Pandemics, Public Policy, and Peltzman Effects

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February 2023

Abstract

The study of infection dynamics routinely relies on versions of the compartmental SIR model. We extend the basic SIR model to explore the trade-offs which govern individual behavior. We limit our analysis to a highly stylized version of the model and analyze peoples' response to specific public policies in closed form. For both, vaccination and lockdown policies we establish Peltzman effects: As policies lower the risk of infections, people become more socially active, in turn, undermining their effectiveness. Data for US states and countries in Western Europe suggest that such effects are shaping actual infection dynamics to a considerable extent.

Keywords: Covid-19, Pandemic, Behavioral adjustment, SIR Model, Risk compensation, Lockdowns, Vaccinations
JEL-Codes: I12, I18

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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. What do we know about the effectiveness of these policies? Tackling this question empirically and establishing well-defined counterfactuals is notoriously difficult (e.g., Born et al., 2021; Brauner et al., 2021; Flaxman et al., 2020). In one sense, however, the success of these policies was limited: They did not halt infection dynamics. In the US and Europe infections numbers reached very high levels after the policies had been put in place for some time. In this paper, we argue that these policies—though clearly desirable from a medical point of view—are bound to deliver somewhat disappointing results: Precisely because they create a safer environment, they induce people to adjust their behavior in ways which may partly (or even fully) offset the direct effect of the policy. Peltzman (1975) was the first to analyse this type of adjustment in the context of traffic safety regulation. We put it at the center stage of our analysis of pandemics.

We use a very stylized Susceptible-Infected-Recovered (SIR) model, as frequently employed to study infection dynamics, which we extend to allow 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, they raise the level of social activity—the Peltzman effect, 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 large, potentially even making vaccination a welfare reducing policy.

Second, we argue that a Peltzman effect is likely to arise also for a lockdown policy, provided it is uniformly imposed on a heterogeneous population where some individuals are more vulnerable to the disease than others. Being aware of their vulnerability, individuals exercise different levels of self-restraint once the pandemic arrives. Consequently, a 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. They have room to adjust and will increase their social activity in response to the lower risk. This unambiguously implies a higher case fatality rate and—depending on parameters—may even increase mortality.

Finally, we present evidence on actual infection dynamics based on monthly observations for US states and countries of the European Union (EU), respectively. First, 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 vaccinations and a later period when vaccinations had become available. We find that the lockdown stringency is negatively related to mortality, and more so in the second period, but positively related to case fatality, and more so in the first period. We argue that these results are again consistent with a Peltzman effect of the type we highlight in our model, since vaccinations lower the degree of heterogeneity in the population in terms of vulnerability.

Pandemics have their own laws of motion. There is a large body of epidemiological literature that goes at length in describing these laws, mostly versions of the SIR-dynamics pioneered by Kermack and McKendrick (1927), and each pandemic sees multiple attempts to combine knowledge about these laws with vast amounts of data in order to generate short term predictions of infections, hospitalizations and deaths caused by the pandemic (see, for instance, Nixon et al., 2022). A fairly large economics literature complements such epidemiological studies by bringing to the fore people's rational response to a changing environment (for a survey, see Bloom et al., 2022). Our paper makes this point as transparent as possible. We study what we consider the simplest extension of the SIR model, building on Farboodi et al. (2021). Due to our simplifications, we are able to provide a succinct, closed form analysis of pandemic-related policies instead of simulation results. Analytical treatments are also offered by Toxvaerd (2019) and Rachel (2022), but our paper differs from these in important ways. First, Toxvaerd (2019) solves for steady-state effects of permanent policies in a continuous-time framework, whereas we focus on short-run effects of temporary policies in discrete time. The difference to Rachel (2022), who also uses a continuous-time model, is that he describes the equilibrium trajectory of private mitigating behavior as well as optimal mitigation policy, but does not investigate how Peltzman effects erode the effectiveness of non-optimal policies, as we do in this paper. Importantly, none of these papers focuses on heterogeneity of the population, which is at the heart of our analysis of social distancing.

Some papers suggest that Peltzman effects may govern the policy impact in the context of the pandemic, for instance when it comes to wearing masks, to mandatory testing (Diederichs et al., 2022; Geloso, 2020), or indeed to vaccination (Guo et al., 2021; Trogen and Caplan, 2021). Our distinct contribution is to offer a formal analysis of these effects. We also add an important additional element, which is heterogeneity among individuals in terms of vulnerability. The need to take account of this is widely acknowledged, and

some of the simulation studies do allow for such heterogeneity (Acemoglu et al., 2021; Brotherhood et al., 2021). However, to the best of our knowledge, the implication of such heterogeneity for lockdowns that we highlight here has so far not been acknowledged in the literature or the policy debate.

1 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.

1.1 Infection dynamics and social activity

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 is

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

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 the infectious recovers and a fraction μ dies, whence the stock of the deceased changes according to $\Delta D_t = \mu I_t$. We assume that the parameters β, μ as well as ρ are time-invariant.[1] Assuming that the recovered are no longer susceptible, we have $N_t = I_t + R_t + S_t$.

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 Equation (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

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

not be of equal length.

