducing policy, although in extreme cases it may. A further key message from our analysis is that heterogeneity among the population may give rise to an unexpected Peltzman effect also for lockdown policies. Due to differential adjustments to the pandemic prior to the lockdown, a lockdown may be non-binding for individuals who are more cautious to start with. These individuals will then feel an incentive to become less cautious in response to the safer environment generated by the lockdown on account of its binding effect on less cautious individuals. Our empirical analysis detects patterns in the data that suggest these effects have been non-negligible in magnitude during the Covid-19 pandemic. They are likely to be relevant also for policies aimed at epidemics in the future.

Appendix A Proof of Propositions

An appendix contains supplementary information that is not an essential part of the text itself but which may be helpful in providing a more comprehensive understanding of the research problem or it is information that is too cumbersome to be included in the body of the paper.

Proposition 2 states:

Rolling out vaccination as described above affects aggregate welfare as follows: a) In general, the welfare effect of vaccination is ambiguous. It will be negative, if the Peltzman-type behavioral adjustment is strong enough.

b) If the net effect of vaccination on the rate of new infections is zero or negative, then its welfare effect is unambiguously positive.

Proof The relevant welfare equation is

$$W(A^*) = u(A^*) + 1 - \delta(A^*). \quad (\text{A1})$$

The proposition is easier to prove using changes in levels, rather than log-changes. In a vaccination scenario, we have

$$\Delta_V \delta(A^*) = \Delta_V (\delta_I \Delta I) = \Delta I \Delta \delta_I + \delta_I \frac{\partial \Delta I}{\partial \beta} \Delta \beta + \delta_I \frac{\partial \Delta I}{\partial A} \Delta A^*. \quad (\text{A2})$$

The first two terms on the right-hand side capture the direct vaccination effect, with $\Delta \delta_I < 0$ and $\Delta \beta < 0$, while the third term captures behavioral adjustment governed by Eq. (8). In a laissez-faire equilibrium we have $\Delta I = \beta A^{*2} SI$, hence $\partial \Delta I / \partial A = 2\beta A^* SI > 0$ and $\partial \Delta I / \partial \beta = A^{*2} SI > 0$. The welfare change emerges as

$$\begin{aligned} \Delta W \approx & [u'(A^*) - \delta_I 2\beta SA^* I] \Delta A^* \\ & - [\Delta I \Delta \delta_I + \delta_I (A^{*2} SI) \Delta \beta] \end{aligned} \quad (\text{A3})$$

The first-order condition (5) allows us to rewrite the bracketed term in the first line above as $-\beta \delta_I SA^* I \Delta A^* < 0$. We know that vaccination increases A^* , hence the first term on the right-hand side is negative, while the expression in the second line is

unambiguously positive as $\Delta\delta_I < 0$ and $\Delta\beta < 0$. In general, therefore, the welfare effect of vaccination is ambiguous, which proves **part a)** of the proposition.

As to **part b)**, note that the net effect of vaccination on new infections is $2\beta SA^*I\Delta A^* + A^{*2}SI\Delta\beta$. If this is equal to zero, we are left with $\Delta W = u'(\cdot)\Delta A^* - \Delta I\Delta\delta_I$, which is clearly positive. The same is true, a fortiori, if the net effect on the rate of new infections is negative. \square

Proposition 3 states:

A lockdown $\bar{a} \in (a_\ell, A_y^*)$

a) unambiguously lowers the rate of new infections among the young, and it

b) raises the rate of new infections among the old if and only if $\eta_o > \frac{1-\zeta_o}{1+\zeta_o}$.

c) It is unambiguously true that $\Delta_L \log \Delta i_o > \Delta_L \log \Delta i_y$.

Proof In view of Eq. (11), the lockdown-induced relative changes in the rates of new infections in the two groups are

$$\Delta_L \log \Delta i_o = \Delta \log A_o + \zeta_o \Delta \log A_o + (1 - \zeta_o) \Delta \log A_y \quad (\text{A4})$$

$$\Delta_L \log \Delta i_y = \Delta \log A_y + \zeta_o \Delta \log A_o + (1 - \zeta_o) \Delta \log A_y \quad (\text{A5})$$

A lockdown $\bar{a} \in (a_\ell, A_y^*)$ implies $\Delta \log A_y = (\bar{a}/A_y^* - 1) < 0$ and risk-compensating adjustment implies $\Delta \log A_o = \log A_o^l - \log A_o^* = -\eta_o \Delta \log A_y$; see Eq. (17). Inserting this, we obtain:

$$\Delta_L \log \Delta i_o = [1 - \zeta_o - (1 + \zeta_o)\eta_o] \Delta \log A_y \quad (\text{A6})$$

$$\Delta_L \log \Delta i_y = [2 - \zeta_o(\eta_o + 1)] \Delta \log A_y \quad (\text{A7})$$

Part a): Since $\Delta \log A_y < 0$, $\Delta_L \log \Delta i_y < 0$ if and only if $2 - \zeta_o(\eta_o + 1) > 0$, which is equivalent to $\eta_o < 2/\zeta_o - 1$. Inserting the definition of $\eta_o := (1 - \zeta_o - \eta_o) / (\zeta_o + \sigma_o)$, this results in $\sigma_o > -\zeta_o(1 + \epsilon_o) / (2 - \zeta_o)$. By definition, $\zeta_o < 1$ and $\sigma_o > 0$, hence the equality is satisfied.

