Optimal Age-based Policies for Pandemics: An Economic Analysis of Covid-19 and Beyond*

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Abstract

This paper investigates the importance of the age composition for pandemic policy design. To do so, it introduces an economic framework with age heterogeneity, individual choice, and incomplete information, emphasizing the value of testing. Calibrating the model to the US Covid-19 pandemic reveals an 80% reduction in the death toll due to voluntary actions and the lockdown implemented in the US. The optimal lockdown, however, is more stringent than what was implemented. Moreover, the social planner follows an asymmetric approach by locking down the young relatively more than the old. We highlight the role of testing in reducing deaths, lowering economic costs, and allowing for a less restrictive lockdown. A social planner would choose a faster ramp-up in testing capacity relative to what occurred in the US. We use the framework to provide insights into pandemics caused by different viruses (including the Spanish flu), and underline the influence of economic conditions on optimal policies.

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1 Introduction

Deciding how governments should implement lockdown measures in response to a pandemic is a complex challenge, given the unique characteristics of each disease. Some diseases are highly contagious, while others are more lethal. Furthermore, the impact of these diseases can vary significantly across different population groups. Certain diseases may disproportionately affect men, women, or children. Many infectious diseases, particularly those caused by viruses, are much more deadly for the elderly than the young—e.g., influenza, swine flu, SARS and most recently Covid-19. This strong age gradient in the effects of these diseases raises a number of important questions: How much voluntary protective behavior do different age groups engage in? What does that imply for policy that aims to balance the economic and health effects of a pandemic? And how would optimal policy be different for a disease with a less steep age gradient? This paper develops an economic framework designed to study these questions.

Section 2 describes our economic model of the pandemic that features *age heterogeneity* and *individual choice*, allowing agents to choose rationally how much social distancing to undertake, taking into account future infection risk and the chance of a vaccine arrival. Social distancing provides protection, but comes at the cost of forgone earnings and diminished leisure enjoyment. These can only be partially substituted by teleworking and safe leisure activities. The deadliness of the disease, the need to earn a living and the natural death probability differ between the working age population and the elderly. The government can curb infections by imposing restrictions on outside activities separately by age to decrease the number of interactions.

One of the novelties of our framework is the fact that individuals can be uncertain about their health status, which renders testing valuable. As individuals can also contract the common cold, initial symptoms leave individuals and the government with *incomplete information* whether they are infected with Covid (we call this the "fever" state). This modeling choice allows us to introduce uncertainty in a tractable way. By assuming that both diseases generate similar initial symptoms and occur simultaneously, we avoid a large increase in the state space that typically arises in models with incomplete information. In our baseline, this uncertainty lasts one period, which is a simplification, but it captures an important feature of the data: individuals are uncertain about their infection status precisely when they are most infectious. This timing makes testing valuable, as it allows the planner to identify and isolate infected individuals at the moment when they pose the greatest risk to others. As a result, testing plays a central role in the model by enabling more targeted interventions that substitute for broader lockdown measures.

To quantitatively assess the impact of different policies, we parameterize our model

using data from the Covid-19 pandemic in the US, as detailed in Section 3. The calibration targets pre-pandemic moments of time allocation across different age groups as well as demographic information. Moreover, we target Covid-specific characteristics such as age-specific hospitalization and death probabilities as well as the lockdown imposed by the US government in the first year of the pandemic. The calibration fits the time series of overall deaths and the rise in home hours well. On top of this, the parameterized model also matches several non-targeted moments: the age-specific mortality rates and changes in time at home, the time series of aggregate employment and the positivity rate of tests.

We first use the calibrated model to assess the importance of behavioral changes in Section 4. In the benchmark, outside activities are restricted both by voluntary protective behavior and by the government-imposed lockdown. This lower activity leads to a death toll 80% lower than in a purely epidemiological model where individuals do not adjust their behavior. Voluntary precautions play a large role. In a no-lockdown equilibrium, older individuals shield themselves substantially. The young also reduce work and outside leisure, but much less so due to their lower risk of dying and the need to earn a living. Though the young can telework, this is a lower-productivity activity. The death toll in this no-lockdown world is 65% lower than in the epidemiological scenario.

We solve for the optimal lockdown policy in Section 5. The social planner's optimal lockdown is stricter than what was implemented in the US, and reduces deaths across both age groups. Increased restrictions predominantly impact the young, who curtail interactions, while the old gain more outdoor time due to a lower threat of infection. This asymmetry is intentional, driven by endogenous behavior. In the no-lockdown equilibrium, the young tend to neglect taking strong precautions to limit the spread of the disease due to low personal risk, leaving the old to bear an undue burden. The optimal lockdown is very stringent in the first few weeks to quickly bring the disease under control. Then the planner maintains restrictions at intermediate levels, tightening them around winters when infections rise. Abstracting from seasons, the stringency of lockdowns gradually declines over time as testing capacity ramps up and enables a more targeted approach to isolating infected individuals.

Given uncertainty about individuals' health status (the "fever" state), testing plays a central role in our framework. Section 6 explores several aspects of testing. First, testing alone does not eliminate Covid-19, but significantly alleviates its impact. More importantly, testing allows the planner to substitute broad lockdowns with targeted isolation of infected individuals, leading to a less restrictive policy. As a result, the optimal lockdown allows for higher mobility and smaller GDP losses. We also analyze how quickly testing capacity should be built up and find that a social planner would choose a much faster ramp-up than what occurred in the US. This faster expansion of

testing capacity further relaxes lockdown measures and increases output.

Our main calibration uses data from the Covid pandemic in the US in the early 2020s. However, our framework is general enough to study policies for different pandemics, which we do in Section 7. We start by recalibrating the model for the Spanish flu pandemic of the 1910s. Optimal lockdown policy would entail milder reductions in social interactions for the Spanish flu, even though the overall death rate of this disease was higher. This result is based on a combination of factors that were different one hundred years prior: a younger population, the inability to telework, a different virus, the unavailability of tests, etc. We decompose the effects of each of these factors and also study how lockdowns should be designed for diseases with different levels of infectiousness and deadliness. Several lessons emerge. Stringent lockdowns are warranted when the basic reproduction number (R_0) is high, but less so when only the case fatality rate (CFR) is elevated. The age gradient is a crucial factor: if the CFR is high among the young, a sizable and active group, fewer additional restrictions are necessary due to substantial voluntary precautions. Economic conditions have a big impact on the optimal lockdown. In scenarios where the older population is smaller, life expectancy is lower, or teleworking is easy, a less restrictive policy is optimal. Importantly, the optimal policy may not completely prevent all deaths, and the welfare benefits can be unevenly distributed across age groups.

Overall, by integrating age-specific behaviors, testing strategies, and lockdown policies into an economic framework, this paper offers crucial insights for shaping policies to minimize the economic and health impacts of pandemics. Before laying down the framework in the next section, we end this introduction with a brief literature review.

1.1 Literature Review

This paper contributes to the literature that combines epidemiological models (e.g., Kermack and McKendrick (1927)) with equilibrium behavioral choice. In economics, efforts to incorporate behavioral responses to disease progression through equilibrium models have mostly been theoretical. Such works have long pointed out a negative externality of too little prevention efforts by self-interested agents; see, e.g., Kremer (1996) for SI models, Quercioli and Smith (2006) and Chen (2012) for SIR models, and more recently Toxvaerd (2019). These studies consider homogeneous populations, though Kremer (1996) also considers heterogeneous preferences for risky activities. In our setting, differences in activity are partially a consequence of different death rates.[1]

[1]Other work that considers heterogeneity includes Galeotti and Rogers (2012) who study two identical populations but with non-random mixing, and work on transmission in networks where individuals occupy different positions (Acemoglu, Malekian, and Ozdaglar 2016).

There are few quantitative economic models of disease transmission that predate Covid. Greenwood et al. (2019) develop a heterogeneous-agent choice-theoretic equilibrium model for the HIV/AIDS epidemic to analyze different mitigation policies. Within this framework, Greenwood et al. (2017) explore particular channels of selective mixing by relationship type, while Greenwood et al. (2013) allow for incomplete information in infection status. In these works, the behavioral response of agents is crucial for the results of different policies. Chan, Hamilton, and Papageorge (2016) argue in a structural model that behavioral adjustments matter for the evaluation of medical innovations. Keppo et al. (2021) use a calibrated homogeneous-agent model to argue that a substantial behavioral elasticity is necessary to match different epidemics.

In the great influx of recent economics papers studying different aspects of Covid-19, most consider homogeneous populations. Some analyze optimal containment policies in a standard epidemiological model that trade off economic well-being of living individuals versus lost lives (e.g., Alvarez, Argente, and Lippi (2021)), while others first feature optimizing agents and then introduce a planner who improves their ex-ante utility (e.g., Eichenbaum, Rebelo, and Trabandt (2021), Farboodi, Jarosch, and Shimer (2021), Garibaldi, Moen, and Pissarides (2020), McAdams (2020), Vandenbroucke (2021)).[2] The main insight of these papers is that private incentives to protect oneself slow the disease and save lives, and optimal policy follows two phases: possibly a short strict start and then near-constant restrictions that keep the reproductive number close to unity. Such a policy keeps the disease at a near-constant low level and preserves lives up to the point of relief through vaccination. In our setting, the optimal lockdown begins with strict restrictions that quickly reduce initial infections. The planner then maintains a stable level of restrictions, tightening them during winters to counter seasonal surges. As testing capacity expands, the planner can decrease the stringency of lockdowns and still keep the disease under control. We analyze the various factors that influence the level of lockdowns, the amount of lives saved, the division of the burden across agents, and how this would vary under different diseases, which has not been systematically studied in the previous literature.

The papers in the preceding paragraph focus on developed countries and abstract from testing. Extensions to Eichenbaum, Rebelo, and Trabandt (2021) comprise Almås et al. (2023) who argue that confinement policies should be laxer when parametrized to developing countries, unless the population is close to their subsistence constraint where policies are less effective. Our decomposition allows us to study a stylized developing country as part of our analysis. Despite a laxer lockdown and increases in deaths for each age group, the aggregate death toll is lower due to a different age

[2] Many extensions to the basic planning problem with an epidemiological model exist, such as accounting for waning immunity (see, e.g., Giannitsarou, Kissler, and Toxvaerd (2021)), or slow vaccine rollout (see Garriga, Manuelli, and Sanghi (2022)).

composition, which is hidden in usual representative-agent settings. Melosi and Rottner (2023) show that contact tracing increases initial lockdowns, though it assumes for simplicity that non-tested individuals assume they are healthy. Other papers introduce uncertainty about one's infection status and the role for testing (Berger et al. (2020), von Thadden (2020), Piguillem and Shi (2022)). Only Eichenbaum, Rebelo, and Trabandt (2022) combine testing with individual choices about social distancing as in our paper, but with homogeneous agents and only a short and stylized lockdown. We study the implications of testing for the optimal lockdown, and also consider the problem of a social planner that chooses the optimal ramp-up of testing capacity.

Our paper differs from the papers above along a number of dimensions, including the broader view beyond Covid and the inclusion of a large number of factors such as teleworking and testing, but one of the biggest differences is in our focus on heterogeneity by age leading to different risk groups. The medical literature is long aware of an age gradient in the severity of infectious diseases in adulthood. Recently, Glynn and Moss (2020) conducted a meta-analysis across 142 studies covering 32 different diseases (19 viral and 13 bacterial) and sum up: "For most infections clinical severity [..] rises in adulthood and more steeply into old age."[3] Covid-19 is no exception and features a strong age gradient. Still, there are only a few other studies in economics that incorporate age differences: Favero, Ichino, and Rustichini (2020) and Gollier (2020a) argue that re-opening should focus on the young while shielding the old, Gollier (2020b) argues that herd immunity has less deaths when built on the young, Glover et al. (2023) analyze how a blanket lockdown affects young and old agents differently and how this leads to disagreement on optimal policy, Alon et al. (2020b) argue that shielding the old while the young work is even more important in developing countries, Acemoglu et al. (2021) characterize the optimal frontier between GDP and lives lost in a model with three age groups and argue that the tension between lives saved vs GDP lost can be best addressed with targeted group-specific policies that confine the old.[4] These papers assume that individual behavior can be finely adjusted directly through policy, but is otherwise fixed (or has a coarse dimension such as which sector to work in), and usually trade off lost production vs lives saved. This implies large benefits to confinement of the elderly who are at risk but do

[3] Glynn and Moss (2020) also study differences of severity during childhood, from which we abstract in our paper.

[4] Glover, Heathcote, and Krueger (2022) extends Glover et al. (2023) to analyze whether the young or the old should be vaccinated first. Brotherhood et al. (2022) extend our framework to study heterogeneity on income and housing arrangements in developing countries (including slums), and spatial heterogeneity has also attracted other papers that abstract from age heterogeneity (see Bognanni et al. (2020), Fajgelbaum et al. (2021), Fernández-Villaverde and Jones (2022)). Akbarpour et al. (2023) uses a mechanism design perspective to study theoretically how available vaccines should ideally be allocated over time to heterogeneous agents. Kaplan, Moll, and Violante (2020) consider heterogeneity in terms of occupational exposure and take into account the ability to telework and self-insure and for the government to redistribute. They consider scenarios rather than optimal policy.

not produce.

Our work focuses on voluntary behavioral change which has been empirically found to be a large driver behind social distancing (e.g., Maloney and Taskin (2020)) and how this interacts with age-specific policies. In our model the old confine themselves voluntarily and further mandatory confinement lowers their welfare. This happens because the additional leisure benefit of social interactions is further restricted, which have largely been abstracted from in the literature on age heterogeneity. While some countries have implemented harsh confinements on the elderly in line with the recommendations of the papers above, Altindag, Erten, and Keskin (2022) empirically documents that this has led to worsened mental health outcomes linked to social and physical isolation in line with the lost leisure benefits modeled in our framework.

Boppart et al. (2025) shares this concern for the leisure of the elderly and individual choice, and their model is closest in structure to ours.[5] They abstract from uncertainty about infection status, testing and seasons. This matters for outcomes. While they argue that full suppression of Covid is rather parameter-dependent, a no-Covid policy that suppresses nearly all infections comes out robustly in our setting.[6] In our systematic investigation of other disease parameters and economic conditions we do find cases where suppression is no longer optimal.

Eichenbaum et al. (2024) also lay out a model with optimizing but possibly misinformed consumers of different ages during the Covid pandemic, but do not consider their respective externalities and optimal government intervention which is the focus of this paper. They also abstract from testing, which is much more common in later waves and—when coupled with some isolation of positive individuals—reduces the risk in the economy. It is one of the elements that allow us to rationalize more risk-taking despite higher Covid incidence in later waves even in a rational expectations equilibrium without misinformation.[7]

[5] Other papers with voluntary behavioral change and multiple groups typically consider scenarios rather than optimal policy. For example, Giagheddu and Papetti (2023) allow for an endogenous contact matrix but restrict optimal lockdown policy to consumption choices while providing a scenario analysis for lockdowns on social interactions. They abstract from teleworking, testing, or validation of the size of their effects during Covid, and do not assess welfare of their social lockdowns because they directly change utility parameters. Dizioli and Pinheiro (2021) consider some regular and some time-constrained individuals, and allow unawareness of infection status. They simulate short and long lockdowns but not optimal policy.

[6] Boppart et al. (2025) highlight the timing of vaccine arrival and the value of a statistical life as key determinants of disease suppression. In our setting the planner avoids nearly all deaths across many scenarios including much later vaccine arrival and much lower value of statistical life. One of the differences is the modeling of testing capacity and associated isolation. Boppart et al. (2022) builds on Boppart et al. (2025) to study the value of vaccination and shows it is high and increasing with age.

[7] Eichenbaum et al. (2024) differs in many ways from our paper, including the nature of risk-taking and differences in transmission mechanisms (in our setting transmission risk depends on own risk-taking, disease incidence, and risk taking of other infected individuals, while they abstract from the latter, turning transmission from quadratic into linear in risk-taking). The US data also displays a larger first peak and higher lockdowns in first peak relative to later ones, compared to the Portuguese data they rely on, making it easier to explain the data with rational expectations.

2 Model

This section describes the model with a focus on Covid. However, we will see in the next few sections that the framework is general enough to study different diseases. The general setup is in discrete time. The economy is populated by a continuum of ex-ante identical agents of two types: young y and old o, so that age $a \in \{y, o\}$.[8] Individuals work, enjoy leisure outside the home and domestic hours. In the presence of the coronavirus, denote the agent's health state by j. A susceptible agent is denoted by $j = s$. By spending time outside the house, the agent may catch a disease, which may be Covid-19 or a common cold.[9] Both lead to mild "fever" symptoms.[10] There is a testing capacity K_t that gets allocated at random among agents with fever symptoms, leading to probability $\xi_t(a)$ of getting tested. With complementary probability, they are not tested and are therefore unsure about the source of their symptoms. While some of these are truly infected (state f_i for "fever-infected") and others are not (state f_s for "fever-susceptible"), they do not know this and can only discern state $j = f$ for "fever." A tested individual knows for sure whether they are infected with Covid. For simplicity, assume that non-tested infected individuals discover their state after one period.[11] Denote by $j = i$ the state where an agent knows they are infected with Covid-19. Each of these elements are now explained in more detail.