Essentially, what we want to analyze is how the number of new infections and, with it, the number of deaths during period 1 are affected by the policy. For easier writing, we drop all time-indexing and define I as pertaining to the beginning of period 1 and ΔI as the number of new infections occurring during the policy period. Similarly, ΔD denotes the number of *additional* deaths caused by these new infections. Moreover, we normalize $N = 1$ so that S and I are shares of the population.

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, Equation (1) changes to

$$\Delta I = \beta A_S S A_I I. \tag{2}$$

As in Farboodi et al. (2021), we assume that the individual does not know her health status (susceptible or infectious), hence we may simplify $A_I = A_S = A$.

We assume that a representative individual's wellbeing may be described by a periodic utility function $u(a, A)$, where a denotes the individual's level activity. Note that periodic utility is the same for susceptible and infectious individuals. The utility function has the following properties (subscripts indicate partial derivatives):

$$u_a(\cdot), u_A(\cdot) > 0,\ u_{aa}(\cdot), u_{AA}(\cdot) < 0,\ \text{and } u_{aA}(\cdot) \geq 0. \tag{3}$$

The second inequality assumes diminishing marginal utility, and the third inequality assumes, plausibly, that the individual and the average level of social activity are complements. In addition to these assumptions, we assume that marginal utility approaches infinity as social activity approaches zero.

Individuals decide about their activity levels at the beginning of period 1. Crucially, we assume that the expected life time utility at the beginning of period 2 is affected by the individual's period-1 activity level only through the probability that the individual survives to period 2. 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. We use $\delta(a)$ to indicate the probability of dying in period 1, depending on the activity level a. Thus, an individual alive at the beginning of period 1 faces an expected life-time utility, denoted by $W(a, A)$, which is given by

$$W(a, A) = u(a, A) + [1 - \delta(a)]\bar{u}_2, \tag{4}$$

where $\bar{u}_2$ is expected life-time utility at the beginning of period 2. For simplicity, we abstract from discounting and normalize utility such that $\bar{u}_2 = 1$.

1.2 Externalities

Assuming individuals know about Equation (2), the perceived likelihood of becoming infected during period 1, *conditional* on being susceptible, is equal to βaAI. By the law of large numbers, the probability of any one individual being susceptible is equal to S, which we assume is known to the individual. Moreover, we assume atomistic individuals who treat the average level of activity A as a constant when deciding about a. Using $\delta_I > 0$ to measure the *excess* probability of dying, caused by an infection, the perceived marginal effect of the individual's activity a on the likelihood of dying is

$$\delta'(a) = \delta_I \beta SAI. \tag{5}$$

Individual behavior is then determined through the following maximization problem:

$$\max_a u(a, A) + 1 - \delta(a) \quad \text{s.t.} \quad \delta'(a) = \delta_I \beta SAI. \tag{6}$$

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

$$u_a(a^*, A) = \delta_I \beta SAI. \tag{7}$$

The second order condition is satisfied from $u_{aa} < 0$. Homogeneity of agents implies that the equilibrium satisfies $a^* = A^*$ and is characterized by

$$u_a(A^*, A^*) - \delta_I \beta SA^* I = 0. \tag{8}$$

Under the assumptions made about the utility function, such an equilibrium exists.

We now compare this laissez-faire equilibrium with the social optimum. The social planner knows she is dealing with a homogeneous population, and she knows that the social activity level of infectious and susceptible individuals enter symmetrically in Equation (2). Hence, she aims at a uniform optimal social activity. Replacing $A_I = A_S = A$ in (2), we have $\partial \Delta I / \partial A = 2\beta ASI$. The social planner takes into account that a symmetric increase in the level of social activity increases both, the risk of becoming infected as well as the risk of becoming a virus spreader. In contrast, the individual is concerned only about the risk of becoming infected.

The social planner solves the following maximization problem:

$$\max_A u(A, A) + 1 - \delta(A) \quad \text{s.t.} \quad \delta'(A) = \delta_I 2\beta SAI. \tag{9}$$

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

$$u_a(\tilde{A}, \tilde{A}) + u_A(\tilde{A}, \tilde{A}) - \delta_I 2\beta \tilde{A} SI = 0. \tag{10}$$

Note that u_a is the marginal utility with respect to an individual's own activity while u_A is the marginal utility with respect to the average activity of the economy at large. Evaluating the left-hand side of Equation (10) at the laissez-faire level A^*, we obtain $u_a(A^*, A^*) - \delta_I 2\beta A^* SI + u_A(A^*, A^*)$. Benchmarking the laissez-faire case characterized by Equation (8) against this expression, we recognize that the individual's optimal choice implies $u_a(A^*, A^*) - \delta_I \beta A^* SI = 0$, which reduces the expression to $-\delta_I \beta A^* SI + u_A(A^*, A^*)$, reflecting two externalities. The first is the so-called *infection externality* which means that the individual ignores the fact that becoming infected raises the odds of others becoming infected, too. The second is a *social externality* which means that the individual ignores the positive effect that her own activity has on the utility of others. If $-\delta_I \beta A^* SI + u_A(A^*, A^*) < 0$, then the infection externality dominates and the laissez-faire equilibrium involves too much activity, and vice versa for $-\delta_I \beta A^* SI + u_A(A^*, A^*) > 0$. If this term is equal to zero, then the two externalities offset each other and the laissez-faire equilibrium is efficient.