Part b): By analogous logic, this follows from setting $1 - \zeta_o - (1 + \zeta_o)\eta_o < 0$.

Part c): Equations (A6) and (A7) imply that $\Delta_L \log \Delta i_o = \Delta_L \log \Delta i_y + \Delta \log A_o - \Delta \log A_y$, which in turn implies $\Delta_L \log \Delta i_o = \Delta_L \log \Delta i_y - (1 + \eta_o)\Delta \log A_y > \Delta_L \log \Delta i_y$. The inequality follows from $\Delta \log A_y = (\bar{a}/A_y^* - 1) < 0$ and $\eta_o > 0$. \square

Proposition 4 states:

A lockdown $\bar{a} \in (a_\ell, A_y^*)$ has the following effects on the number of deaths caused by the pandemic:

a) It unambiguously increases the fatality rate.

b) It increases the mortality if and only if $-\frac{\omega \Delta_L \log \Delta i_o}{(1 - \omega) \Delta_L \log \Delta i_y} > \frac{\delta_{Iy}}{\delta_{Io}} \frac{A_y^*}{A_o^*}$.

Proof Part a): We rewrite the laissez-faire case fatality rate as

$$f = \delta_{I_o} \frac{\Delta I_o}{\Delta I} + \delta_{I_y} \frac{\Delta I_y}{\Delta I} = \delta_{I_y} + (\delta_{I_o} - \delta_{I_y}) \frac{\Delta I_o}{\Delta I}, \quad (\text{A8})$$

where the second equality follows from $\Delta I = \Delta I_o + \Delta I_y$. The lockdown-induced change in f follows as

$$\Delta_L f = (\delta_{I_o} - \delta_{I_y}) \Delta_L \frac{\Delta I_o}{\Delta I} \quad (\text{A9})$$

Remember that $I_o = \omega i_o$, whence $\Delta I_o = \omega \Delta i_o$ and $\Delta_L \log \Delta I_o = \Delta_L \log \Delta i_o$. In view of $\delta_{I_o} > \delta_{I_y}$, $\Delta_L f$ is positive if and only if $\Delta_L \log \Delta I_o > \Delta_L \log \Delta I$. Since $\Delta I = \Delta I_o + \Delta I_y$, $\Delta_L \log \Delta I$ is a weighted average of $\Delta_L \log \Delta I_o = \Delta_L \log \Delta i_o$ and $\Delta_L \log \Delta I_y = \Delta_L \log \Delta i_y$. But we know from Proposition 3 that $\Delta_L \log \Delta i_o > \Delta_L \log \Delta i_y$. Hence, this condition is fulfilled.

Part b): The mortality rate m is driven by new infections according to

$$m = \delta_{I_o} \omega \Delta i_o + \delta_{I_y} (1 - \omega) \Delta i_y \quad (\text{A10})$$

Differentiating this equation yields

$$\Delta_L m = \delta_{I_o} \omega \Delta i_o \Delta_L \log \Delta i_o + \delta_{I_y} (1 - \omega) \Delta i_y \Delta_L \log \Delta i_y. \quad (\text{A11})$$

Setting $\Delta_L m > 0$, we obtain

$$\delta_{I_o} \omega \Delta_L \log \Delta i_o + \delta_{I_y} (1 - \omega) \frac{\Delta i_y}{\Delta i_o} \Delta_L \log \Delta i_y > 0. \quad (\text{A12})$$

In view of Eq. (11), we may set $\frac{\Delta i_y}{\Delta i_o} = \frac{A_y^*}{A_o^*}$. Remember we have assumed $s_o = s_y$. Since $\Delta_L \log \Delta i_y < 0$, we may rearrange terms in the above inequality to obtain the inequality in part b) of the proposition. \square

Appendix B Additional tables

This table replicates the estimation in Table 1, with the dependent variables in levels, instead of logs. Monthly observations 2020:03–2021:12; containment index is the log of stringency index, dependent variables are measured per 100 thousand population for Deaths and Infections; robust standard errors used; t statistics in parentheses, based on robust standard errors; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 2 Robustness: effect of public policy measures

	US states			Western Europe		
	Infections _{<i>t</i>}	Deaths _{<i>t</i>}	CFR _{<i>t</i>}	Infections _{<i>t</i>}	Deaths _{<i>t</i>}	CFR _{<i>t</i>}
γ_1	-24.21*** (-3.33)	-0.224 (-1.67)	0.560** (2.67)	5.233 (0.74)	0.127 (1.13)	1.565*** (3.60)
γ_2	-26.98*** (-4.44)	-0.276* (-2.43)	-0.00669 (-0.03)	-11.65 (-0.97)	-0.136 (-1.28)	1.251** (3.08)
ξ	14.33*** (3.78)	0.133 (1.85)	-0.278 (-1.74)	12.63 (1.78)	-0.0900 (-1.60)	-0.0970 (-0.50)
Controls	Yes	Yes	Yes	Yes	Yes	Yes
State FE	Yes	Yes	Yes	Yes	Yes	Yes
N	988	988	983	282	282	281
r2	0.702	0.640	0.487	0.594	0.560	0.643

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