Disease progression is mechanical once an individual contracts Covid and does not depend on the agent's subsequent choices. The individual can develop more serious symptoms that require hospitalization, a state denoted by $j = h$. This happens with probability $\alpha(a)$. An agent being treated in a hospital can die with probability $\delta(a)$, on top of the natural death probability $\overline{\delta}(a)$ that exists even in the absence of the pandemic. An infected agent recovers from the disease with probability $\phi(0, a)$, which changes to $\phi(1, a)$ for a hospitalized agent. All these parameters are age-dependent, allowing us to capture the age difference in the severity of the disease. If the agent recovers, they become immune (or resistant) to future infections, a state denoted by $j = r$. Thus, the set of all health states is $j \in \{s, f, i, h, r\}$.

[8] The model can easily accommodate any number of age groups, but we focus on just two. Acemoglu et al. (2021) highlight large benefits from separately targeting the elderly and the working-age population but little further improvements in sub-dividing those who work. Moreover, we intentionally limit heterogeneity to age to provide a transparent picture, though computationally other dimensions of heterogeneity could be handled.

[9] Our model features seasonal transmissability of both Covid and the common cold, but abstracts from different variants of Covid-19. These could be introduced as in Brotherhood and Santos (2022).

[10] Ours is one of the few models in the literature that captures partial information—rather than full knowledge or no knowledge about infection state. The specific modelling assumption is for tractability, and evidently does not capture truly asymptomatic cases. The US Center for Disease Control estimates 85% of Covid-19 cases to be symptomatic (97.1 million symptomatic cases out of 114.6 million total cases, and similarly for the over-18-year-old 0.74 million symptomatic out of 0.88 total cases) as of April 2021. See CDC (2021). This suggests that our setting might capture the vast majority of cases.

[11] Section 6 relaxes the assumption that non-tested agents learn their health status after one period.

A vaccine arrives with probability $\chi(\mathcal{V}_t)$ each period. If a vaccine is already present at the start of a period, we denote this as $\mathcal{V}_t = 1$, where we omit the time subscript if it is obvious. In this case it will persist, i.e., $\chi(1) = 1$. If a vaccine is not present ($\mathcal{V}_t = 0$) at the beginning of a period, its arrival suppresses infections this period and in the future. That means that we abstract from slow rollout. A vaccine does not heal those that are already infected.

Infections are endogenous and may happen to susceptible people when they leave their house for either work, n, or leisure, ℓ. Taking the risk Π_t per unit of time outside as given, the infection probability for a susceptible individual in a period is

$$\pi(n + \ell, \Pi_t, \mathcal{V}_t) = (n + \ell + \underline{m})\Pi_t(1 - \mathcal{V}_t).$$

The longer the individual spends outside, the riskier it gets, though some contacts are unavoidable, as captured by $\underline{m}$.[12] How risky the environment is (i.e, the value of Π_t) is determined by equilibrium behavior and seasonality as described below. The last term simply states that transmissions are suppressed when a vaccine is available.

Uncertainty without testing arises because people can also catch a common cold, which is transmitted similarly to Covid and triggers uncertainty with probability

$$\pi^*(n + \ell, \Pi_t^*) = (n + \ell + \underline{m})\Pi_t^*,$$

where Π_t^* follows the same seasonality as Covid; i.e., $\Pi_t^* = \psi_t \Pi^*$ for an exogenous parameter Π^* and a seasonal component ψ_t.

The probability that the agent catches either disease is

$$\pi_f(n + \ell, \Pi_t, \Pi_t^*, \mathcal{V}_t) = \pi(n + \ell, \Pi_t, \mathcal{V}_t) + \pi^*(n + \ell, \Pi_t^*),$$

which implicitly assumes that these are mutually exclusive events.[13] If this happens and the agent is not tested (probability $1 - \xi_t(a)$), the agent enters next period $t + 1$ in the fever state $j = f$, in which they cannot distinguish between the common cold and Covid, assigning probability

$$\Pi_t/(\Pi_t + \Pi_t^*) \tag{1}$$

to having Covid. They assign a high probability to Covid when this disease is very prevalent (Π_t is high) but not otherwise. Importantly, agents do not need to condition on their past behavior as a state variable when forming this belief, since behavior affects the probability of both diseases in a similar way. This feature keeps the state space

[12] This could include unavoidable visits to one's home, such as those introduced by children for the young or nursing needs for the old. Allowing for some exogenous transmissions is common in the literature; e.g., Eichenbaum, Rebelo, and Trabandt (2021) and Hur (2023).

[13] This is a good approximation when the probability of either event is sufficiently small, in which case the chance of getting both becomes negligible.

compact and the model tractable while still capturing uncertainty about infection status at the time when individuals are most infectious. If they are tested (probability $\xi_t(a)$), they immediately learn whether they are infected ($j = i$) or not ($j = s$). Otherwise, they learn at the end of the period whether the fever was due to Coronavirus or not, which again keeps the state space tractable.

To recap, individuals enter a period with health state j and make their consumption and leisure choices. Those who started in state $j = f$ find out whether or not they truly were infected already at the beginning of the period. A vaccine may arrive, and then people's health state changes as a consequence of their actions in this period, and new fever symptoms can arise. Individuals then might get tested. They might die of natural reasons, and hospitalized individuals might also die of Covid. The timing implies that individuals can be confused about their health state for multiple periods, because they can experience a fever multiple periods in a row. The actual infectious period under Covid is short, but the one-period difference between information provision through testing and no testing implies confusion exactly when individuals are most infectious to others. Testing also allows the government to isolate individuals, which we assume impossible for fever-individuals that are not tested.

For production and leisure, each agent is endowed with one unit of time per period. This can be divided into outside work hours n, teleworking hours v, leisure outside the house ℓ and hours at home d ("domestic" leisure). Agents enjoy utility from consumption c, a composite leisure good when they leave home g, and domestic hours d. The good g is produced using hours ℓ and buying "intermediate" goods x according to $g = g(x, \ell)$. We normalize the utility after death to zero and capture the bliss from being alive through the parameter b. The utility function is given by:

$$u(c, g, d, v) = \ln c + \gamma \ln g + \lambda \ln d + b,$$

where γ and λ are positive constants. Agents discount the future at a common factor $\tilde{\beta}$, but since the natural survival probability $\Delta(a) = 1 - \overline{\delta}(a)$ is age-specific, their effective discount factor is $\beta(a) = \tilde{\beta}\Delta(a)$.

The time constraint is $\tilde{n}+\tilde{\ell}+d+v = 1$, where in the absence of government lockdowns $\tilde{n} = n$ and $\tilde{\ell} = \ell$, so that the sum of all time allocations in a period add to the unit amount of time that agents have.

Government lockdowns are represented as a tax $\tau_t(j, a, \mathcal{V})$ on time spent outside the house, and $\tilde{n}$ and $\tilde{\ell}$ represent the pre-tax time used for outside work and leisure.[14]

[14]We do not model separate taxes on time spent working or socializing because our modelling assumptions imply that both have equal externalities on the rest of the economy and so the planner does not gain by differentiating between them. An alternative way to model lockdowns is via a utility penalty on activities outside the house. See Brotherhood et al. (2020) for details. Effects of either approach turn out to be very similar.

While the taxes can condition on the time period and on age, it seems unlikely that a planner has the information and enforcement capabilities to tailor lockdowns separately to individuals with rather similar appearance such as the susceptible, those with mild symptoms, or the recovered. This can be captured by assuming that $\tau_t(s, a, \mathcal{V}) = \tau_t(f, a, \mathcal{V}) = \tau_t(r, a, \mathcal{V})$. Allowing for this is the main reason to introduce taxes rather than letting the planner directly control agents' choices.[15] Without it, the planner could trivially control the disease through targeted heavy taxes on those who are infected with mild symptoms, thus avoiding the large-scale lockdowns that characterize the actual response to the pandemic.

The policy implies that an individual in state $j \in \{s, f, i, h, r\}$ who aims to spend $\tilde{n}$ units of time at work gets paid only for $n = \tilde{n}(1 - \tau_t(j, a, \mathcal{V}))$ units, i.e. loses $\tau_t(j, a, \mathcal{V})\tilde{n}$ units of outside work. The same applies to leisure time outside the house. The difference $\tilde{n} - n$ and $\tilde{\ell} - \ell$ is not lost to the economy, but is rebated lump-sum through transfers $T_t(j, a, \mathcal{V})$. This transfer ensures that taxes alter choices and therefore well-being but do not by themselves create deadweight losses.[16] The resulting time constraint is:

$$v + d + (n + \ell)/(1 - \tau_t(j, a, \mathcal{V})) = 1 + T_t(j, a, \mathcal{V}). \tag{2}$$

The monetary budget constraint is determined by the wage per unit of time worked outside, w. The wage rate for teleworking hours depends on the amount of telework the agent performs, according to $w\iota(v)$, where $\iota(v) = \iota_0 - \iota_1 v$. The first few activities moved to telework do not carry significant wage penalties. However, as more work is moved to the home, it becomes more costly. This creates a clear trade-off with teleworking: the more one works from home, the less likely it is to catch a disease but the lower is the wage. Old agents live off retirement savings amounting to a fixed income

[15] A usual approach to a planning problem is to let the planner make choices on behalf of the agents, and to compare this with the choices that the agents would make themselves. This can be captured through type-specific taxes, and we will use such an unconstrained planning problem as a comparison later. The advantage of taxes is that they offer a representation that can relax the assumption that the planner has the health information and enforcement capabilities to set different lockdowns for individuals with rather similar outward appearance. The tax thus achieves a uniform government treatment of, e.g., the susceptible and those with mild symptoms, while allowing each of them to react to this treatment in potentially different ways.

[16] These lockdown taxes are intended to serve as a "proxy for any containment measures aimed at reducing social interactions" (Eichenbaum, Rebelo, and Trabandt 2021). In real life, lockdowns restrict several outside activities, like doing yoga in a studio, going to the movies, or eating in a restaurant. In response, individuals may engage in some alternative activities in their home, like doing yoga in their living room, watching streaming services, and cooking at home. Time is thus not lost; it is reallocated in a second-best fashion from the point of view of the individual. This is guaranteed by having the time transfer depend on the state of the individual: $T_t(j, a, \mathcal{V})$. More restrictions in outside activity are represented by higher taxes in our model, which leads individuals to change their time allocation in response to the policy. Representing distortions to the first order conditions in optimal choice problems as "tax wedges" that are lump-sum rebated has been widely adopted in the economics literature going back to seminal papers such as Chari, Kehoe, and McGrattan (2007) and Hsieh and Klenow (2009). Our first order conditions (29) and (30) in Appendix E illustrate the same purpose for our paper.

$\bar{w}$. Total earnings can then be written as follows:

$$w(a,n,v) = \begin{cases} w[n+\iota(v)v] & \text{if } a = y \\ \overline{w} & \text{if } a = o, \end{cases}$$

and aggregate output of the economy in a given period can be computed by $w(y,n,v)$ times the number of young agents in the economy (see Appendix A for details). The budget constraint of each individual agent is then given by:

$$c + x = w(a,n,v). \tag{3}$$

The value function for susceptible (s) agents of age a in period t that starts with vaccine availability $\mathcal{V}$ is given by:

$$\begin{aligned} V_t(s,a,\mathcal{V}) = \max_{c,x,n,v,\ell,d} & u(c,g(x,\ell),d,v) + \\ & \beta(a)[1-\pi_f(n+\ell,\Pi_t,\Pi_t^*,\mathcal{V}) + \pi_t^*(n+\ell,\Pi_t^*)\xi_t(a)]W_{t+1}(s,a,\mathcal{V}|s) + \\ & \beta(a)\xi_t(a)\pi(n+\ell,\Pi_t,\mathcal{V})W_{t+1}(i,a,\mathcal{V}|s) + \\ & \beta(a)(1-\xi_t(a))\pi_f(n+\ell,\Pi_t,\Pi_t^*,\mathcal{V})W_{t+1}(f,a,\mathcal{V}|s) + \\ & \text{s.t. (2) and (3).} \end{aligned} \tag{4}$$

The first line captures the utility from consumption and leisure. The following three lines capture the continuation when no vaccine is available by the end of the period. If the agent has no fever or has fever but tested negative for Covid, they continue as a susceptible person, captured in the second line. The third line captures the continuation for a feverish person who gets tested and had been infected, and the fourth line corresponds to the fever state (fever symptoms and no test). For healthy individuals the continuation values depends on whether a vaccine is available or not by the end of the period, as transitions to state $j \neq s$ only happen in the absence of a vaccine:

$$W_{t+1}(j,a,\mathcal{V}|s) = [(1-\chi(\mathcal{V}))V_{t+1}(j,a,0) + \chi(\mathcal{V})V_{t+1}(s,a,1)].$$

The value function for an agent who knows that they are infected with coronavirus

but do not need hospitalization is given by:

$$
\begin{aligned}
V_t(i,a,\mathcal{V}) = \max_{c,x,n,v,\ell,d} & \; u(c,g(x,\ell),d,v) + \\
& \beta(a)\phi(0,a)W_{t+1}(r,a,\mathcal{V}) + \\
& \beta(a)(1-\phi(0,a))\alpha(a)W_{t+1}(h,a,\mathcal{V}) + \\
& \beta(a)(1-\phi(0,a))(1-\alpha(a))W_{t+1}(i,a,\mathcal{V}) \\
& \text{s.t. (2) and (3).}
\end{aligned} \tag{5}
$$

The second line captures the case in which the agent recovers from the disease and becomes resistant to the virus.[17] The third line gives the value for the case in which the agent does not recover and requires hospitalization. The fourth line is the case in which the agent does not recover and does not require hospitalization and, thus, remains infected. Even though future health states for these individuals are not affected by vaccines, the arrival of a vaccine is relevant as it affects the persistence of lockdown measures. The continuation value in state j is therefore averaged according to:

$$W_{t+1}(j,a,\mathcal{V}) = [(1-\chi(\mathcal{V}))V_{t+1}(j,a,0) + \chi(\mathcal{V})V_{t+1}(j,a,1)].$$

To define the value for an agent in the fever state, it is convenient to denote by $\tilde{V}_t(c,x,n,\ell,d;s,a,\mathcal{V})$ the terms in lines two to four on the right hand side of (4), and by $\tilde{V}_t(c,x,n,\ell,d;i,a,\mathcal{V})$ the corresponding lines in (5). They represent the continuation values conditional on choices made this period, both for the susceptible and the infected. Those in the fever state get their current utility and the weighted average of these continuation values, weighted by the probability of being infected with Covid:

$$
\begin{aligned}
V_t(f,a,\mathcal{V}) = \max_{c,x,n,v,\ell,d} & \; u(c,g(x,\ell),d,v) + \\
& \frac{\Pi^*_{t-1}\,\tilde{V}_t(c,x,n,\ell,d;s,a,\mathcal{V})}{\Pi_{t-1}+\Pi^*_{t-1}} + \frac{\Pi_{t-1}\,\tilde{V}_t(c,x,n,\ell,d;i,a,\mathcal{V})}{\Pi_{t-1}+\Pi^*_{t-1}} \\
& \text{s.t. (2) and (3).}
\end{aligned} \tag{6}
$$

This again illustrates the mathematical simplicity of the uncertain/fever state in our model. This simple representation is possible because the similarity in transmission for Covid and the common cold leads to tractable beliefs given in (1), but also because the initial uncertainty of the fever state is revealed at the end of the period.[18]

[17] We equate recovery from infection with permanent immunity for simplicity, but extend our analysis in Appendix C to allow some reinfections among individuals who had been infected before.

[18] In the full-information states ($j = s$ or $j = i$) the individual trivially knows at the end of the period in which state they started (even though they might be uncertain about whether they contracted Covid during the period). To make the fever state comparable, the initial uncertainty needs to be resolved at the end of the period. Online Appendix F shows how the additional complications from multiple

For individuals in hospital care, we set their flow utility equal to that of death (i.e., zero) to account for the harsh nature of the disease at this stage. They can enjoy the utility of normal life again if they recover. These agents provide no labor ($n = v = 0$) but we assign an exogenous level of "outside" time ($\ell = \bar{\ell}_h$) to account for the infection burden they impose onto their carers. The value function for a hospitalized person is:

$$V_t(h, a, \mathcal{V}) = \beta(a) \left[\phi(1, a) W_{t+1}(r, a, \mathcal{V}) + (1 - \phi(1, a))(1 - \delta(a)) W_{t+1}(h, a, \mathcal{V})\right] \quad (7)$$
$$\text{s.t. (2) and (3).}$$

This captures the case of recovery as well as the chance of remaining in the hospital, and the continuation value after dying is set permanently to zero.