2 Vaccination

The first policy we consider is vaccination. 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 was implemented. The effects of vaccination are common knowledge. Specifically, people understand that vaccination has two *direct* effects. First, it lowers δ_I, the excess death probability caused by an infection (medical protection). And secondly, it lowers the likelihood of susceptibles becoming infected as well as the likelihood of infectious individuals to transmit the virus (epidemiological protection), thus lowering β.[2]

For easier writing, we define $u'(A) := u_a(A, A)$ and assume that $u''(A) = u_{aa}(A, A) + u_{aA}(A, A) < 0$. This means, plausibly, that the marginal utility of social activity is diminishing as social activity is increased in "pari-passu" fashion (i.e., equally by all individuals). Moreover, we define $\sigma := -[u''(A^*)/u'(A^*)]A^* > 0$, which we refer to as the coefficient of relative risk aversion (or the inverse of the elasticity of intertemporal substitution).[3] Writing Equation (8) 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.[4] This implies:

$$\Delta_V \log A^* \quad = \quad -\eta \left(\Delta \log \delta_I + \Delta \log \beta\right) > 0. \tag{11}$$

In this equation, we use $\eta := 1/(1 + \sigma)$, a parameter which lies between zero and unity, with a lower value of η indicating a higher degree of risk aversion. The inequality in (11) 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 activity. This is the Peltzman (1975) effect for vaccination, which we also refer to as risk-compensating adjustment below.

We may write Equation (2) for the laissez-faire equilibrium as $\Delta I = \beta(A^*)^2 SI$. Taking logs and differentiating, we obtain $\log \Delta I = \Delta \log \beta + 2\Delta \log A^*$. In the sequel, we write $\Delta_V \log \Delta I$ for the change in the number of new infections brought about by vaccination, and likewise for deaths D. Substituting from Equation (11), we obtain:

$$\Delta_V \log \Delta I = (1 - 2\eta)\Delta \log \beta - 2\eta \Delta \log \delta_I. \tag{12}$$

[2]Plausibly, we assume that the medical protection effect does not apply to individuals who were infected already prior to the start of the vaccination program.

[3]The elasticity σ describes the curvature of the utility function $u(A, A)$. One might also describe the degree of risk aversion through the curvature of $u(a, A)$ with respect to a, holding A constant. Obviously, for comparative static analysis we must use the above definition of σ.

[4]This expression relies on a first-order Taylor series approximation $\Delta \log u'(A^*) \approx \frac{u''(A^*)}{u'(A^*)} A^* \frac{A - A^*}{A^*}$ and on $\frac{A - A^*}{A^*} \approx \log(A) - \log(A^*) = \Delta \log A^*$.

The effect of vaccination on new infections is potentially ambiguous since, depending on η, the direct vaccination effect and the risk-compensating adjustment run in opposite directions.

Next, turn to the effect of vaccination on the number of deaths. We have $\Delta D = \delta_I \Delta I$ for the additional pandemic-induced deaths, or $\log \Delta D = \log \delta_I + \log \Delta I$. Substituting from above, we have

$$\Delta_V \log \Delta D \quad = \quad (1 - 2\eta) \left(\Delta \log \beta + \Delta \log \delta_I \right). \tag{13}$$

Proposition 1 (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 Equation (13), 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 $\sigma > 1 + 2\frac{\Delta \log \delta_I}{\Delta \log \beta}$. □

The intuition for this proposition is straightforward. The strength of the Peltzman effect is higher with a lower degree of risk aversion σ. For $\sigma > 1$, 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 vaccination helps for new infections only on account of $\Delta \log \beta$.

To see how vaccination affects welfare, we recall that the laissez faire equilibrium involves two externalities: the infection externality working towards too much activity and the social externality working in the opposite direction. Except for the knife-edge case where these externalities exactly offset each other, vaccination has a first-order effect on welfare. Since vaccination unambiguously raises the equilibrium level of activity, the first-order effect is positive if the social externality dominates the infection externality. Adding the medical effect of vaccination it follows that in this case vaccination is unambiguously welfare increasing. However, if the infection externality dominates, then the first-order effect is negative and vaccination raises welfare only if the Peltzman effect is not too strong.

Proposition 2 (Vaccination and welfare)**.**

Rolling out vaccination as described above affects aggregate welfare as follows:
a) *If the social externality weakly dominates the infection externality, $u_A(A^*, A^*) \geq \beta S A^* I$, then vaccination is unambiguously welfare increasing.*
b) *If the infection externality dominates the social externality, then the welfare effect of vaccination* ***may*** *be negative.*
c) *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. □

That vaccination may reduce welfare is a somewhat provocative result since the direct effect of vaccination, reducing the risk of infection and the risk of dying from an infection, is surely welfare increasing. But once we recognize the presence of a negative first-order effect deriving from risk-compensating behavioral adjustment the welfare-reducing potential is quite obvious.