Finally, an agent who has already recovered and is resistant to the virus enjoys utility:

$$V_t(r, a, \mathcal{V}) = \max_{c,x,n,v,\ell,h} u(c, g(x, \ell), d, v) + \beta(a) W_{t+1}(r, a, \mathcal{V}) \quad (8)$$
$$\text{s.t. (2) and (3).}$$

Appendix E provides a discussion on the trade-offs individuals face by using the first order conditions of a problem akin to the ones presented above. This appendix also explains how this can be used in our computational algorithm.

To define an equilibrium, we need to aggregate agents' actions for given parameters and tax rates. Their decisions depend also on the risk in the economy Π_t, the transfers $T_t(j, a, \mathcal{V})$ and the testing probability $\xi_t(a)$ for each period, which are equilibrium objects. Conjecture the tuple $\{\Pi_t(\cdot), T_t(\cdot), \xi_t(\cdot)\}$ that agents take as given, and let $n_t(j, a, \mathcal{V}_t)$ and $\ell_t(j, a, \mathcal{V}_t)$ denote the agent's choices consistent with their value functions. Given the measure of $M_t(j, a)$ of agents of each type j of age a in period t, these actions determine the corresponding measures $M_{t+1}(j, a)$ in the subsequent period. We derive these laws of motion in Appendix A. This constitutes an equilibrium if actions $n_t(\cdot)$ and $\ell_t(\cdot)$ and resulting distribution $M_t(\cdot)$ indeed give rise to the tuple $\{\Pi_t(\cdot), T_t(\cdot), \xi_t(\cdot)\}$, along the lines we outline in the three steps below.

First, in equilibrium taxes are lump-sum rebated to agents according to

$$T(j, a, 0) = \tau_t(j, a, 0)[n_t(j, a, 0) + \ell_t(j, a, 0)]/[1 - \tau_t(j, a, 0)]. \quad (9)$$

Note that $n/[1 - \tau]$ and $\ell/[1 - \tau]$ capture the work and leisure time outside before taxation, which is multiplied by the tax rate and reimbursed. That is, each agent spends one unit of time on activities inside and outside the house in equilibrium, but the lockdown taxes represent "wedges" in agents' choices that distort their behavior at the

periods of uncertainty can be addressed.

margin. Taxes and transfers are set to zero after vaccine arrival.

Second, if no vaccine arrived in period t, the testing capacity K_t is assigned randomly across all individuals that reach the testing stage of the current period with new fever symptoms:

$$\xi_t(a) = K_t / \left[\sum_{\tilde{a},j \in \{s,f_s,r\}} M_t(j,\tilde{a}) \pi_f(n_t(j,\tilde{a},0) + \ell_t(j,\tilde{a},0), \Pi_t, \Pi_t^*, \mathbf{1}_{j=r}) \right]. \tag{10}$$

We include in the denominator the recovered who catch a cold, to capture the fact that recovered individuals still had to be tested to be able to go to work or join leisure activities if they had flu symptoms. In our model they get symptoms only because of a common cold, which is captured in the equation through indicator $\mathbf{1}_{j=r}$, which equals one if recovered and zero otherwise, signifying that for the recovered infections arrive as if a vaccine was present. If a vaccine is actually present for everyone, testing serves no further purpose and we set its probability to zero.

Third and most relevant, the instantaneous rate of getting infected depends on the time that every other infected individual spends outside. This is multiplied by an infectivity constant Π_0 (which we assume to be age-independent in light of no evidence to the contrary) and a seasonal component ψ_t as follows:

$$\hat{\Pi}_t = \Pi_0 \psi_t \sum_{a,j \in \{f_i,i,h\}} \left(n_t(j,a,0) + \ell_t(j,a,0) + \underline{m}\right) M_t(j,a). \tag{11}$$

This can be rationalized by assuming a common space in which agents are distributed uniformly, so that within each unit of space an individual encounters the number of infected people represented by the sum in (11), and each transmits the virus at the exogenous rate $\Pi_0 \psi_t$. This entails the standard random mixing assumption where everyone meets everyone else with equal probability.[19] Expression (11) gives the rate at which infections get transmitted on a given unit of space during an instant. We can integrate over a (weekly) unit of time to obtain the probability of at least one encounter that leads to infection:

$$\Pi_t = 1 - e^{-\hat{\Pi}_t}. \tag{12}$$

When $\hat{\Pi}_t$ is small, it holds approximately that $\Pi_t \approx \hat{\Pi}_t$. At the peak of an unregulated pandemic $\hat{\Pi}_t > 1$ is possible, reflecting that individuals in expectation get infected more than once in a period, and (12) ensures that only one of these gets counted.[20]

[19] While some (e.g., Mossong et al. (2008)) report that contact patterns are assortative with age, others report contact patterns with considerable pre-pandemic interactions across age groups (see, e.g., the matrix of pre-pandemic close contacts by age in the survey data for the US in Belot et al. (2020)). In our earlier working paper (Brotherhood et al. 2020) we discuss interventions that separate parts of the outside activities by age group, leading to selective mixing and age-specific infection risk.

[20] This problem could in principle be avoided in the calibration by choosing a small period length.

3 Calibration and Model Fit

3.1 Calibration

This section describes how we discipline the parameters of the model. Some parameters are exogenously calibrated without having to solve the model. A set of parameters is chosen by solving the steady state of the model without Covid and some are chosen in order to fit the time series path of the pandemic.

The time period is one week. Suppose the old (who do not work in the model) are those above 65 years old. According to U.S. Census Bureau (2018), this fraction is 0.214 of the adult population in 2018. Moreover, we use data from the Social Security Actuarial Tables in 2019 (Social Security Administration 2019) to compute the life expectancy of individuals in the different age groups. The median individual over 65 is 74 years old and expects to live an additional 12.9 years. Thus, set the weekly survival rate for old agents to $\Delta(o) = 1 - 1/(12.9 \times 52) = 0.9985$. The median young agent (between 20 and 64) is 42 and expects to live 38.9 years more. Hence, set $\Delta(y) = 0.9995$.

The leisure good g is produced according to $g(x, \ell) = [\theta x^{\rho} + (1 - \theta)\ell^{\rho}]^{1/\rho}$. The parameter ρ controls the elasticity of substitution between leisure time outside the home and leisure goods. Following Kopecky (2011), set $\rho = -1.72$, which implies an elasticity of 0.368 so that goods x and leisure time ℓ are complements.[21] The parameters γ (utility weight of leisure goods g), λ (utility weight of leisure time at home), ι_0 and ι_1 (which control the productivity of telework), and θ are jointly chosen to match five data targets. First, we match a 40-hour work week ($n + v = 40/112 = 0.357$) in a world without the pandemic. A fraction of 8% of these 40 hours (or 3.2 hours) are spent on telework.[22] Moreover, individuals spend 17.3 hours on non-working outside activities ($\ell = 17.3/112 = 0.154$).[23] Additionally, when 36% of work time is devoted to telework, we assume that income declines by 10%.[24] We also match a fraction of income spent

But a small period length also shortens the duration of uncertainty in our model.

[21] Appendix Table B1 computes the no-policy equilibrium for different levels of ρ, corresponding to an elasticity twice as high and half as high. The results are similar.

[22] Bick, Blandin, and Mertens (2023) report that around 8% of individuals were working from home in February 2020.

[23] This comprises the average hours per week spent on purchasing goods and services; caring for and helping nonhousehold members; organizational, civic, and religious activities; socializing and communicating; arts and entertainment (other than sports); sports, exercise and recreation; and travel related to leisure and sports. The data comes from the American Time Use Survey (ATUS) (U.S. Bureau of Labor Statistics 2018), with data retrieved through the BLS data portal (U.S. Bureau of Labor Statistics 2026a). Note that, in our model, the old do not work. In our calibration, the old spend 23 hours in leisure outside; i.e. more than the young. This is consistent with the data; the old spend 19.5 hours in leisure outside. If we include the small reported time at work, this number rises to 25.2 hours per week in the data.

[24] According to Bick, Blandin, and Mertens (2023) in May 2020, 35.2% of workers were working from home; similarly, Aum, Lee, and Shin (2022) report a 36% decline in in-loco work in the same period. Further, US GDP declined by 10% in 2020Q2.

on outside goods, x, equal to 12.5% ($x/[w(n+\iota(v)v)] = 0.125$).[25] Set the discount factor to $\tilde{\beta} = 0.96^{1/52}$.

The parameter b represents the value of being alive over and above the value of consumption/leisure. This influences how "afraid" agents are of dying. To discipline this parameter, we target a value of statistical life (VSL) of 9.3 million dollars. This is the value used by the Environmental Protection Agency (see Eichenbaum, Rebelo, and Trabandt (2021)).[26]

Normalize the wage to $w = 1$. According to Biggs and Springstead (2008), the replacement rate for social security benefits for a median-income household ranges between 46% and 64%. Set the replacement rate in the model to 60%, a value towards the upper bound of the range since households may have savings outside the official social security income. This implies $\overline{w} = .6w[n + \iota(v)v] = .6 \times .357 = .215$.

The parameters described above are summarized in Table 1. Turn now to the health-related parameters, which are summarized in Table 2.

Once the agent is infected with the coronavirus, there is a probability of recovering from the disease ($\phi(0, a)$), and if not recovered, a probability for hospitalization ($\alpha(a)$). Set $\alpha(a) = 1$ such that an infected agent spends one week with mild symptoms and recovers (probability $\phi(0, a)$) or needs a hospital (probability $1 - \phi(0, a)$), which corresponds to the length of time a Covid-positive person is infectious (CDC 2023). The parameter $\phi(0, a)$ then also controls the fraction of agents that move to an ICU. Agents in need of hospitalization may die (probability $\delta(a)$) or recover ($\phi(1, a)$). Verity et al. (2020) report that patients with severe symptoms were discharged after an average of 24.7 days, or 3.52 weeks. This yields $\phi(1, a) = 1/3.52 = 0.284$. Centers for Disease Control and Prevention (2021) reports age-specific hospitalization (including ICU) rates and infection fatality rates (IFR) for Covid-19 patients. A fraction of 1.24% of patients aged 20-64 required being moved to an ICU, whereas this number was 12.88% for those above 65 years. Centers for Disease Control and Prevention (2021) also reports the following IFRs: 0.23% for those aged 20-64 and 9% for those above 65 years old. These targets yield the following parameters: $\phi(0, y) = 0.988$, $\phi(0, o) = 0.871$, $\delta(y) = 0.090$ and $\delta(o) = 0.921$.

Hospitalized agents in the model cannot work and do not make any decisions. We assume a flow utility level of 0, i.e. the same as death. These individuals still interact

[25] This comprises expenditures on food away from home, public transportation, medical services and entertainment. The data comes from the Consumer Expenditure Survey (CEX) data for 2018 (U.S. Bureau of Labor Statistics 2020).

[26] To fit the VSL, we consider a young person in a no-Covid world, increase the quarterly probability of death by 1/10,000 and compute b such that this person needs to be given 930 dollars per quarter to be indifferent. To interpret monetary units in the model as dollars, we assume that the quarterly income of young agents before the pandemic is proportional to 15,000$, which corresponds to the quarterly US GDP per capita in 2018. The old in the model have a VSL 57% that of the young, which is close to estimates in the literature (e.g., 70% in Krupnick et al. (2002)). We also check robustness with respect to a lower VSL of 5 million dollars (see discussion in Footnote 31).

Table 1: Calibration – Economic & Preference Parameters

Parameter	Value	Interpretation
	0.214	Fraction of old in population
ρ	-1.72	Elasticity of subst. bw leisure time and goods
θ	0.033	Production of leisure goods
γ	0.635	Rel. utility weight - leisure goods
λ	1.562	Rel. utility weight - leisure at home
b	15.63	Flow value of being alive
$\tilde{\beta}$	$0.96^{1/52}$	Discount factor
w	1	Wage per unit of time
ι_0	1.055	Parameter related to telework productivity
ι_1	0.960	Parameter related to telework productivity
$\overline{w}$	0.215	Retirement income
$\Delta(y)$	0.9995	Weekly survival (natural causes), young
$\Delta(o)$	0.9985	Weekly survival (natural causes), old

with others (e.g., doctors and nurses) for a fraction $\bar{\ell}_h$ of their time. Butler et al. (2018) estimate that patients in ICUs spend 7.6 hours a day interacting with other people. Thus, set $\bar{\ell}_h + \underline{m} = 7.6/24$, where we add the exogenous amount of interactions all individuals engage in, $\underline{m}$. This will be elaborated on momentarily.

We assume that individuals face a constant probability of vaccine arrival and that they expect this will take on average one and a half year, or 78 weeks. Hence, $\chi_t = 1/78$ for all $t < T^*$. For $T^* = 156$, we set $\chi_{T^*} = 1$ so that the pandemic is over after 3 years. This latest possible end for the pandemic is set for computational reasons and does not alter the results.

According to Heikkinen and Järvinen (2003), the average American has between two and four colds every year. Suppose an agent in the model has an average of three colds per year. This implies a weekly infection rate of 0.058. The infectiousness of the common cold is controlled by Π^* and we set it to hit this infection rate in a world without Covid. The resulting value is $\Pi^* = 0.094$.

Individuals in the model can be tested each period according to the time-varying probability ξ_t. To discipline this, we take the number of tests performed in the US for each week (Our World in Data 2022) (Figure 6b below) and use equation (10) to back out ξ_t for every t. The main assumption in this procedure is that there is no reallocation of tests over time.

Covid-19 follows a seasonal pattern of infections similar to influenza such that it is easier to contract this type of diseases in winter months (Hoogeveen and Hoogeveen 2021). The seasonality of Covid in the model is controlled by ψ_t. We assume a "triangular" shape for ψ_t: the period of high-infectiousness lasts for 16 weeks, 8 weeks before and after a peak on December 1st, as CDC (2022) reports that the flu season

Table 2: Calibration – Disease Parameters

Parameter	Value	Interpretation
α	1	Prob(hospitalization | no recovery from mild)
$\phi(0,y)$	0.988	Prob of recovering from mild Covid-19, young
$\phi(0,o)$	0.871	Prob of recovering from mild Covid-19, old
$\phi(1,y)$	0.284	Prob of recovering from hospitalization, young
$\phi(1,o)$	0.284	Prob of recovering from hospitalization, old
$\delta(y)$	0.090	Weekly death rate (among hospitalized), young
$\delta(o)$	0.921	Weekly death rate (among hospitalized), old
$\bar{\ell}_h$	0.174	Infections through the health care system
χ_t	1/78	Prob of vaccine arrival (average = 78 weeks)
Π^*	0.094	Weekly infectiousness of common cold/flu
Π_0	9.078	Infectiousness of Covid-19
$\bar{\psi}$	1.51	Peak of infectiousness during winter
η_0	4.33e-6	Stringency index function
η_1	2.553	Stringency index function
$\bar{\tau}_i$	0.379	Time tax rate for isolation (positive tests)
$\underline{m}$	0.143	Exogenous Covid infections
I_0	0.0011	Initial fraction of infected people

Table 3: Fitted Moments – Model vs. Data

Moment	Model	Data
R_0 (winter)	3	3
Deaths per 100,000, March 22 2020 (start)	1.280	1.277
Deaths per 100,000, April 12 2020 (1st peak)	6.665	6.812
Deaths per 100,000, June 21 2020 (1st trough)	1.462	1.519
Deaths per 100,000, January 3 2021 (2nd peak)	9.179	10.288
Deaths per 100,000, April 25 2021 (2nd trough)	1.673	1.654
% rise in home hours rel. to no-Covid, March 29 2020 (1st peak)	39.815	39.203
% rise in home hours rel. to no-Covid, September 6 2020 (1st trough)	19.998	16.801
% rise in home hours rel. to no-Covid, January 17 2021 (2nd peak)	24.479	22.402
% rise in home hours rel. to no-Covid, June 20 2021 (2nd trough)	10.076	11.201

usually occurs in the fall and winter in the US. We normalize the low-infectiousness level to $\psi_t = 1$. 8 weeks before December 1st, it starts to linearly increase until it hits $\bar{\psi}$ on December 1st. It then linearly decreases during 8 weeks back to 1. The level of $\bar{\psi}$ will be determined jointly with other parameters as described below.

The parameter Π_0 controls how infectious Covid-19 is. We pick Π_0 in order to match the basic reproduction number (R_0) of Covid-19. R_0 represents the average number of new infections that a random person who gets infected at the start of the pandemic is expected to generate over the course of their disease. In our model, this is closely related to Π_0. We thus pick this parameter to generate an R_0 of 3 in the peak of the

winter. Given our calibrated value for $\bar{\psi}$, this implies an R_0 of around 2 in the summer. This falls within the estimates provided by Centers for Disease Control and Prevention (2021). Π_0 will be jointly calibrated with other parameters as well.

The time series for τ_t must also be set. This controls how strict lockdowns are in the benchmark economy. To discipline this, we use the stringency index from the *Oxford Coronavirus Government Response Tracker*, which aggregates nine metrics related to Covid restrictions in the US (Roser 2021). Since this stringency index does not have a cardinal interpretation, we impose the following function: $\tau_t = \eta_0(\text{stringency}_t)^{\eta_1}$, where stringency_t is the stringency index in the data and τ_t is a uniform time tax that applies to all individuals. Additionally, an individual is also required to stay at home longer if they test positive for Covid. Denote the time tax rate an individual in this situation faces by $\bar{\tau}_i$. If the period lockdown rate τ_t happens to be above $\bar{\tau}_i$, we assume an individual that tests positive faces the higher of the two values. So $\tau_t(i, a, 0) = \max\{\bar{\tau}_i, \tau_t\}$ for infected individuals and $\tau_t(j, a, 0) = \tau_t$ for everyone else. The parameters η_0, η_1 and $\bar{\tau}_i$ will be determined jointly with other parameters.

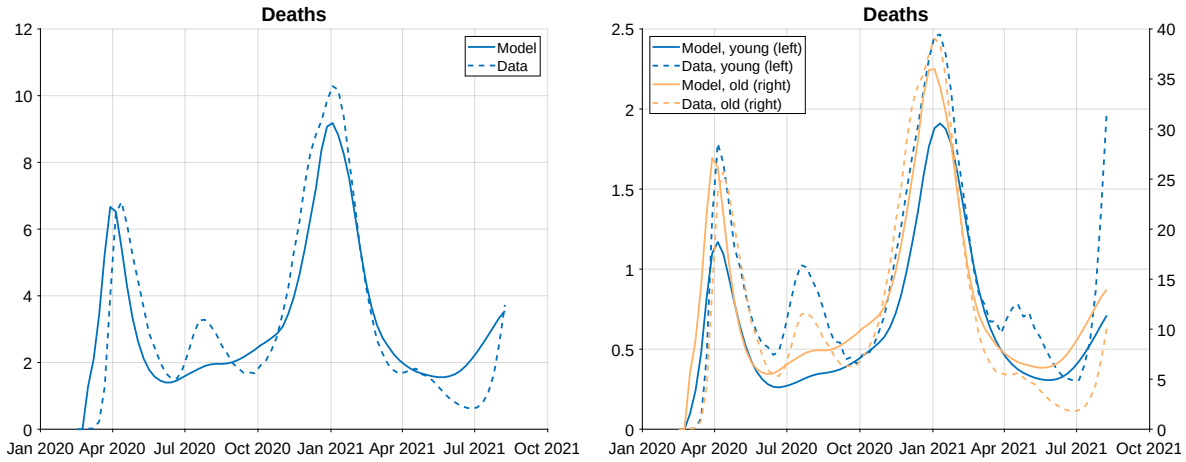
The parameter $\underline{m}$ controls the exogenous Covid infections. That is, infections that individuals cannot prevent by spending less time outside. Moreover, denote by I_0 the initial fraction of infected individuals at the outset of the pandemic in the model. These two parameters will be determined jointly with other parameters.

We are thus left with the following seven parameters to determine: $\bar{\psi}$, η_0, η_1, $\bar{\tau}_i$, Π_0, $\underline{m}$ and I_0. We discipline these parameters by targeting ten data moments. The first moment is R_0. The remaining nine targets are moments throughout the first year and a half of the pandemic. In particular, we target the overall death rates (for young and old combined) provided by the CDC/NCHS (National Center for Health Statistics 2026) in the following five dates: March 22 2020 (beginning of the pandemic), April 12 2020 (first peak), June 21 2020 (first trough), January 3 2021 (second peak) and April 25 2021 (second trough). Moreover, we target *Google Mobility's* data on the rise in time spent at home relative to the no-disease baseline (Google LLC 2022) in the following four dates: March 29 2020 (first peak), September 6 2020 (first trough), January 17 2021 (second peak) and June 20 2021 (second trough). The model fit is displayed in Table 3. Despite targeting ten moments with only seven parameters, the fit of the model is very good.

One may wonder about the size of the isolation tax $\bar{\tau}_i$ following a positive Covid test. The calibrated value corresponds to roughly a 50% reduction in time spent outside. Isolation is therefore not perfect, which is consistent with casual observation and evidence from other settings.[27]

[27]The isolation tax captures a mixture between strictly enforced isolation and strongly worded governmental appeal to self-isolate with only moderate enforcement. Epidemiologists have been interested in the consequences of the latter in the context of other epidemics. When surveyed, the overwhelming majority indicates willingness to comply with such appeals even without enforcement. To our knowl-

Figure 1: Deaths per 100,000 people, model versus data



(a) Deaths, all ages

(b) Deaths by age group

Note: Data from CDC/NCHS (National Center for Health Statistics 2026).

Our model also has implications for where infections happen which can be checked against the data. Ferguson et al. (2006) consider transmissions outside of the household and claim that 53% of those take place in schools and workplaces. Mossong et al. (2008) consider the number of interactions associated with work and with leisure activities, and attribute 57% of those to work. According to our calibrated model, at normal activity level, 61% of such infections are attributed to work-related activities. Hence, our calibrated model matches these data relatively closely. In the next section, we assess how well the model matches several non-targeted moments over time.

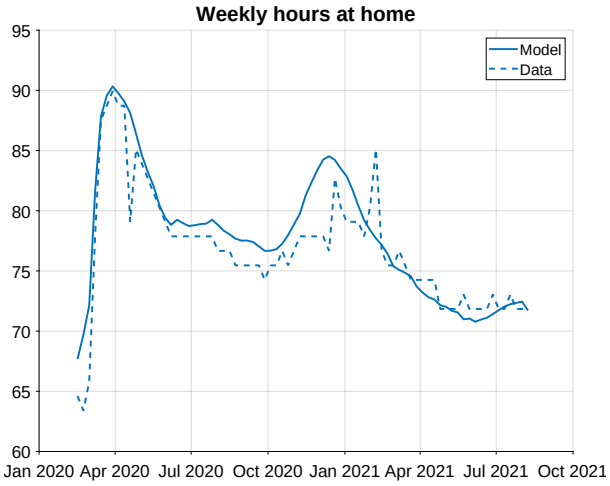
3.2 Non-targeted Moments: Mortality and Behavior over Time

The calibrated model captures the timing of the pandemic over the first year and a half quite well. Only a few points of the time series of aggregate mortality and time at home were included as calibration targets (see Table 3). Yet, the model matches the entire paths of mortality (see Figure 1a) and time at home (see Figure 2) quite closely.

The model further matches the behavior by age, which was also not targeted. Figure 1b shows death rates by age over time in the model compared to the data. We also compare the change in time devoted to outside leisure in the model with the data, see Table 4. Though the match is not perfect, the decline in outside leisure was larger for the old than the young, both in the model and the data. This is true both for the time periods between May and July as well as May through December, two periods with different Covid experiences. Naturally, the young reduced both their outside leisure

edge, Rizzo et al. (2013) is the only study that reports actual adherence, which averaged roughly 50% in the context of the swine flu across Italy, Finland and Romania, which might indicate that our calibrated magnitude is reasonable.

Figure 2: Time at home, model versus data



Note: Data from Google Mobility (Google LLC 2022).

Table 4: Change in (non-work) time outside by age, model versus data

	May-July		May-December	
	Model	Data	Model	Data
Young	-12.0	-24.9	-11.1	-16.1
Old	-35.5	-31.4	-47.5	-28.9

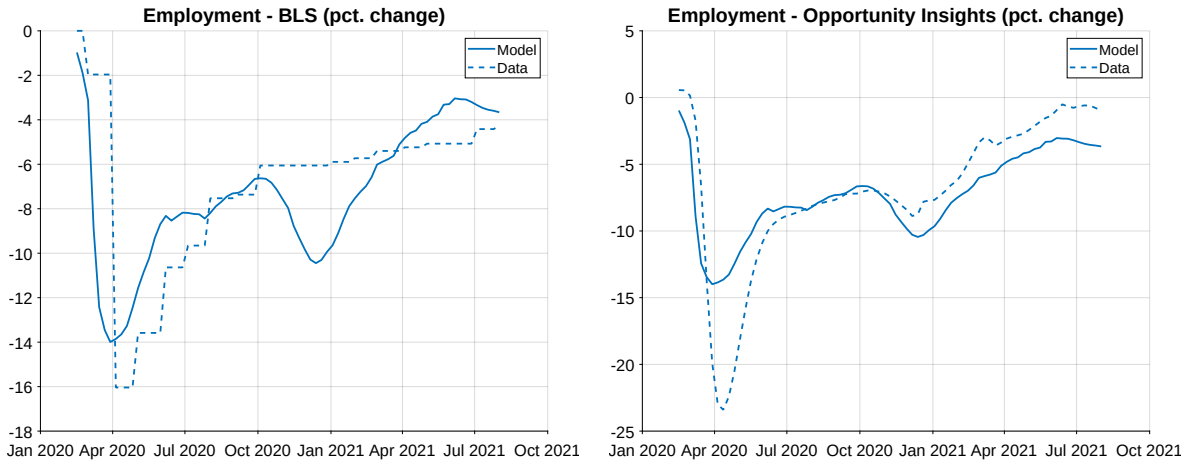
Note: Data from the American Time Use Survey (ATUS) (U.S. Bureau of Labor Statistics 2021). Time outside in the data comprises the activities listed in Footnote 23. Declines relative to the same months in 2019.

and their working hours. To verify that our model captures this decline in work time, we compare it to BLS employment data (U.S. Bureau of Labor Statistics 2026b) and employment estimates from the Opportunity Insights Project (Opportunity Insights 2026), see Figure 3 (Panels a and b respectively). The model matches the data well, both in magnitude and the timing.

Finally, the test positivity rate changes over the course of the pandemic. Figure 4 shows that it was highest early on in the pandemic, then dropped substantially and increased again dramatically during the winter of 2021. The model captures these two peaks quite well. The data displays additional smaller ups and downs that the model does not capture. This is perhaps not surprising since policies on testing changed over time, a feature our model abstracts from.

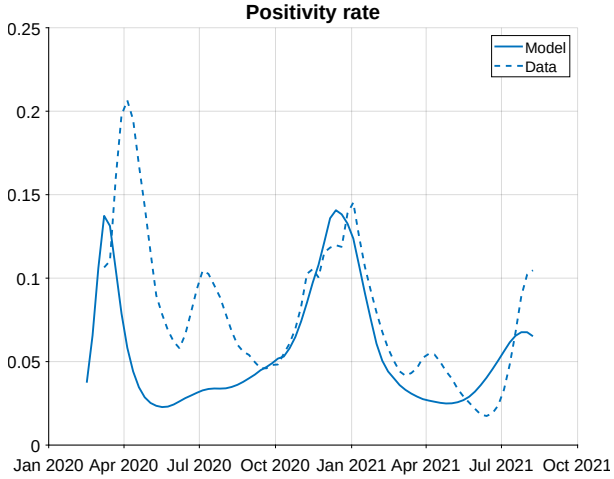
In sum, our model captures the timing of the pandemic over the first year and a half quite well. This is true both in terms of the timing of mortality, but also behavioral change, and it is true by age as well. We thus believe our model is a good starting point to ask what policymakers could have done better, i.e. for policy exercises.

Figure 3: Time at work, model versus data



Note: BLS data corresponds to the employment-to-population ratio (U.S. Bureau of Labor Statistics 2026b). Employment from the Opportunity Insights project aggregates data from Earnin, Intuit, Kronos, and Paychex (Opportunity Insights 2026).

Figure 4: Positivity rate of tests, model versus data



Note: Data shows the share of reported tests returning a positive result. Source: Our World in Data and WHO (Our World in Data 2022; World Health Organization 2026).

4 The Importance of Behavioral Changes

Table 5 reports our results based on the calibrated economy described in the previous section (Column Benchmark). Panel A provides statistics for different time horizons: 1 year and 1.5 years. The 1.5 year time horizon corresponds to the expected vaccine arrival time in the calibrated model and also the approximate date when the vaccine became widely available in the data. In the discussions below, we thus equate the total death toll of the pandemic with the number of deaths by vaccine arrival, which is 1.5 years for Covid. The pandemic's peak infection rate is observed in week 44. As seen in Figure 1, this date corresponds to the second wave of the disease in the winter of 2020-21. The death count over the first year and a half (i.e., before the vaccine became available) is substantial: 2.48 deaths per 1,000 people. This number masks considerable age heterogeneity: the death rate is 9.76 per 1,000 old individuals and 0.50 for the young.[28] By the time the vaccine arrives, about 20% of the population had been infected and recovered.

Panel B provides the expected statistics at the onset of the pandemic when individuals look ahead at a 3-year horizon. This computation takes into account the positive probability of vaccine arrival at different time intervals, such as one week, two weeks, and so forth. Since the vaccine is a perfect solution to the disease in the model, the pandemic is over upon its arrival. The expected death toll is a little lower: 1.94 dead per 1,000 people. These expected statistics will be important in the upcoming sections when we solve for the optimal lockdown policy that a planner chooses to maximize expected utility, not knowing when the vaccine will actually arrive.

While the death toll is substantial, it is much lower than most predictions at the onset of the pandemic. The peak is also much later than what epidemiological models predicted in early 2020 (Ferguson et al. 2020). Why? Because people changed their behavior and acted to "flatten the curve." To understand the role of behavioral changes, we compute an epidemiological version of the model (column 2 in Table 5), which assumes behavior does not adjust at all. That is, people keep on living their lives as if the disease did not exist. By the arrival of the vaccine, the death toll in the benchmark is 80% lower than in this epidemiological version of the model (2.48 versus 12.12). The lack of behavioral change implies a much swifter pandemic: the peak occurs already in week 10, aligning with the 3-month horizon many epidemiologists predicted at the onset of the pandemic (Ferguson et al. 2020).

The change in behavior responsible for this marked difference is due to a combination of voluntary precautions and policy. These factors jointly cause people of both ages to spend more time at home than they did in the no-disease world (see Column

[28] Our (realistic) assumption that the old have a higher probability of dying from natural causes plays a role here. If the old faced the same lower probability as the young, they would be even more careful leading to a death rate of only 8.85 per 1,000 instead.

Table 5: Benchmark Results

		Benchmark (1)	Epidemiological (2)	No lockdown (3)	Age ext. partial (4)	Age ext. general (5)
Panel A. Statisitics assuming vaccine arrives in 1.5 years						
Weeks to peak infections	Young	44	10	10	45	1
	Old	45	10	10	45	2
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	17.02	0	6.87	40.89	21.91
	Old	11.56	0	14.47	11.56	4.70
Dead p/ 1,000, first year	Young	0.37	1.81	0.98	0.19	0.04
	Old	7.40	50.03	16.31	7.40	1.17
	All	1.87	12.12	4.26	1.73	0.28
Dead p/ 1,000 (by vaccine arrival)	Young	0.50	1.81	1.02	0.26	0.06
	Old	9.76	50.03	17.08	9.76	1.74
	All	2.48	12.12	4.46	2.29	0.42
Recovered , % (by vaccine arrival)	Young	23.17	78.74	44.84	11.90	2.99
	Old	10.63	51.44	17.89	10.63	1.90
	All	20.48	72.90	39.07	11.62	2.76
GDP 1 year, % change w.r.t. no-disease		-12.96	-1.44	-5.77	-33.72	-16.91
Panel B. Expected statistics						
Hrs @ home, diff. w.r.t. no-disease	Young	14.03	0	6.10	37.22	21.02
	Old	10.28	0	11.87	10.28	4.66
Dead p/ 1,000	Young	0.40	1.61	0.81	0.21	0.06
	Old	7.57	44.44	13.30	7.57	1.54
	All	1.94	10.78	3.48	1.78	0.38
GDP, % change w.r.t. no-disease		-11.33	-1.78	-5.67	-31.25	-16.66

Note: Vaccine arrival: 1.5 year.

1). While the old spend more time at home even without the disease (since they do not work), they still cut leisure outside substantially and increase their time at home by 11.5 weekly hours on average during the first year of the pandemic. The young cut both time at work and leisure outside, adding 17 extra weekly hours at home. These changes by both groups reduce the overall number of infected/recovered people by about 50 percentage points by the time a vaccine becomes available. The total death rate declines from 12.12 to 2.48 per 1,000 people, but markedly more so among the old. The economic costs of this lower level of activity is sizable: GDP for the first year of the pandemic is reduced by about 13% relative to a no-disease world.