3 Social distancing with heterogeneous individuals

We now turn to an analysis of social distancing measures, or "lockdowns". Our baseline model suggests that constraining people's social behavior is a natural way to achieve the optimal level of social activity. In this section, we demonstrate that behavioral adjustment comes back with a vengeance and may hamper the effectiveness of lockdowns once we consider a heterogeneous population and assume—in line with actual practice—that a lockdown is imposed uniformly on this population.

3.1 Heterogeneity and social activity under laissez-faire

To begin with, we present the extension of our model looking at the laissez-faire equilibrium. We distinguish between two types of people, differing only in terms of the excess death risk that they face once infected. For want of a better term, we call the more vulnerable group *the old* and the less vulnerable group *the young*, and we denote variables relating to these groups by a subscript index $g = y, o$. Denoting the group-specific excess death risk by δ_{Ig}, we assume $\delta_{Io} > \delta_{Iy}$. We use ω to denote the share of the old in the population and i_g to denote the initial (beginning of the lockdown period) share of infectious individuals

within group g and s_g for the share of susceptibles. The initial share of infectious old in the population is then equal to $I_o = \omega i_o$ (likewise for the young), and accordingly for the shares of susceptibles S_o. By analogy to Equation (2), we have

$$\Delta i_g = \beta A_g s_g \left(A_o I_o + A_y I_y\right). \tag{14}$$

The key assumption here is that in their social activity people mingle across groups. The term $(A_o I_o + A_y I_y)$ thus captures the risk to susceptibles that emanates jointly from the infectious in the two groups. We assume that individuals know about their vulnerability status g but are not sure about their health status, as in the baseline model.

Periodic utility for the two groups is perfectly analogous to the homogeneous case and written as $u_g = u(a_g, A)$, where $A := \omega A_o + (1-\omega)A_y$. We have $u_a > 0$, $u_{A_o} = u_A \omega > 0$ and $u_{A_y} = u_A(1-\omega) > 0$. Lifetime utility of a type-g individual is given by:

$$W_g(a_g, A) = u(a_g, A) + [1 - \delta_g(a_g)]\bar{u}_{2g}, \quad g = y, o \tag{15}$$

where the term $\bar{u}_{2g}$ denotes the expected life time utility at the beginning of period 2.

Given the dynamics of infections in (14), the probability of an individual becoming infected, *conditional* on being susceptible, is equal to $\beta a_g \left(A_o I_o + A_y I_y\right)$. Invoking the law of large numbers, the individual sets the probability of being susceptible equal to s_g, which we assume to be public knowledge. Consequently, the individual's perceived probability of dying during period 1 is $\delta_g(a_g) = \delta_{I_g} \beta a_g s_g \left(A_o I_o + A_y I_y\right)$, and

$$\delta_g'(a_g) = \delta_{I_g} \beta s_g \left(A_o I_o + A_y I_y\right) > 0. \tag{16}$$

In what follows, we simplify by assuming $s_y = s_o = s$ and by writing $v_g := \delta_{I_g} \bar{u}_{2g} > 0$, referred to as group g's vulnerability.

The first-order conditions for an optimal level a_g^* are:

$$u_a\left(a_g^*, A\right) = v_g \beta s \left(A_o I_o + A_y I_y\right), \quad g = y, o. \tag{17}$$

Note that the "atomistic" individual treats the average level of activity A as given. The second-order condition is satisfied from the assumption of diminishing marginal utility.

Equilibrium requires that $a_g = A_g$ for $g = y, o$ and is therefore determined by

$$u_a(A_o, A) = v_o \beta s \left(A_o I_o + A_y I_y\right), \tag{18}$$

$$u_a(A_y, A) = v_y \beta s \left(A_o I_o + A_y I_y\right). \tag{19}$$

Note that u_a indicates the derivative of u with respect to the first argument, i.e., the individual activity level, but evaluated at the group average. This is a two equation system that jointly determines the equilibrium average activity levels within each group, which we denote by A_y^* and A_o^*. Equation (18) describes the "best-response" for the average activity of the old to alternative average activity levels of the young, and conversely for Equation (19).[5]

Dividing Equation (18) by Equation (19), we obtain

$$\frac{u_a(A_o^*, A^*)}{u_a(A_y^*, A^*)} = \frac{v_o}{v_y}. \tag{20}$$

This implies that $A_o^* < A_y^*$, the old are less active than the young, provided that they are more vulnerable, $v_o > v_y$, an assumption we make for the rest of our analysis.

To pave the ground for a comparative static analysis of a lockdown, we derive the slopes of these best-response functions. By analogy to the homogeneous case above, we define $u_o'(A_o, A) := u_a(A_o, A)$ and $u_o''(A_o, A) := u_{aa}(A_o, A) + u_{aA}(A_o, A)\omega$. While it is plausible that $u_{aA} \geq 0$ (as assumed above), it is no less plausible that $u_o''(A_o, A) < 0$, meaning that the direct effect $u_{aa}(A_o, A) < 0$ is dominating. We define $\sigma_o := -\left[u_o''(A_o^*, A^*)/u_o'(A_o^*, A^*)\right] A_o^* > 0$, which is the analogue to the elasticity σ for the homogeneous case. Moreover, we introduce the cross- elasticity $\epsilon_o := [u_{oy}'(A_o^*, A^*)/u_o'(A_o^*, A^*)]A_y^* \geq 0$, where u_{oy}' denotes the derivative of u_o' with respect to A_y. The inequality follows from the assumption that $u_{aA} \geq 0$.