The behavioral changes observed in the benchmark are partly due to voluntary individual precautions but also due to the government lockdown. To disentangle the two reasons, column (3) shows results for an equilibrium without any lockdown, so that the difference between Columns (2) and (3) is due to voluntary precautions. Without the policy, the behavior of both groups changes markedly. The young stay less time at home compared to the benchmark, while the old stay more time at home (comparing Columns 1 and 3). Without the lockdown, the disease is more prevalent, prompting the old to protect themselves more. By the time the vaccine arrives, 39% of the population has recovered from the disease, almost twice as much as in the benchmark. Consequently, more deaths materialize: 4.46 per 1,000 people versus 2.48 in the benchmark. The old in particular experience a death rate of 17.08 per 1,000. At the same time, the lives saved even without a lockdown are sizable. By the time of vaccine arrival, the epidemiological model predicts 12 deaths per 1,000, the no-lockdown model only 4.5, and the model with voluntary precautions and lockdown only 2.5. Thus,

voluntary precautions averted more deaths than the government-imposed lockdown.

The no-lockdown scenario in Table 5 still contains some public policies that were implemented during the pandemic: testing and the isolation of individuals who are found positive for Covid. Appendix Table B2 provides the results for two alternative no-lockdown scenarios: one with testing but without isolation of positive cases and another with no testing at all. The experiment with no lockdown and no testing can be thought as a pure laissez-faire equilibrium in which the government does not intervene at all. The testing-and-no-isolation scenario features more deaths and, consequently, more precautionary behavior with individuals spending more time at home. Higher cases and deaths materialize here because individuals that test positive and are not isolated are more likely to leave their homes since they cannot catch the disease again.[29] The no-testing scenario actually features fewer deaths compared to the testing-and-no-isolation case because agents are unsure whether they have been infected or not and thus behave more cautiously. Hence, to be effective in bringing down cases and deaths, testing must be accompanied by strict isolation of positive cases. Section 6 explores testing scenarios in more depth.

The young have stronger incentives to leave their house because of work. As a result, they contribute more to the spread of the disease; and the burden will fall more heavily on the old. This is a multi-group equivalent of the immunity externality discussed in Garibaldi, Moen, and Pissarides (2023). To quantify this dynamic externality, we run two counterfactuals. Suppose the preferences of the young feature the same death and symptoms probabilities as those of the old (keeping the actual transition rates at their calibrated values). That is, the young, who still need to work for their income, believe they are subject to the same risks as the old. One counterfactual runs such scenario in partial equilibrium: we observe the difference in behavior of the young assuming they cannot affect the aggregate infection rates. The other counterfactual performs the same experiment in general equilibrium. In partial equilibrium (Column 4), the young become more careful and substantially increase their hours at home. This lowers the infections among this group and they consequently die in much lower numbers. In general equilibrium, this extra cautiousness of the young affects the old in two ways. First, by being more careful, less infections take place. On the other hand, if the young are more reckless and face more infections, they end up contributing more to herd immunity and less of the burden falls on the old (Gollier 2020b). In the experiment with our benchmark calibration (Column 5), when the vaccine arrives for everyone after only 1.5 years, the economy remains far from herd immunity. Thus,

[29] These results echo those in Eichenbaum, Rebelo, and Trabandt (2022) where testing without isolation leads those who test positive to become more risky as they have less to lose. These results would change with altruism as we show in a previous version of this paper (Brotherhood et al. 2021). With altruism people do not engage in fatalistic behavior once they are tested positive, so that testing even without isolation can have some positive effects.

more careful young individuals have a positive effect on the old and the death rate among the latter group declines. This safer behavior by the young also lowers the externality among their own age group. With a less prevalent disease in this experiment, the young die less and can afford to reduce their hours at home.

5 Optimal Lockdown

Throughout the pandemic, several governments around the world implemented lockdowns. This section investigates the impact of such policies on the dynamics of the disease and the economy. We solve the problem of a utilitarian planner that maximizes the population share-weighted discounted utility of the old and the young at time $t = 0$. To do this, the planner chooses how strict the confinement must be by setting $\tau_t(a)$ to alter individual choices. In particular, we allow the planner to choose time-varying age-specific policies. These apply as long as no vaccine is available and apply to everyone, except to those who test positive and are subject to isolation.[30] Since the economy has no aggregate shocks and evolves deterministically in the absence of a vaccine, the planner can perfectly forecast its path and does not need to condition on any other state variable. One can view this policy as a time-zero commitment, and individual agents take it into account when they make their forward-looking choices.

Table 6 reports the results for the optimal policy. The optimal lockdown substantially decreases the death toll and averts 97% of the deaths that would occur in a no-lockdown equilibrium.[31] Under the benchmark lockdown, expected deaths decrease by only 44.4% instead. Welfare (measured as consumption equivalent variation, CEV) increases with the optimal policy for both groups, and especially so for the old.

How does the planner achieve such an outcome? The optimal lockdown is much stricter than the actual policy implemented in the United States during the pandemic. Average time taxes in the first year are roughly 50% higher at the optimal policy.[32] Accordingly, the average time spent at home is also substantially higher. To understand the magnitude of the tax effect, one can do a simple thought experiment. Imagine indi-

[30] In the model section we discussed the assumption that susceptible, fever and recovered individuals are treated equally to capture the information and enforcement constraints that might hinder the government to target its lockdown more finely and seem characteristic of actual lockdown policy. It means that $\tau_t(j, a, 0) = \tau_t(a)$ for everyone except those who test positive, for whom $\tau_t(i, a, 0) = \max\{\bar{\tau}_i, \tau_t(a)\}$, where $\tau_t(a)$ is the optimal age-dependent lockdown policy and $\bar{\tau}_i$ is the calibrated isolation parameter. After vaccine arrival $\tau_t(j, a, 1) = 0$ since lockdowns serve no further purpose. We provide results for an unconstrained planner later for comparison.

[31] Such a "no-Covid" policy that avoids nearly all deaths is not sensitive to our specific choice of value of a statistical life that underlies parameter b: when we reduced the VSL to 5 million dollars, the no-lockdown equilibrium featured slightly increased economic activity and associated deaths, but the amount of deaths averted under the optimal policy remains virtually unchanged (see Tables B3 and B4 in the appendix).

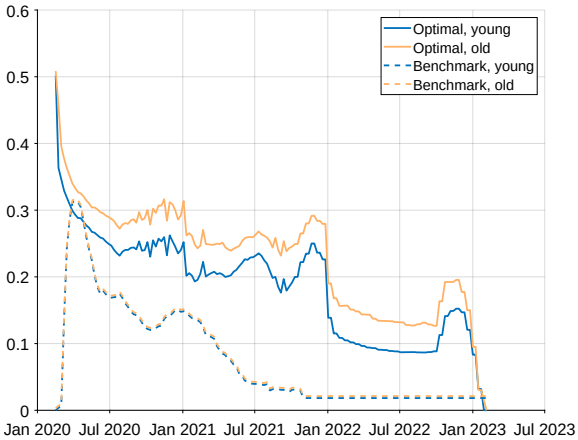
[32] The optimal taxes amount to a rebate of slightly under 10% of the total time budget for the young, compared to a rebate of just above 6% at the baseline policy.

Table 6: Optimal Lockdown

		Optimal lockdown (1)	Benchmark lockdown (2)
Panel A. Statisitics assuming vaccine arrives in 1.5 years			
Avg tax., first year	Young	0.26	0.17
	Old	0.31	0.17
Hrs @ home - avg. first year, diff. w.r.t. no-lockdown	Young	15.76	10.15
	Old	-9.16	-2.90
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	22.63	17.02
	Old	5.30	11.56
% Deaths averted, first year (rel. to no-lockdown)	Young	98.93	62.40
	Old	98.19	54.61
	All	98.32	56.01
% Change in GDP rel. to no-lockdown, 1 year		-12.44	-7.64
Panel B. Expected statistics			
Hrs @ home - diff. w.r.t. no-lockdown	Young	16.89	7.93
	Old	-6.38	-1.58
Hrs @ home - diff. w.r.t. no-disease	Young	22.99	14.03
	Old	5.49	10.28
% Deaths averted, rel. to no-lockdown	Young	98.36	50.30
	Old	97.40	43.08
	All	97.58	44.41
% Change in GDP, rel. to no-lockdown		-11.39	-5.55
Panel C. Welfare			
CEV rel. to no-lockdown (%)	Young	0.35	0.24
	Old	16.97	7.41

Figure 5: Optimal Lockdown

(a) Time tax rates (τ)



(b) Time outside

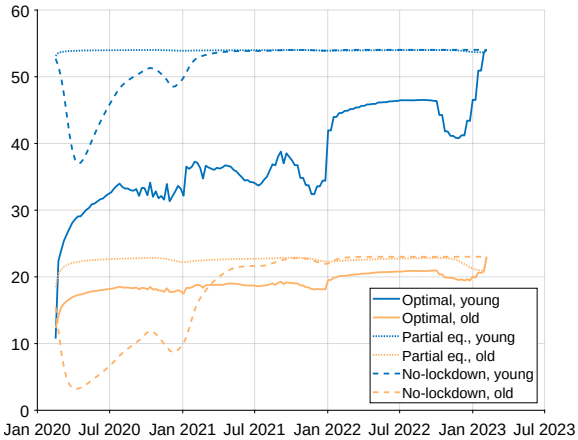
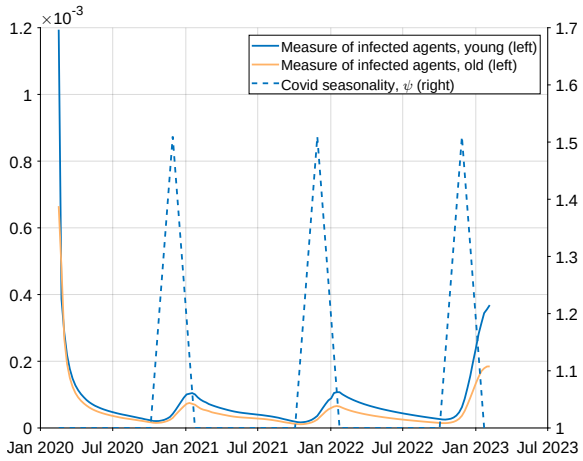
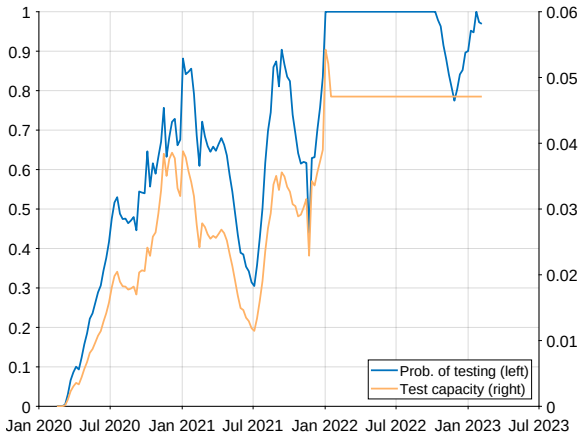


Figure 6: Infections and Testing under the Optimal Lockdown

(a) Infections and Seasonality



(b) Testing



viduals are not yet aware of the disease, but face a time tax. This changes their time at home, and therefore the number of new infections that a single infected person would generate. That means it changes R_0. In case of a tax of 17% as in the benchmark, this thought experiment reduces R_0 from its normal value of 2 (3) in the summer (winter) down to 1.31 (1.98) in summer (winter), implying that an infected person still generates a substantial number of new infections and the disease grows roughly at 31% (100%) per week.[33] Applying the same thought experiment to the average optimal tax of 26% and 31% instead, reduces the associated R_0 further to 1 (1.51) in summer (winter), so that the disease would be stable in the summer and only expand in the winter. Once individuals are aware of Covid, they obviously aim to avoid it, and testing and associated isolation also reduce transmissions, implying that with such taxes infections decline during the summer at that tax rate. This thought experiment gives only a rough idea of the impact of the lockdown, though, since lockdown taxes are not constant over the first year.

Panel (a) in Figure 5 shows time path of taxes, and illustrates that the optimal tax (solid lines) is substantially higher than the tax associated with the actual lockdown (dashed lines). Optimal taxes start high and quickly fall to a level where they remain relatively stable over the first year, with slight increase towards the winter. This strict initial response curbs infections drastically over the first weeks of the disease (see Panel a of Figure 6). This more controlled disease allows the planner to subsequently relax the restrictions, and for the remainder of the first year infections slowly decline further in the summer and increase in the winter. Subsequently, the planner keeps "fighting" seasonality, with taxes slightly rising in the winter to counter surges in infections, followed by a drop in the level of taxes over the rest of the year till the next

[33] Most transmissions happen when individuals are either in state i or f, which in our calibration lasts one week, which closely links R_0 to the weekly rise in infections.

winter, when taxes rise again. The level of taxation overall declines over time. How much the planner can ease restrictions is influenced by the availability of tests over time. With the ramp-up in the supply of testing and a low number of cases, the probability of testing increases to almost 1 from January 2022 onward (Panel b in Figure 6). Hence, the planner can reduce the level of taxation and still control the disease.[34]

Now consider the two age groups separately. Start with the young. Under the optimal policy, the young spend less time outside compared with the no-lockdown equilibrium. Part of the extra time at home is spent in productive telework while part of it is devoted to leisure. With less time outside, the young cut both leisure outside and the more productive time at work outside. This reallocation of time causes GDP to fall. In the first year, output is 12% lower compared to the no-lockdown equilibrium (versus 7.6% lower under the benchmark lockdown). Another way to gauge the strictness of the optimal lockdown is to ask what a single individual would like to do differently under such a policy. This exercise tells us how constrained an individual "feels." The answer is: very. The dotted line labeled "Partial eq., young" Panel (b) of Figure 5 depicts the optimal behavior of an individual young person, holding the behavior of everyone else constant at the optimal policy. The figure shows that the young would like to essentially behave as in the no-Covid world. Hence, relative to what they would like to do, the optimal policy restrictions feel very strict. At the optimal policy, Covid is almost eradicated. Hence, the probability of infection is very low and people are not concerned. However, if no policy was implemented and they had to self-protect instead (the dashed line) they would be a lot more cautious simply because the risks would be higher. Yet, far from cautious enough.

The policy prescription for the old is markedly different. The planner allows the old to spend *more* time outside compared to the benchmark (see Table 6). The lower disease prevalence enables an environment safe enough to allow more social interactions for the old. Even though the old do not work, they value leisure, which is taken into account by the planner. This is in contrast with frameworks that only take into account the output produced by different age groups (as in Acemoglu et al. (2021) for example). The old can go out more than in the benchmark, but also more than they would during the first year in a world without any lockdown (see the dashed and solid line in Panel b in Figure 5). The risk in the no-lockdown environment induces them to voluntarily refrain from going out despite the high costs in terms of lost utility from such interactions. Under the optimal lockdown the planner needs to place

[34]Restrictions can be lifted slightly over the years not just because of improved testing but simply because the disease is brought more and more under control with each season, as in the scenario without taxation studied in Section 6. Yet, sharp declines in taxes as we see here after 2022 seem tightly linked to improved testing capacity. Taxes do not fall to zero after 2022 because testing alone does not control the disease (again, see Section 6). The final increase in infections in Panel (a) of Figure 6 is an artifact of the time horizon $T^* = 156$ that we set for computational reasons, as discussed in Section 2.

restrictions on the old, though, as otherwise they would free-ride too much on the low infection risk. If an old individual were allowed to choose without constraints, they would spend even more time outside (see the partial equilibrium choices, represented by dots in Panel b of Figure 5). Still, the social optimum (solid line) and the privately optimal choice (dotted line) are closer together for the old than the young because the old spend less time outside and are intrinsically more responsive to the disease.

The welfare gains for the old are close to an unconstrained planning problem where taxes can be set separately for all health states (see Appendix Table B5). The reason is that the optimal policy is essentially a "no-Covid" policy that avoids most deaths, and that is where the welfare gains for the old come from. The young would gain substantially in the unconstrained optimum as the costs of reducing transmissions would be largely concentrated on those who tested positive or have a fever, while everyone else is hardly restricted. As discussed in the model section, it seems unrealistic that a planner has the required information to implement such a policy and we view this only as a theoretical benchmark rather than an actual policy.[35]

Our model assumes that recovered individuals are immune forever, while in reality reinfections happen. How sensitive are our results to the possibility of reinfections? Surprisingly little. We computed a version of the model with reinfections and find only minimal changes in optimal policy. The reason is that the optimal policy without reinfections is essentially a no-Covid policy in which nearly all deaths are averted. When most infections are averted, reinfections play little role and accordingly the optimal policy is similar.[36]

In summary, the optimal lockdown chosen by the social planner is stricter than what was actually implemented in the US. This stricter lockdown averts more deaths for both age groups. Even though the optimal restrictions are larger for both age groups, effectively they only constrain the young who substantially cut back on out-

[35]It seems unlikely that a planner has the information and enforcement capabilities to tailor lockdowns separately to individuals with rather similar appearance such as susceptible, mild symptoms, and recovered, or that the planner could set arbitrarily high taxes on those who test positive. This certainly was not part of the policy focus in the US Covid-19 pandemic. In the theoretical benchmark where a planner is able to do so, the policy would impose virtually no lockdowns on susceptible individuals, but instead impose much stricter lockdowns on fever individuals and a nearly complete lockdown on infected individuals (see Appendix Table B5).

[36]We introduce reinfections in Appendix C. Qualitatively, higher reinfection risk—all else equal—increases infections and deaths and reduces welfare. Quantitatively, the effects at benchmark calibration parameters are minor when reinfection risk is set in line with early non-Omicron variants (see Column 3 in Table C12). Optimal policy in terms of lockdown taxes and hours at home relative to a no-disease world hardly changes (see Table C13). Results relative to no lockdowns differ because, without lockdowns, reinfections lead to many more deaths. This renders individuals more cautious, with associated higher hours at home and lower GDP. This effect is more pronounced the larger the reinfection probability. At a 90% probability, voluntary precautions are so high that additional policy lockdowns are quite mild. However, at reinfection rates calibrated to the early Covid-19 pandemic (22%), the additional hours at home imposed by the planner are quite similar to those in the model without reinfection risk.

side interactions. The optimal restrictions actually allow substantially more time outside for the old: despite higher restrictions, the threat of infection is lower and allows for more interactions. The planner desires this asymmetry because of endogenous behavior: in the no-lockdown equilibrium, the young do not sufficiently take account of the disease while the old bear too much of the burden through being cautious. How strict the planner is depends on disease seasonality and testing capacity. Restrictions increase during more infectious winter seasons. Generally, as testing capacity ramps up, the planner decreases the overall strictness of the lockdown. Consequently, to arrive at the optimal approach to tackle the pandemic, it is key to jointly model behavior, testing and policies.

6 The Importance of Testing

Testing is a crucial tool in the fight against Covid-19. Several governments have employed extensive testing to identify and isolate cases swiftly. Can testing alone stop the pandemic? How does testing capacity affect the optimal lockdown? What would be the optimal testing strategy? In particular, is it worth investing more resources to build up testing infrastructure quickly? In this section, we start by comparing different testing scenarios in the baseline model to assess the effects of testing. We then modify the model to allow for test infrastructure build-up and let the planner choose the optimal number of tests alongside the optimal lockdown.

First, it should be mentioned that testing has bite in our model even though uncertainty lasts only one week because in our calibration the infectious period also lasts for only one week. Therefore, uncertainty coincides exactly with the time when individuals are infectious (though it abstracts from uncertainty for recovered individuals). Testing thus brings certainty exactly at the time when it matters for infections, and a robustness exercise with two periods of uncertainty shows little quantitative difference.[37] Testing serves two roles: it allows individuals to know if they are infected or susceptible, and it imposes $\bar{\tau}_i$ on the infected to induce isolation. Testing means that individuals spend less time outside both when infected (due to isolation) and when not (to avoid catching the disease) compared to non-tested "fever" individuals.

Is testing then a silver bullet? That is, can enough testing eradicate the disease? Suppose every fever agent is tested; that is, $\xi(y) = \xi(o) = 1$. In this case, every positive

[37] Appendix F lays out the model with two-period uncertainty where those who enter the fever state know only after two periods the state at which they entered the period. This introduces uncertainty both about being infected and being recovered. The model loses some of its tractability because the level of outside activity in the current period matters for the continuation values multiple periods ahead. The state space thus becomes much larger. Qualitatively and even quantitatively, this robustness exercise has minor effects as already in our one-period model the entire duration of infectiousness was spanned by the uncertainty (see Table F15 which shows robustness of the main results in Table 7).

Table 7: The Effects of Testing

		Covid R_0 no testing (1)	Covid R_0 full testing (2)	$0.5 \times$ Covid R_0 no testing (3)	$0.5 \times$ Covid R_0 full testing (4)
Panel A. Realized statisitics					
Wks to peak infections	Young	10	45	48	1
	Old	11	46	48	1
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	7.42	3.86	1.05	0.05
	Old	15.32	11.68	4.83	0.27
Dead p/ 1,000 1 year	Young	1.15	0.44	0.11	0.01
	Old	19.75	7.60	2.25	0.19
	All	5.13	1.97	0.57	0.05
Dead p/ 1,000 LR (vacc. arrival)	Young	1.23	0.54	0.17	0.01
	Old	21.16	8.90	3.11	0.19
	All	5.49	2.33	0.80	0.05
Recovered in LR (vacc. arrival), %	Young	53.88	23.85	7.52	0.40
	Old	22.23	9.48	3.34	0.19
	All	47.11	20.77	6.63	0.35
GDP 1 year, % change w.r.t. no-disease		-6.16	-3.60	-1.86	-1.28
Panel B. Expected statistics					
Hrs @ home - expectation 3 years, diff. w.r.t. no-disease	Young	6.33	2.56	0.78	0.12
	Old	12.62	8.75	3.84	0.61
Dead p/ 1,000 LR (expectation, 3 years)	Young	0.97	0.37	0.11	0.01
	Old	16.29	6.05	2.11	0.18
	All	4.24	1.58	0.54	0.05
GDP, expectation 3 years, % change w.r.t. no-disease		-5.84	-3.17	-2.07	-1.69

Note: Simulations based on an economy without a lockdown policy. Vaccine arrival = 1.5 year.

case can be found and the individual isolated. In our benchmark for Covid, this is still not enough to eradicate the disease (see Table 7). The reason is that the isolation is not perfect. Individuals that test positive spend more time at home but still go out somewhat. Moreover, there is a fraction of exogenous contacts that cannot be avoided.[38] Nevertheless, this policy is still quite effective in decreasing death rates. A no-lockdown world without testing sees a death toll of 5.49 per 1,000 people. With full testing, this statistic goes down to 2.33. This reduction is achieved despite a smaller fall in GDP since individuals do not have to protect themselves as much. The no-testing world experiences a fall in GDP of 6.16% in the first year of the pandemic while in the full-testing scenario GDP decreases by only 3.6%.

However, depending on the characteristics of the disease, testing can indeed essentially stop a pandemic on its tracks. Consider a virus that is 50% less infectious than that of Covid; that is, its R_0 is half of that of Covid. Table 7 compares the no-lockdown equilibrium for this disease when there is no testing and with full testing where again every fever agent is tested. The no-testing equilibrium features a death toll equal to 0.8 per 1,000 people. With full testing, the death toll drops to only 0.05. Hence, full testing can nearly eradicate low-R_0 diseases, but not one as infectious as Covid.

[38] In a previous version of our paper we had shown that testing together with stricter isolation works quite well, but that was in a model without exogenous contacts (Brotherhood et al. 2020).

Next, we want to understand whether the availability of tests affects the optimal lockdown strategy. As we show in Section 7 (see Table 11 below), the optimal lockdown in a Covid pandemic is similar in terms of deaths averted with and without testing, but it achieves this outcome in different ways. Without testing, the planner must rely more heavily on broad restrictions, requiring the young to spend more time at home (about 2 additional hours per week in the first year). As a result, the lockdown is stricter and leads to larger output losses, with GDP falling by about 2 percentage points more relative to a scenario with testing, where isolation of detected cases allows for more targeted interventions.

6.1 Optimal Build-up of Testing Capacity

We have seen so far that testing and lockdowns interact: the availability of testing leads to much lighter optimal lockdowns. So a natural question is how a planner would choose both tests and lockdowns simultaneously.

In reality, tests come at a cost and it is also not feasible to build up an infinite amount of test capacity within one period. We therefore now modify our model by adding the build-up of testing infrastructure at a cost and let the planner choose optimal testing capacity and lockdowns simultaneously. Specifically, assume test capacity is zero at the beginning of the pandemic and it costs $f(k_t) = \kappa k_t^2$ to increase capacity by k_t units in a given week. Test capacity in week t is then $K_t = \sum_{i=1}^{t} k_i$, which implicitly assumes no depreciation. The maximum number of tests that can be used in period t is then K_t. There is also a marginal cost of using a test, p.

To operationalize this, we must discipline the parameters κ and p. We pick κ to match the outlays in the US during 2020. According to Figure 7, testing capacity increased approximately linearly until it reached 3.8% of the US population by the end of that year. The Department of Health and Human Services (HHS) spent $25 billion in 2020 for necessary expenses to research, develop, validate, manufacture, purchase, administer, and expand capacity for Covid-19 tests (U.S. Department of Health and Human Services 2020). Using these numbers as targets, we set $\kappa = 590.2$. There is no single price for a test in the data. The Health System Tracker provides a distribution of prices (Health System Tracker 2020). The 25th percentile is around $50. We use this somewhat lower bound to discipline p, as the HHS expenditures are quite comprehensive and a portion of the HHS resources may have been used to partially offset the unit cost of each test. See Appendix D.2 for details on this calibration.

To keep the planning problem tractable, we let the planner choose a target level of tests $\overline{K}$ and how quickly to reach this target. We assume the planner builds up test capacity linearly, such that $k_t = \overline{k}$ for all t in which $K_t = t\overline{k} \leq \overline{K}$.[39] So, the planner

[39] If an additional investment of $\overline{k}$ would exceed the desired capacity (i.e., $K_{t-1} \in (\overline{K} - \overline{k}, \overline{K}]$), the

chooses $\overline{k}$ and $\overline{K}$. We start with a version where the planner uses all tests up to the capacity limit each period.

We further assume that the costs associated with building the test capacity and implementing the tests are financed by perpetual lump sum taxes that are imposed once the pandemic ends (i.e., when a vaccine is found in our model). Assuming that a vaccine arrives in period T, the government's balanced budget condition is given by

$$\sum_{t=0}^{T-1} \frac{E_t}{(1+r)^t} = \sum_{t=T}^{\infty} \frac{L_T M_t}{(1+r)^t}. \tag{13}$$

The left-hand side represents the present value of testing expenditures, where the discount rate is $r = \beta^{-1} - 1$, and period-t expenditures are given by $E_t = \kappa k_t^2 + p\, M_t^{\text{tested}}$. The right-hand side represents the present value of government revenues. Here, L_T is a lump-sum tax used to finance testing expenditures when the vaccine arrives in period T, and M_t denotes the mass of agents alive in period t. This mass accounts for the fact that, after the pandemic ends, agents may die from natural causes as well as from COVID if they are infected at the time the vaccine arrives. Since the vaccine may arrive at different dates, there is a distinct lump-sum tax L_T for each possible arrival time. During the pandemic, agents' continuation values take into account that the lump-sum tax they will face depends on the realized timing of the vaccine arrival.

How would the economy change if the planner could choose the optimal build-up of testing capacity alongside the optimal lockdown? According to Figure 7, the planner chooses a much faster build-up than in reality. Already by July 2020, it is possible to test around 5.5% of the population each week, a level only approached by the US around January 2022.

To understand the effects of this faster ramp-up, Table 8 presents different scenarios. To interpret the results in this setting where testing capacity varies, we report several statistics relative to a particular comparison scenario: a laissez-faire equilibrium with no government intervention, i.e., no lockdowns and no tests.[40] Column (3) presents the results for optimal build-up of testing capacity and associated optimal lockdown. For comparison, it also contains two scenarios with "benchmark" tests, which we define as the maximum weekly number of tests performed in the US up to that point in time. This ensures that testing capacity in the benchmark is non-decreasing, consistent with our assumption in this section that test capacity does not depreciate.[41] The two comparison scenarios comprise the equilibrium with benchmark

actual investment in t just fills the remaining gap so that $k_t = \overline{K} - K_{t-1}$ and, therefore, $K_t = \overline{K}$.

[40] As discussed in Section 4, this laissez-faire scenario (Column 4 in Appendix Table B2) features more deaths, more precautions and lower GDP compared to our previous no-lockdown benchmark (Column 2 in Appendix Table B2). Both abstract from lockdowns but the latter still features some tests and associated isolation and, therefore, less disease.

[41] Benchmark tests are the upper envelope of the testing capacity in Figure 6b (ignoring the short-lived

lockdown and benchmark tests (Column 1), akin to our calibrated economy in Section 4, and the optimal lockdown with benchmark tests (Column 2), akin to the optimal lockdown in Section 5.[42]

spike in the test data of January 2022), as depicted in Figure 7.

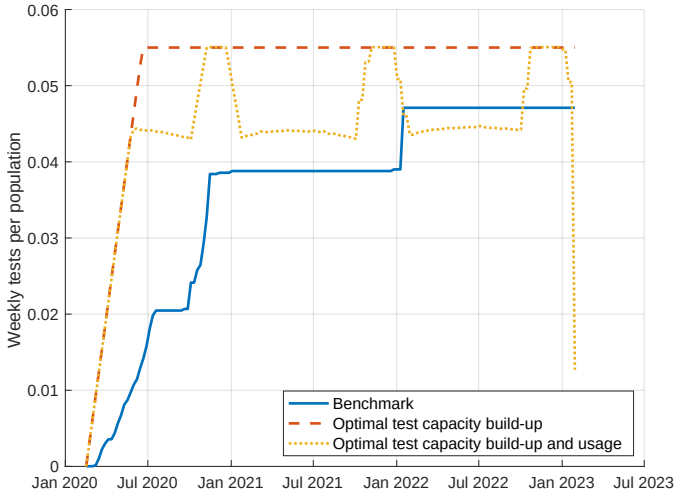
[42]Taxes and hours at home relative to a no-disease world in Column (1) and Column (2) of Table 8 are virtually identical to those in Column (2) and Column (1) in Table 6. This is not surprising as the only difference is that the testing capacity for Table 6 was sometimes decreasing, while the benchmark test capacity for Table 6 is constructed to be weakly increasing. We will later discuss a similar result that some reductions in test usage outside of the winter season have minimal impact when we will compare Columns (3) and (4) in Table 8.

Table 8: The Importance of Testing Capacity Build-up

		Benchmark lockdown Benchmark tests (1)	Optimal lockdown Benchmark tests (2)	Optimal lockdown Optimal test build-up (3)	Optimal lockdown Optimal test build-up Optimal test usage (4)
Panel A. Realized statistics					
Avg tax., first year	Young	0.17	0.26	0.20	0.20
	Old	0.17	0.31	0.26	0.26
Hrs @ home - avg. first year, diff. w.r.t. laissez-faire	Young	9.60	15.21	9.58	9.58
	Old	-3.90	-10.03	-10.75	-10.74
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	17.02	22.63	17	17
	Old	11.42	5.29	4.57	4.57
% Deaths averted, 1 year (rel. to laissez-faire)	Young	69.96	99.12	99.06	99.06
	Old	64.47	98.54	98.50	98.50
	All	65.44	98.64	98.60	98.60
% Change in GDP rel. to laissez-faire, 1 year		-7.25	-12.07	-7.28	-7.27
Panel B. Expected statistics					
Hrs @ home - diff. w.r.t. laissez-faire	Young	7.53	16.65	12.18	12.18
	Old	-3.07	-7.19	-7.69	-7.69
Hrs @ home - diff. w.r.t. no-disease	Young	13.86	22.98	18.51	18.51
	Old	9.55	5.43	4.93	4.94
% Deaths averted, expectation 3 yrs (rel. to laissez-faire)	Young	65.80	98.96	98.51	98.51
	Old	61.55	98.33	97.82	97.82
	All	62.31	98.44	97.95	97.95
% Change in GDP, expectation 3 yrs (rel. to laissez-faire)		-5.08	-11.14	-7.09	-7.08
Panel C. Welfare					
CEV rel. to laissez-faire (%)	Young	0.46	0.50	0.69	0.69
	Old	12.97	21.35	21.25	21.25

Note: The laissez-faire comparison scenario refers to an equilibrium in which there is no government intervention: no tests and no lockdowns. Benchmark tests denote the maximum weekly number of tests performed in the US up to that point in time.

Figure 7: Tests in equilibrium



Note: The benchmark line corresponds to the data and represents the upper envelope of the actual tests performed in the US.

In the first year, the results regarding hours at home, deaths averted and GDP under optimal test build-up and lockdown in Column (3) look like a combination of results from columns (1) and (2): For the old, hours at home are similar to those under optimal lockdown with benchmark tests in Column (2), and the number of deaths averted for both age groups also remain close to that. But the young are allowed around 6 extra weekly hours outside, and with this additional activity, GDP is about 5 percentage points higher relative to Column (2). In fact, hours at home for the young in the new optimum in Column (3) are comparable to those under the benchmark lockdown in Column (1), and so is GDP. So, optimal test capacity build-up allows the planner to retain much of the good outcomes for the old in particular and for disease suppression, while simultaneously retaining high output and mobility for the young as in the benchmark lockdown.