Armed with these definitions, we now describe the slope of the best-response function for the old, evaluated at the equilibrium. Differentiating Equation (18) gives $[u_{aa} + u_{aA}\omega]\,\mathrm{d}A_o + u_{aA}(1-\omega)\mathrm{d}A_y = v_o \beta s \left(I_o \mathrm{d}A_o + I_y \mathrm{d}A_y\right)$, where all derivatives are evaluated at the equilibrium values A_g^* and $A^* = \omega A_o^* + (1-\omega)A_y^*$. Collecting terms and moving to log-changes, we arrive at

$$-\left(\sigma_o + \zeta_o\right) \mathrm{d}\log A_o = \left(1 - \zeta_o - \epsilon_o\right) \mathrm{d}\log A_y, \tag{21}$$

[5] 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.

where the elasticities σ_o and ϵ_o are as defined above and $\zeta_o := A_o I_o/(A_o I_o + A_y I_y)$. Approximating $\mathrm{d}\log A_o \approx \Delta \log A_o$ and $\mathrm{d}\log A_y \approx \Delta \log A_y$, we arrive at

$$\Delta \log A_o \quad = \quad -\eta_o \Delta \log A_y, \tag{22}$$

where $\eta_o := (1 - \zeta_o - \epsilon_o)/(\zeta_o + \sigma_o)$. The term η_o is the analogue for the case of a heterogeneous population to $\eta := 1/(1+\sigma)$ in the homogeneous case above. A perfectly analogous equation can be derived for the log-slope of group-y's best-response function with respect to A_o. Note that $\epsilon_o \geq 0$ means complementarity: with strict inequality, the marginal utility of the old increases as the young become more active. On this account, if group y becomes more active, individuals of group o will respond by becoming more active too. At the same time, however, a higher activity level A_y increases the risk of infection for group-o individuals, depending on $1 - \zeta_o$, the share of group y in the overall "infection-base" $A_o I_o + A_y I_y$. This will prompt individuals of group o to become more cautious, reducing A_o. We assume that this latter effect dominates, $1 - \zeta_o > \epsilon_o$, in which case $\eta_o > 0$ and the best-response function is downward-sloping. A corresponding assumption is made for the young. Taken together, these assumptions rule out extreme values of ζ_g. Note that η_g, like η in the homogeneous case above, is increasing in σ_o, the coefficient of risk aversion. But unlike η, η_g is not bounded from above by unity.

3.2 The effectiveness of lockdown policies

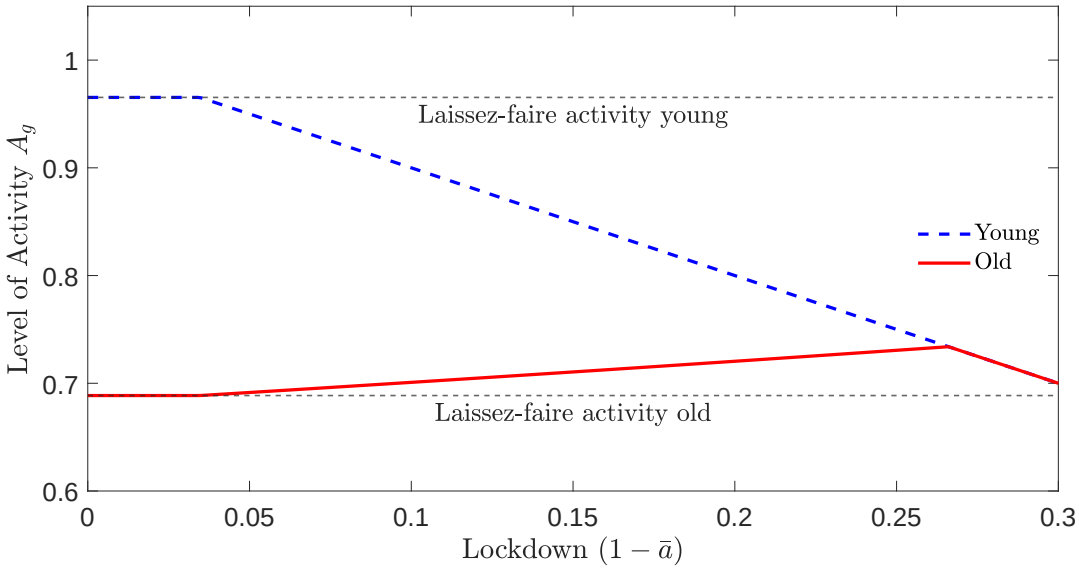
We are now in a position to study how a lockdown affects new infections, the case fatality and mortality. We assume the lockdown is introduced in a laissez-faire equilibrium with $A_y^* > A_o^*$. More specifically, the lockdown imposes a uniform maximum level of activity $\bar{a} < A_y^*$. We assume full compliance.