The results in Column (3) are achieved through time taxes that are substantially lower than those under the optimal lockdown with benchmark testing (Column 2) because more testing and isolation allows for disease suppression that otherwise needs to be achieved through lockdowns. These taxes, however, are higher than in the benchmark lockdown (Column 1) because individuals take less precautions themselves in the near absence of the disease. In terms of welfare, it is the young who benefit in particular, given their higher level of activity. The old retain most of the large welfare gains relative to Column (1), but welfare falls slightly short of that in Column (2) for the following reason: more testing increases the benefit of outside time for the young relative to the cost to the old, leading to a small welfare loss for the old. Since there are far fewer old individuals in the economy, the optimal combination of lockdown and testing still leads to sizable average welfare gains. This materializes even though

the cost of the faster test build-up and higher usage is taxed from the economy. By the end of 2020, the cost of the optimal test build-up is approximately 2.5 times as high as what was spent in reality, totaling around $63 billion. The average welfare increase from Column (2) to (3) arises because these costs are outweighed by the gains in GDP and outside leisure.

We solved for the planner's decision regarding the optimal build-up of testing capacity under the assumption that, once capacity was built, all tests would be used every week. However, since tests are costly to implement ($p > 0$), it may be optimal not to use the full capacity at all times. Solving for the optimal weekly test usage is a more complex problem, as it requires determining an entire time series of test usage. The solution is shown in Figure 7. The total testing capacity is virtually identical to the case where all tests are used. This capacity is fully utilized during winters, while usage in other seasons is reduced by roughly 20%. Column (4) in Table 8 reports several statistics for this scenario. Whether or not the planner is allowed to choose weekly test usage makes virtually no difference in terms of hours at home, deaths averted, or welfare, despite the non-trivial fluctuations in test usage (see Figure 7). This result arises because the marginal cost p of each test is relatively low. Moreover, during times when the planner reduces test usage, the benefits of doing so are roughly equal to the marginal cost of testing, leaving the overall economy nearly unchanged.

Individuals who test positive are isolated according to the tax rate $\bar{\tau}_i$. What would happen if the planner also chose this level of isolation? Appendix Table B6 provides the results of this experiment. The planner would choose a very high level of isolation, $\bar{\tau}_i = 0.99$, much higher than the calibrated value of 0.38. With more targeted isolation, the planner can relax the blanket lockdown and allow both age groups more time outside. To do that, the planner ramps up testing more quickly to suppress the disease, but stops at a lower level of testing capacity since not all capacity is necessary given the quick disease suppression. GDP increases, as does welfare, especially for the young. Such a high level of isolation may not be feasible in practice. However, these results point to the substantial gains from targeted isolation of infected individuals.[43]

Summing up, testing is not the magic bullet that eradicates the disease by itself, but testing eases the burden of the disease substantially. Second, the optimal lockdown changes with the testing regime: testing allows for a less restrictive lockdown and thus lower GDP losses. Third, the optimal testing capacity build-up is much faster than what was observed in the US during the Covid pandemic.

[43]One could additionally envision a robustness check that allows the planner to target tests more to the old or more to the young. We lay this out in an earlier working paper (Brotherhood et al. 2023), where we show that the planner gains from targeting the young, but omit it here for brevity.

7 Optimal Lockdown for Other Pandemics

We now examine how other diseases impact optimal lockdown. We start with an analysis of the Spanish Flu from 1918 and find that optimal restrictions would have been milder at the time, even though it was an overall more deadly disease. A decomposition of the two disease scenarios shows why. We then explore implications for possible future pandemics that may feature diseases with different characteristics.

7.1 Spanish Flu Compared to Covid

The Spanish flu was another deadly pandemic that hit the world about 100 years before Covid-19. Millions of people also died (Barro, Ursúa, and Weng 2022; Barry 2005). While some lockdown measures were implemented at the time (Markel et al. 2007), those were much shorter and milder compared to the severe lockdowns implemented in many countries in response to the coronavirus pandemic in 2020. An obvious question to ask is whether a more severe lockdown would have been better at the time. Or whether the disease itself, or other conditions, were so different from 2020 that a milder lockdown was indeed optimal. To answer this question, we now calibrate our model to the Spanish flu episode and solve for the optimal policy.

Table 9 summarizes how the Spanish flu pandemic differs from the Covid world. First, the population was much younger in the second decade of the 20th century: only 7.7% of the population was above 65 years old, versus 21.4% in 2020. The population was poorer as well, with an average income of 19% of that in 2020. Nowadays telework is possible due to a variety of technological advances. We thus assume that work from home was not possible in the 1910s. Each age group also faced a lower life expectancy, a few years lower than in 2020. While we assume an expected arrival rate of 1.5 years during the Covid pandemic, this time lag was much longer for the Spanish flu. We assume a 10 year expected arrival time for a vaccine/cure. Several modern tests (PCR and antigen) were available for Covid but not for the Spanish flu. We also assume the same seasonality pattern for both diseases and a slightly less infectious disease for the Spanish flu. The age gradient for the case fatality rate (CFR) for the Spanish flu was very different from Covid: The young were substantially more likely to die of the Spanish flu, by a factor of almost 10. The old, on the other hand, faced a lower CFR. This lower fatality for the old is partly due to a younger population among the old in the 1910s. Appendix D.1 provides more details for the calibrated parameters for the Spanish flu world.

We start by comparing the no-lockdown equilibria for the two pandemics in Table 10. To be clear, we compare equilibria taking the voluntary protective behaviors into account, which is quite different from comparing the two diseases through the lens of

Table 9: Covid vs. the Spanish Flu

Characteristic	Covid	Spanish flu
Fraction of old in population	0.214	0.077
Income relative to 2020	1	0.190
Is telework possible?	yes	no
Remaining life expectancy, young	38.9	32.4
Remaining life expectancy, old	12.9	8.7
Expected time until vaccine arrival, years	1.5	10
Are tests available?	yes	no
R_0, winter	3.0	2.7
Seasonality	yes	yes
Case fatality rate, young (%)	0.23	1.9
Case fatality rate, old (%)	9.00	4

Note: Appendix D.1 provides details for the Spanish flu calibration.

an epidemiological model without endogenous choices. The Spanish flu was a more deadly disease. Without any lockdown, almost twice as many people would have died during the Spanish flu than one hundred years later during Covid (8.71 versus 4.46 deaths per 1,000 people). During the first year of the pandemics, GDP losses would have been more than three times as high for the Spanish flu. The higher GDP losses are due to both the higher death rates, especially among the young, and the inability to telework at the time. Even though the overall death rates are much higher for the Spanish flu, this is not true when zooming in on the old specifically, who die in much larger numbers during Covid. This difference in the mortality age gradient drives differences in voluntary precautions in the two worlds. Without any lockdown policies, the young increase their time at home during the Spanish flu by 11 hours on average during the first year, compared to only 7 during Covid. The opposite is true for the old, who increase their time at home by only 4 hours during the Spanish flu, but 14 hours during Covid.

The Spanish flu and Covid differ along many dimensions (Table 9). Columns (3) through (9) of Table 10 each vary one dimension at a time, keeping all others at the values of the Covid calibration. For example, column (3) features an environment and disease exactly like in the Covid world, except that people are as poor as in the 1910s. An important thing to learn from Table 10 is that it is not the different features of the virus that cause mortality to be higher in the Spanish flu no-lockdown equilibrium. On the contrary, column (7) shows that just based on the deadliness of the virus, mortality would have been *lower* (only 2.49 per 1,000 people compared to 4.46 for Covid). Rather, a combination of other factors are responsible. First, teleworking was not possible at the time (Column 4), lowering the incentive for the young to engage in voluntary cautious behavior. This leads to more infections and more deaths. In fact, the disease

is so swift in such a case that, during the very early peak, the old self-protect more. But, given this swiftness, the disease is soon over and the old can return to leaving the house. These changes actually aggregate to the old spending *less* time at home during the first year of this counterfactual pandemic. Second, the inability to test people also contributes to the higher death rates (Column 8). Finally, both income (Column 3) and life-expectancy (Column 6) were lower one hundred years ago, leading people to value life less, and hence being less cautious voluntarily. The later vaccine arrival, on the other hand, plays a small role (Column 9). Together, these various factors lead to a death rate for the Spanish flu that is almost twice as high compared to Covid.

The different mortality age gradient has implications for policy. Table 11 shows that the optimal tax rate is higher for the Spanish flu, both for the old and the young. Yet, looking at tax rates alone is not enough. These taxes change the marginal condition between staying at home and going outside. However, other factors also matter for this decision; e.g., income levels or the ability to telework. More informative about the strictness of the policy is the induced change in actual hours at home. The additional hours at home (relative to a no-lockdown world) are almost 16 hours for the young under Covid compared to only 9 hours under the Spanish flu. So, in terms of restrictions for the young, the optimal lockdown for Covid is stricter than that for the Spanish flu. The same is not true for the old, who actually spend 9 *more* hours outside under the optimal Covid lockdown, compared to virtually no change under that of the Spanish flu. Both policies are quite successful in averting deaths, but much more so under Covid (98% for Covid and 78% for the Spanish flu) at the cost of a reduced GDP (12% and 19% respectively). The young disproportionately suffer from the lockdowns (since it reduces their ability to work) and the old gain disproportionately from the reduced mortality, especially under Covid. Hence, the welfare gains are distributed unevenly. Welfare measured in consumption equivalence units increases by 17% for the old and only 0.35% for the young under Covid. Since mortality rates are more similar across age groups during the Spanish flu, welfare gains are distributed more equally: 2.1% for the young compared to 4.6% for the old.

It may seem puzzling that even though mortality is much higher in the Spanish flu no-lockdown equilibrium compared to Covid, the optimal policy is laxer (smaller increase in hours at home, especially for the young). So what causes the optimal lockdown to be laxer for the young during the Spanish flu? Consider the decomposition in Table 11. The fact that there were fewer old people in 1910s plays a role (Column 5). The second factor is that mortality for the young was higher and hence voluntary precautions are higher, which requires less policy intervention. This can be seen in column (7) in Table 11 which shows the optimal policy for a Spanish flu virus in today's world. From Table 10 we know that equilibrium death rates are high in this world. Yet, optimal policy, measured by additional government-mandated hours at home for the

young, is quite lax. The reason is that infection and mortality probabilities are similar across age groups so that the cross-age externality is minimized. Put differently, the reason for a strict policy is that individually optimal and socially optimal behavior are quite different. This was precisely the case during Covid with young people spreading the disease and old people dying. With a different virus that affects people across the age distribution similarly, the discrepancy between socially and individually optimal behavior is much smaller.

Several forces in the decomposition in Table 11 point in the opposite direction (i.e., to a stricter lockdown): the fact that income was lower and telework did not exist (Columns 3 and 4), that testing was not available (Column 8), and that it would take much longer to develop a vaccine (column 9). That lower income and no ability to telework lead to a stricter lockdown may seem puzzling. The reason is that these features lead people to engage in less voluntary protective behavior and hence a stricter government mandated lockdown is needed.

Policy is only much stricter during Covid if measured as the gap to what people would be doing in the no-lockdown world. Another perspective is to compare behavior in the optimal policy world with what people were doing before the arrival of the disease. By this metric, the optimal policies are more similar for the two pandemics. Relative to a no-disease world, time at home increases by 22.6 hours for the young and 5.3 for the old under Covid, compared to 20 and 4.4 under the Spanish flu, which is not vastly different. This is also visible when we apply the thought experiment from Section 5. Consider individuals not yet aware of the Spanish flu and adjust their behavior according to the average first year taxes to recalculate R_0. This reduces R_0 from its normal level of 1.8 (2.7) down to 1 (1.59) in the summer (winter). Thus, even though tax rates are different, their impact on R_0 is quite similar in the two pandemics.

Table 10: Decomposition: No-lockdown Equilibrium

		Covid (1)	Spanish Flu (2)	1910s income (3)	No telework (4)	Fewer old (5)	Lower life expect. (6)	Different virus (7)	No testing (8)	Later vaccine (9)	Developing country (10)
Panel A. Realized statistics											
Weeks to peak infections	Young	10	10	9	10	10	10	44	10	9	10
	Old	10	11	10	11	10	10	44	11	10	10
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	6.87	10.81	6.10	2.40	6.78	6.47	15.70	7.42	6.62	1.79
	Old	14.47	4.31	7.39	10.21	14.13	13.14	4.91	15.32	14.30	4.03
Dead p/ 1,000, first year	Young	0.98	7.21	1.09	1.34	0.99	1	1.81	1.15	0.98	1.44
	Old	16.31	7.34	17.44	20.77	17.27	17.10	2.41	19.75	16.41	23.14
	All	4.26	7.22	4.59	5.50	2.24	4.44	1.94	5.13	4.29	3.11
Dead p/ 1,000 (by vaccine arrival)	Young	1.02	8.69	1.12	1.34	1.03	1.04	2.33	1.23	1.03	1.44
	Old	17.08	8.92	17.88	20.91	17.89	17.76	3.05	21.16	17.26	23.15
	All	4.46	8.71	4.71	5.53	2.33	4.61	2.49	5.49	4.50	3.11
Recovered , % (by vaccine arrival)	Young	44.84	45.80	49.19	58.71	45.04	45.58	12.37	53.88	45.22	62.90
	Old	17.89	23.16	18.64	21.62	18.70	18.87	7.79	22.23	18.06	24.09
	All	39.07	44.06	42.65	50.77	43.01	39.87	11.39	47.11	39.41	59.91
GDP 1 year, % change w.r.t. no-disease		-5.77	-20.44	-5.32	-5.54	-5.73	-5.75	-11.92	-6.16	-5.60	-4.75
Panel B. Expected statistics											
Hrs @ home, diff. w.r.t. no-disease	Young	6.10	5.43	5.73	2.58	6.09	5.83	13.70	6.33	3.09	2
	Old	11.87	2.27	6.21	9.89	11.56	10.84	4.30	12.62	6.76	4.39
Dead p/ 1,000	Young	0.81	8.17	0.92	1.17	0.82	0.83	1.77	0.97	1	1.27
	Old	13.30	8.37	14.33	17.82	14.05	13.94	2.31	16.29	16.58	20.16
	All	3.48	8.19	3.79	4.73	1.84	3.64	1.88	4.24	4.33	2.72
GDP, % change w.r.t. no-disease		-5.67	-13.73	-5.48	-6.21	-5.68	-5.81	-10.88	-5.84	-5.32	-5.53

Note: Expected time for vaccine arrival is 1.5 year for all columns, except "Spanish flu" and "Later vaccine", which is 10 years. Column (10) provides the results for a synthetic developing country with the same income, life expectancy and age composition as the US in the 1910s; same expected vaccine arrival rate as the US in 2020; no teleworking and no testing.

Table 11: Decomposition: Optimal Policy

		Covid (1)	Spanish Flu (2)	1910s income (3)	No telework (4)	Fewer old (5)	Lower life expect. (6)	Different virus (7)	No testing (8)	Later vaccine (9)	Developing country (10)
Panel A. Realized statistics											
Avg tax., first year	Young	0.26	0.48	0.48	0.61	0.24	0.26	0.23	0.29	0.27	0.28
	Old	0.31	0.42	0.05	0.56	0.28	0.30	0.26	0.33	0.30	0.20
Hrs @ home - avg. first year, diff. w.r.t. no-lockdown	Young	15.76	9.17	33.60	22.55	14.13	15.82	4.02	18.00	16.92	8.81
	Old	-9.16	0.05	-6.98	0.52	-8.94	-7.95	-0.78	-9.61	-9.33	1.96
Hrs @ home, avg - first year, diff. w.r.t. no-disease	Young	22.63	19.97	39.69	24.95	20.92	22.29	19.72	25.42	23.54	10.60
	Old	5.30	4.36	0.41	10.73	5.19	5.19	4.13	5.70	4.98	5.99
% Deaths averted, first year (rel. to no-lockdown)	Young	98.93	78.14	99.66	97.37	98.19	98.76	96.84	99.10	99.19	25.41
	Old	98.19	78.23	99.49	95.99	97.09	97.97	96.51	98.48	98.63	24.25
	All	98.32	78.15	99.52	96.26	97.54	98.11	96.75	98.59	98.73	24.75
% Change in GDP rel. to no-lockdown, 1 year		-12.44	-19.41	-24.33	-41.00	-10.87	-12.36	-3.60	-14.62	-13.44	-15.84
Panel B. Expected statistics											
Hrs @ home - diff. w.r.t. no-lockdown	Young	16.89	13.36	29.55	22.01	14.79	16.64	6.73	19.55	17.10	7.10
	Old	-6.38	0.24	-5.70	0.79	-6.27	-5.52	0.05	-6.70	-3.17	1.00
Hrs @ home - diff. w.r.t. no-disease	Young	22.99	18.79	35.28	24.58	20.88	22.47	20.43	25.88	20.19	9.10
	Old	5.49	2.52	0.51	10.68	5.29	5.32	4.35	5.93	3.59	5.40
% Deaths averted, rel. to no-lockdown	Young	98.36	74.27	99.60	96.09	97.17	98.06	96.47	98.67	98.99	27.09
	Old	97.40	74.54	99.38	94.44	95.79	97.09	96.14	97.87	98.36	25.94
	All	97.58	74.29	99.42	94.76	96.36	97.27	96.39	98.01	98.47	26.43
% Change in GDP, rel. to no-lockdown		-11.39	-26.25	-16.59	-39.79	-9.94	-11.09	-3.70	-15.05	-12.20	-11.20
Panel C. Welfare											
CEV rel. to no-lockdown (%)	Young	0.35	2.12	2.34	-1.00	0.46	0.36	1.58	0.29	0.07	0.04
	Old	16.97	4.61	12.44	20.94	17.57	17.82	2.24	21.25	21.84	4.15

Note: Expected time for vaccine arrival is 1.5 year for all columns, except "Spanish flu" and "Later vaccine", which is 10 years. Column (10) provides the results for a synthetic developing country with the same income, life expectancy and age composition as the US in the 1910s; same expected vaccine arrival rate as the US in 2020; no teleworking and no testing.