Proposition 3 (lockdown binding only for the young)**.**
a) *A lockdown $\bar{a} \in (a_\ell, A_y^*)$ is binding for the young but non-binding for the old, where $a_\ell = \frac{\eta_o+1}{\alpha^*\eta_o+1} A_o^* = \frac{\alpha^*(\eta_o+1)}{\alpha^*\eta_o+1} A_y^*$, with $\alpha^* := A_o^*/A_y^* < 1$.*

b) *As a result of this lockdown, the old will increase their activity level according to $\Delta \log A_o^* = -\eta_o \left(\bar{a}/A_y^* - 1\right) > 0$.*

Proof. The lockdown enforces a change in the activity level of the young equal, in percentage terms, to $\left(\bar{a}/A_y^* - 1\right) < 0$. Using A_o^ℓ to denote the desired activity level for

Figure 1: Quantitative illustration of how lockdown impacts activity levels



Notes: 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 7.

the old after imposition of the lockdown on the young, Equation (22) implies $\log A_o^\ell - \log A_o^* = -\eta_o \left(\bar{a}/A_y^* - 1\right) > 0$. But the lockdown will only allow them to respond in this way if $A_o^\ell < \bar{a}$. Writing $\log A_o^\ell - \log A_o^*$ as $A_o^\ell/A_o^* - 1$ and solving for A_o^ℓ, we obtain $A_o^\ell = -\eta_o \bar{a}\alpha^* + (1+\eta_o)A_o^*$. Setting $A_o^\ell = \bar{a}$ gives us an equation which determines the value a_ℓ for $\bar{a}$, which may be written as in part of the proposition. □

The intuition for Proposition 3 is that of a Peltzman (1975) effect. Intuitively, the range of lockdown stringency levels allowing for this type of adjustment depends on the degree of asymmetry of the initial equilibrium, captured—inversely—by α^*. This is seen from fact that the value a_ℓ is rising in α^*. The window of risk-compensating adjustment by the old closes if $\alpha^* = 1$, i.e., if heterogeneity vanishes. The fraction α^* also becomes larger with a higher value of η_o, which implies a lower degree of risk aversion σ_o.[6]

The situation is illustrated by Figure 1, based on a simple parameterization of the utility

[6]There is an interesting corollary of Proposition 3 relating to the degree to which the young are restricted by the lockdown $\bar{a}$. Ex ante, that degree may be measured through the ratio $A_y^*/\bar{a}$. Ex post, however, the degree to which the young will feel constrained by the lockdown is lower. The reason is that, given risk-compensating adjustment by the old, the young would choose an activity level lower than A^*.

function.[7] The equilibrium levels of A_o and A_y are depicted on the vertical axis, with the lockdown stringency measured by $1-\bar{a}$, on the horizontal axis. The dashed blue line holds for the young, with a flat segment for low lockdown-stringency levels $a_\ell > A_y^*$. Here the lockdown is not binding for the young, because the young, too, show some social restraint under laissez faire. This changes once $1-\bar{a}$ becomes smaller 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. Absent any lockdown, the old exercise much more social restraint than the young because they are more vulnerable. Yet once the lockdown binds for the young, the old start to raise their activity level—the Pelzman effect. At some point the activity levels of both groups are equalized: The two lines intersect once the lockdown stringency reaches the $1-a_\ell$. 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 unity.

Assessing the overall effect of the lockdown on infections, we must consider two opposing effects: a lower activity level of the young coupled with a higher activity level of the old. Without the lockdown, we have

$$\Delta i_g = \beta s A_g (A_o I_o + A_y I_y), \tag{23}$$

where Δi_g denotes the rate of new infections within group g. For the economy at large we have $\Delta I = \Delta I_o + \Delta I_y$, where $\Delta I_o = \omega \Delta i_o$. In the following, we use the operator Δ_L to denote changes brought about by the lockdown. For instance, $\Delta_L \log \Delta I_g$ denotes the lockdown-induced relative change in the aggregate rate of new infections. Obviously, we have $\Delta_L \log \Delta I_g = \Delta_L \log \Delta i_g$.

Proposition 4 (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* $\Delta_L \log \Delta i_o > \Delta_L \log \Delta i_y$.

[7] We chose the functional form $u(a, A_y, A_o) = \ln a + \omega \ln A_y + (1-\omega) \ln A_o - a + 1$. This specification satisfies all assumptions made above. Our calibration assumes that at the beginning of the policy period 2% of the population in each group are infected: $i_o = i_y = 0.2$. 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 $\bar{u}_{2,y} = 100$ and $\bar{u}_{2,o} = 10$ 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 of a 126 times larger mortality rate if infected for those 65 years or older. The parameter β is set to 1.

Proof. The proof is provided in the appendix. □

A lower activity level of the young means they pose less of a threat (to either group) if infectious and are less exposed to this threat if susceptible. This is the direct effect of the lockdown. The induced Peltzman effect implies that the exact opposite holds true for the old. Part a) of the proposition simply tells us that for the young the direct effect always dominates, since they are affected by the Peltzman effect only on account of a higher infection threat from the old. However, for the old, the Peltzman effect strikes twice since their higher activity level also renders them more exposed to their own higher threat. In addition, it also counteracts the lower threat they now face from the young. Hence, if strong enough the Peltzman effect may dominate for the old, as stated in part b). Part c) is a simple corollary of the asymmetry implied by parts a) and b).

Next, we look at the number of deaths relative to the population (mortality rate, denoted by m) and the number of deaths relative to the newly infected persons (case fatality, denoted by f). Under laissez-faire, we have

$$m = \delta_{Io}\omega\Delta i_o + \delta_{Iy}(1-\omega)\Delta i_y \quad \text{and} \quad f = m/\Delta I. \tag{24}$$

Proposition 5 (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 fatality rate.*
b) *It increases the mortality if and only if* $-\dfrac{\omega\Delta_L \log \Delta i_o}{(1-\omega)\Delta_L \log \Delta i_y} > \dfrac{\delta_{Iy}}{\delta_{Io}}\dfrac{A_y^*}{A_o^*}$.

Proof. The proof is provided in the appendix. □

Part a) of this proposition is an obvious consequence of $\Delta_L \log \Delta i_o > \Delta_L \log \Delta i_y$; see part c) of Proposition 4. This implies that the composition of those infected changes in favor of the old who are more likely to die from the disease. To understand part b), suppose that risk aversion is high whence $\Delta_L \log \Delta i_o < 0$. In this case, since we always have $\Delta_L \log \Delta i_y < 0$, the condition in part b) is clearly violated, so that the lockdown lowers mortality. But suppose risk aversion is low so that $\Delta_L \log \Delta i_o > 0$. Then, the condition will be violated if ω is sufficiently large and $\frac{\delta_{Iy}}{\delta_{Io}}\frac{A_y^*}{A_o^*}$ is sufficiently small due to strong heterogeneity, $\delta_{Iy} < \delta_{Io}$.

4 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.[8] 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 January 2022. 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_i are defined relative to 100,000 people. The monthly case fatality rate is denoted by 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 (Pachetti et al., 2020).[9]

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, namely the log of infections, the log of 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.

[8] Specifically, we include Austria, Belgium, Denmark, Finland, France, Germany, Greece, Ireland, Italy, Netherlands, Norway, Portugal, Spain, Sweden and the United Kingdom.

[9] Formally, we use:

$$\text{CFR}_t = \frac{\sum_{d=1}^{N} \text{Deaths}_{d+14}}{\sum_{d=1}^{N} \text{Infections}_d}, \tag{25}$$

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.

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

$$x_{it} = \gamma_1(1 - V_{it-1})s_{it-1} + \gamma_2 V_{it-1}s_{it-1} + \eta v_{it-1} + \beta X_{it} + \theta_i + u_{it}. \tag{26}$$

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 η, 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. The first line reports the estimated coefficient 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 lockdown 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 $[0, 1]$, rather than between zero and one, as our indicator variable V_{it-1}. Here again we detect a striking pattern. Contrary to commonsense 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 they trigger a Peltzman effect which may limit or even offset their direct medical effect.[11] Nor does the presence of

[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.

[11] Consistent with this interpretation, Guo et al. (2021) provide cross-country evidence suggesting that

Table 1: Effect of public policy measures

	US states			Western Europe		
	Infections$_t$	Deaths$_t$	CFR$_t$	Infections$_t$	Deaths$_t$	CFR$_t$
γ_1	-1.025***	-0.726**	0.630**	-1.565**	-0.448	1.251**
	(-3.87)	(-2.75)	(-2.97)	(-3.31)	(-0.73)	(-2.64)
γ_2	-1.275***	-1.010***	0.152	-1.815***	-1.120*	0.769*
	(-4.70)	(-4.48)	(-0.65)	(-3.48)	(-2.39)	(-1.97)
η	0.415*	0.321*	-0.237	-0.377	-0.418	0.181
	(-2.51)	(-2.14)	(-1.49)	(-1.47)	(-1.76)	(-1.06)
Controls	yes	yes	yes	yes	yes	yes
State FE	yes	yes	yes	yes	yes	yes
N	988	975	975	282	277	277
r2	0.812	0.790	0.500	0.795	0.773	0.707

Notes: parameter estimates for model (26); monthly observations 2020:03–2022:01; 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;
* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

a Peltzman effect suggest vaccinations is a welfare reducing policy.

5 Conclusion

Our analysis highlights the role of Peltzman effects—or risk-compensating behavioral adjustment—for the effectiveness of public policies aimed at reducing the health hazard from a Covid-19-like pandemic. With almost all public policies implemented during the Covid-19 pandemic over the past two years, policy makers have at some point been frustrated by disappointing results. Our analysis demonstrates that risk-compensating behavioral adjustment may lie behind this. Our analytical framework extends the well-known SIR-dynamics of virus transmission to include an incentive for social activity that fosters virus transmission. Weighing the marginal benefit of present social activity against the marginal cost deriving from a higher likelihood of infection and earlier death, individuals adjust to the emergence of the pandemic as such, but more importantly, also to public policies aimed at reducing the risk of infection and/or the risk of death caused by an infection. We analyse two types of public policies, vaccination and lockdowns. Our theoretical analysis suggests the presence of Peltzman effects: People become less careful in their behavior as a result of risk-reducing policies and thus erode the effectiveness these policies. Such effects likely reduce the apparent effectiveness of policies. Our empirical analysis detects

vaccination increases social mobility.

pattern in the data that suggest these effects are non-negligible in magnitude.

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A Appendix. Proof of Propositions

Proposition 2 states:
Rolling out vaccination as described above affects aggregate welfare as follows:
a) *If the social externality weakly dominates the infection externality, $u_A(A^*, A^*) \geq \beta S A^* I$, then vaccination is unambiguously welfare increasing.*
b) *If the infection externality dominates the social externality, then the welfare effect of vaccination* ***may*** *be negative.*
c) *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

$$W(A^*, A^*) = u(A^*, A^*) + 1 - \delta(A^*). \tag{1}$$

In a vaccination scenario, we have

$$\Delta_V \delta(A^*) = \Delta_V(\delta_I \Delta I) = \Delta I \Delta \delta_I + \delta_I \frac{\partial \Delta I}{\partial \beta} \Delta\beta + \delta_I \frac{\partial \Delta I}{\partial A} \Delta A^*. \tag{2}$$

The first two terms capture the direct vaccination effect, with $\Delta\delta_I < 0$ and $\Delta\beta < 0$ while the third captures behavioral adjustment governed by Equation (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 $\partial \Delta I / \partial \beta = \beta A^{*2} SI > 0$. The welfare change emerges as

$$\begin{aligned} \Delta W \quad \approx \quad & \left[u_a(A^*, A^*) + u_A(A^*, A^*) - \delta_I 2\beta S A^* I\right] \Delta A^* \\ & - \left[\Delta I \Delta \delta_I + \delta_I \left(A^{*2} SI\right) \Delta\beta\right] \end{aligned} \tag{3}$$

The first-order condition (8) allows us to rewrite the bracketed term in the first line above as $[u_A(A^*, A^*) - \beta \delta_I S A^* I] \Delta A^*$. The expression in the second line is unambiguously positive. Moreover, we know that vaccination increases A^*.
Adding the first-order condition for individual behavior it then follows that under the condition stated in **part a)** of the proposition the welfare effect must be positive. By the same token, it may be negative if this condition is violated, wich proves **part b)**.
As to **part c)**, note that the net effect of vaccination on new infections is $2\beta S A^* I \Delta A^* + A^{*2} SI \Delta\beta$. If this is equal to zero (part a), we are left with $\Delta W = [u_a(\cdot) + u_A(\cdot)] \Delta A^* -$

$\Delta I \Delta \delta_I$, which is clearly positive. The same is true, a fortiori, if the net effect on the rate of new infections is negative. □

Proposition 4 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 Equation (23), the lockdown-induced relative changes in the rates of new infections in the two groups are

$$\Delta_L \log \Delta i_o = \Delta \log A_o + \zeta_o \Delta \log A_o + (1-\zeta_o)\Delta \log A_y \quad (4)$$
$$\Delta_L \log \Delta i_y = \Delta \log A_y + \zeta_o \Delta \log A_o + (1-\zeta_o)\Delta \log A_y \quad (5)$$

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 Equation (22). Inserting this, we obtain:

$$\Delta_L \log \Delta i_o = [1 - \zeta_o - (1+\zeta_o)\eta_o] \Delta \log A_y \quad (6)$$
$$\Delta_L \log \Delta i_y = [2 - \zeta_o(\eta_o + 1)] \Delta \log A_y \quad (7)$$

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 (6) and (7) 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 $\eta_o > 0$. □

Proposition 5 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

$$f = \delta_{Io} \frac{\Delta I_o}{\Delta I} + \delta_{Iy} \frac{\Delta I_y}{\Delta I} = \delta_{Iy} + (\delta_{Io} - \delta_{Iy}) \frac{\Delta I_o}{\Delta I}, \tag{8}$$

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

$$\Delta_L f = (\delta_{Io} - \delta_{Iy}) \Delta_L \frac{\Delta I_o}{\Delta I} \tag{9}$$

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 $\delta_{Io} > \delta_{Iy}$, $\Delta_L f$ 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 4 that $\Delta_L \log \Delta i_o > \Delta_L \log \Delta i_y$. Hence, this condition is fulfilled.

Part b): Differentiating equation for m in (24) yields

$$\Delta_L m = \delta_{Io} \omega \Delta i_o \Delta_L \log \Delta i_o + \delta_{Iy} (1-\omega) \Delta i_y \Delta_L \log \Delta i_y. \tag{10}$$

Setting $\Delta_L m > 0$, we obtain

$$\delta_{Io} \omega \Delta_L \log \Delta i_o + \delta_{Iy} (1-\omega) \frac{\Delta i_y}{\Delta i_o} \Delta_L \log \Delta i_y > 0. \tag{11}$$

In view of Equation (14), 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. □