The optimal lockdown for Covid essentially strives for a no-Covid world as the policy averts virtually all deaths. However, this is not the case for the Spanish flu, in which the optimal policy achieves a decrease of around 78% of total deaths relative to a no-lockdown world. Interestingly, there is no single feature of the Spanish flu pandemic that solely explains this disparity across the two optimal policies. Every policy across columns (3)-(9) in Table 11 averts over 94% of deaths. Instead, it is a combination of factors that lead to a laxer lockdown for the Spanish flu pandemic. For example, the inability to telework as in Column (4) increases the costs of lockdowns while fewer old individuals as in Column (5) reduces the benefits of lockdowns. When both are present, a planner would optimally avert only a third of all deaths (see Appendix Table B7). The Spanish flu is not quite as extreme, still averting about 78% of all deaths, since some of the other features work in the opposite direction.

7.2 Covid in Developing Countries

The comparison between the Spanish flu and Covid-19 yields insights relevant for developing countries today. Some of the differences across time in the US are also present between the US and other countries today. For instance, in the late 1910s, the US income was about 17% of the 2020 level. This is similar to Algeria, Ecuador or Vietnam in the 2020s. The life expectancy in the US right before the Spanish flu pandemic was 51 years. This statistic in 2020 was below 55 years for countries like Somalia and Nigeria. We can thus use our model to explore the effects of the Covid pandemic for developing countries. We construct a synthetic developing country that has the same level of income, life expectancy and age distribution as the US in the late 1910s. Further, we assume telework and testing are not available, while expected vaccine arrival is the same as in the US.

The no-lockdown equilibrium for our synthetic developing country is reported in Column (10) in Table 10. Both the young and the old die in larger numbers. However, given that there are more young people and this group has a lower case fatality rate, the overall death rate is much lower. Despite the higher numbers of infections and deaths, both groups spend less time at home compared to the US. This increased level of activity translates into a milder fall in GDP.

The prescription for the optimal policy is also quite different from the US (see Column 10 in Table 11). The lockdown is much milder in the developing country: The young spend substantially less additional time at home (about 7 fewer hours) compared to the US (Column 1). This increased activity implies a lot more deaths. Compared to the optimal policy in the US in which the planner essentially follows a no-Covid strategy, only around 25% of deaths are averted in the developing country. Tax rates for the old are much lower than for the young, which is natural because the

old are much more afraid of the disease and therefore cut behavior even without the taxes. This effect is visible because optimal policy retains a significant risk of getting infected.[44] Since there is no telework, despite the increased activity by the young, GDP still falls by more than in the US. Finally, the welfare gains from the optimal policy are much smaller compared to the US.

7.3 Future Pandemics

Are there any general lessons to be learned for future pandemics? Several viruses exist that have the potential to cause a pandemic such as Ebola, Sars-Cov1, MERS, and even Tuberculosis.[45] These viruses are all quite different from each other. For example Ebola is a much more deadly disease but it is less infectious (Althaus 2014). MERS is not as deadly as Ebola but still has a much higher case fatality rate than Covid (Breban, Riou, and Fontanet 2013) and arguably is also more infectious (Choi et al. 2018). Tuberculosis also appears less infectious at least in developed country settings (Ma et al. 2018) and with a lower case fatality rate at least for the old (Straetemans et al. 2011). The next pandemic might be totally different yet. We now use our setup to draw conclusions for possible future pandemics.

Infectious diseases differ essentially along three dimensions: how contagious they are, how deadly they are, and whether (and how) they differ by age. We now use our model to study diseases with different levels of infectiousness (R_0) and case fatality rates (CFR). Table 12 displays mortality in no-lockdown scenarios and the percentage of deaths averted for various disease scenarios. Covid is in the center of the tables. Generally speaking, the higher the R_0 and the higher the CFR, the more people will die in a no-lockdown equilibrium. The higher mortality is, the larger the need for policy intervention is. However, whether mortality is high because of a high CFR or a high R_0 makes a difference. The higher the CFR, the larger are voluntary precautions, and hence the need for additional lockdowns is lower than for a more infectious disease (see Table 13). The reason is that the size of the externality increases with R_0. At the opposite extreme, for a highly lethal disease without any spill-overs, i.e., a disease where the risk can be minimized through own precautions alone, no policy intervention would be needed at all. Additional home hours imposed by the lockdown on the young are highest in the combination where CFR is only half that of Covid but R_0 is 50% higher. Yet the no-lockdown mortality rate is roughly similar to Covid, in fact even a bit lower (3.97 vs. 4.26). The reason is that voluntary adjustments are more

[44] For the US calibration, this is not visible in the taxes directly since optimal policy contains nearly no risk as infections remain very low. The different motives are visible in our usual way of comparing optimal policy with the no-lockdown world as the old already engage in a lot of protective behavior even without taxes in the no-lockdown scenario, where risk does remain present.

[45] We do not consider viruses here that are largely sexually transmitted, such as monkeypox and HIV, as the mechanisms and possible prevention strategies are quite different.

Table 12: Mortality, synthetic diseases

	Part A. Mortality (1st year) all ages, no lockdown			Part B. Deaths averted (1st year) all ages, optimal lockdown		
	CFR $= 0.5\times$ Covid CFR	CFR $= 1.0\times$ Covid CFR	CFR $= 1.5\times$ Covid CFR	CFR $= 0.5\times$ Covid CFR	CFR $= 1.0\times$ Covid CFR	CFR $= 1.5\times$ Covid CFR
$R_0 = 0.5 \times$ Covid R_0	0.13	0.21	0.28	72.74	75.28	75.51
$R_0 = 1.0 \times$ Covid R_0	2.59	4.26	5.25	97.77	98.32	98.28
$R_0 = 1.5 \times$ Covid R_0	3.97	7.03	9.13	98.30	98.79	98.82

Note: Part A. shows the mortality for all age groups during the first year of the pandemic for the no-lockdown equilibrium. Part B. shows the percentage of deaths averted during the first year under the optimal lockdown relative to the no-lockdown equilibrium. The different rows represent diseases with different levels of infectiousness (R_0), while the columns vary the case fatality rate (CFR).

sizable, and hence there is less need for government intervention.

Many optimal policies avert almost all deaths (96-99%), but this is not necessarily always the case (see Part B. in Table 12). Sometimes the cost of additional restrictions is simply too high. Or the benefit in terms of additional lives saved is too small. This is the case when mortality in a no-lockdown world is low due to a low R_0. In these cases, the ratio of the additional home hours through policy relative to the voluntary extra home hours is particularly high. The marginal benefit from an even stricter policy is too low relative to the costs it would impose.

Moreover, disease details matter for who gains most from the optimal policy. Welfare increases under Covid are small for the young and large for the old. Thus, perhaps not surprisingly there was much political pressure from the working age population to loosen restrictions early. Table 14 shows the welfare increase of the optimal policy for the young and the old, respectively, for various combinations of parameters. If CFR was only half of Covid but the infectiousness 1.5 times as high (i.e., the case discussed earlier where the externality is particularly relevant), then in fact the young lose from the optimal policy compared to the no-lockdown world. These results point to interesting political economy implications of infectious diseases.[46]

The exercises above systematically vary R_0 and the CFR while holding the age gradient constant. In contrast, Tables 15 and 16 hold both the R_0 and the aggregate CFR fixed, but change the age gradient. To interpret them, consider a researcher or policymaker at the onset of a pandemic who understands the two key aggregate epidemiological parameters: the infectiousness of the disease (R_0) and the population-weighted aggregate CFR.[47] Is that enough, or does it matter how this aggregate CFR is divided

[46]The full results of the experiments in Tables 12-14 are available in Appendix Tables B8 and B9.

[47]Here we hold the population-weighted CFR constant. Alternatively, one could think of a researcher who sees a given average CFR of infected agents at the beginning of the pandemic but might face different age gradients. To model this would require us to hold the time-outside-weighted average CFR constant, where time outside refers to the no-Covid time outside. Replicating columns 1-4 in Tables (15) and (16) with the alternative notion of constant time-outside-weighted average CFR has quantitatively

Table 13: Home hours, synthetic diseases

	Part A. Increase in home hours (1st year) young, no lockdown			Part B. Increase in home hours (1st year) young, optimal lockdown		
	CFR $= 0.5\times$ Covid CFR	CFR $= 1.0\times$ Covid CFR	CFR $= 1.5\times$ Covid CFR	CFR $= 0.5\times$ Covid CFR	CFR $= 1.0\times$ Covid CFR	CFR $= 1.5\times$ Covid CFR
R_0 = 0.5 $\times$ Covid R_0	0.19	0.32	0.41	1.89	2.17	2.46
R_0 = 1.0 $\times$ Covid R_0	3.67	6.87	9.20	18.36	15.76	13.88
R_0 = 1.5 $\times$ Covid R_0	4.11	8.50	12.23	27.73	23.50	20.54

Note: Part A. shows the average increase in home hours for the young during the first year in a no-lockdown equilibrium relative to a no-disease world. Part B. shows the average increase in home hours for the young during the first year under the optimal lockdown relative to the no-lockdown equilibrium. The different rows represent diseases with different levels of infectiousness (R_0), while the columns vary the case fatality rate (CFR).

Table 14: Welfare increase, synthetic diseases

	Young			Old		
	CFR $= 0.5\times$ Covid CFR	CFR $= 1.0\times$ Covid CFR	CFR $= 1.5\times$ Covid CFR	CFR $= 0.5\times$ Covid CFR	CFR $= 1.0\times$ Covid CFR	CFR $= 1.5\times$ Covid CFR
R_0 = 0.5 $\times$ Covid R_0	0.00	0.01	0.01	0.33	0.57	0.75
R_0 = 1.0 $\times$ Covid R_0	0.02	0.35	0.59	10.10	16.97	21.85
R_0 = 1.5 $\times$ Covid R_0	-0.59	-0.04	0.41	16.29	31.11	43.18

Note: Welfare is measured as Consumption Equivalent Variation (CEV) between the optimal lockdown and the no-lockdown equilibrium. The different rows represent diseases with different levels of infectiousness (R_0), while the columns vary the case fatality rate (CFR).

between the old and the young? That is, how important is the age gradient itself?

Covid has a strong age gradient where the CFR of the old is 39 times higher than that of the young. Consider the reverse where the CFR of the young is 39 times higher than that of the old, holding R_0 and aggregate population-weighted CFR constant. Column (1) in Table 15 shows the no-lockdown equilibrium for this case. The disease progresses more slowly, reaching its first peak four times later. This slower pace is driven by the behavior of the young who stay nearly 15 more hours per week at home compared to regular Covid. This roughly halves the number of deaths. This happens even though the old increase their time outside by nearly 15 hours and take essentially no precautions. Their mass is too small to offset the change in behavior of the young. Since only the young work, this lower death toll comes at a nearly threefold cost in lost GDP. This arises not only in the extreme case where the young die 39 times more than the old, but also when the young die twice as often (Column 2), equally as often (Column 3), or half as often (Column 4).[48] As deaths shift towards the old, the young

small effects and does not alter the qualitative points we discuss (see Tables B10 and B11).

[48]Column 4 closely resembles the "different disease" counterfactual for the Spanish flu in the previous section (Table 10, column 7). The Spanish flu had roughly twice the CFR of the old compared to the young with essentially unchanged population-weighted CFR, and only a mildly different R_0.

go out more and the old less, but with only minor effects relative to the other extreme (Covid) where the young die 39 times less (last column). The reason why columns 1-4 look so similar for the young is that all these scenarios hold the average population-weighted CFR constant. Since the young are a very large group, their own CFR does not change much except when the burden of the disease massively shifts to the old, as in standard Covid. In standard Covid, the CFR of the young is 0.23%, while it remains at 2.66, 2.36, 2.11 and 1.73 over columns 1-4. So going from the case where the young die twice as much as the old to the case where they die 39 times as much as the old does not change the young's CFR much, while it substantially drops the CFR of the old, but they are too small a group to substantially affect the disease dynamics.

Table 15: Different age gradient: No-lockdown Equilibrium

		CFR old/yng 1/39 (1)	CFR old/yng 1/2 (2)	CFR old/yng 1/1 (3)	CFR old/yng 2/1 (4)	CFR old/yng 39/1 (Covid)
Panel A. Realized statistics						
Weeks to peak infections	Young	44	44	44	43	10
	Old	43	43	43	43	10
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	22.35	21.50	20.73	19.47	6.87
	Old	0.12	1.93	3.47	5.77	14.47
Dead p/ 1,000, first year	Young	2.29	2.24	2.19	2.11	0.98
	Old	0.05	0.90	1.67	2.94	16.31
	All	1.81	1.95	2.08	2.29	4.26
Dead p/ 1,000 (by vaccine arrival)	Young	3.20	3.10	3.01	2.85	1.02
	Old	0.07	1.19	2.19	3.83	17.08
	All	2.53	2.69	2.83	3.06	4.46
Recovered , % (by vaccine arrival)	Young	12.24	13.41	14.61	16.84	44.84
	Old	10.41	10.61	10.83	11.32	17.89
	All	11.84	12.81	13.80	15.66	39.07
GDP 1 year, % change w.r.t. no-disease		-17.48	-16.76	-16.11	-15.07	-5.77
Panel B. Expected statistics						
Hrs @ home, diff. w.r.t. no-disease	Young	19.57	18.71	17.94	16.67	6.10
	Old	0.10	1.66	2.99	4.97	11.87
Dead p/ 1,000	Young	2.58	2.47	2.38	2.22	0.81
	Old	0.05	0.92	1.68	2.91	13.30
	All	2.04	2.14	2.23	2.37	3.48
GDP, % change w.r.t. no-disease		-15.59	-14.90	-14.28	-13.29	-5.67

Note: The different columns change the age gradient of the case fatality rate (CFR) while keeping the population-weighted average CFR constant and equal to that of Covid.

The age gradient also matters substantially for the optimal policy, as reported in Table 16. When the odds of dying between age groups are reversed relative to Covid holding average CFR constant (Column 1), the GDP costs of the optimal policy relative to no-lockdown are smaller. The reason is that the young are already doing a lot of precautions voluntarily in the no-lockdown equilibrium. The planner therefore has to restrict them relatively less. The old now have to be constrained much more and no longer spend more hours outside relative to the no-lockdown scenario, but fewer.

Table 16: Different age gradient: Optimal Policy

		CFR old/yng 1/39 (1)	CFR old/yng 1/2 (2)	CFR old/yng 1/1 (3)	CFR old/yng 2/1 (4)	CFR old/yng 39/1 (Covid)
Panel A. Realized statistics						
Avg tax., first year	Young	0.30	0.30	0.29	0.29	0.26
	Old	0.30	0.30	0.31	0.31	0.31
Hrs @ home - avg. first year, diff. w.r.t. no-lockdown	Young	4.31	4.73	5.12	5.82	15.76
	Old	4.73	3.01	1.55	-0.67	-9.16
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	26.66	26.22	25.85	25.28	22.63
	Old	4.85	4.95	5.01	5.10	5.30
% Deaths averted, first year (rel. to no-lockdown)	Young	94.82	95.28	95.68	96.29	98.93
	Old	95.30	95.48	95.65	95.97	98.19
	All	94.82	95.30	95.68	96.20	98.32
% Change in GDP rel. to no-lockdown, 1 year		-4.31	-4.67	-5	-5.57	-12.44
Panel B. Expected statistics						
Hrs @ home - diff. w.r.t. no-lockdown	Young	7.37	7.80	8.20	8.91	16.89
	Old	4.83	3.38	2.13	0.22	-6.38
Hrs @ home - diff. w.r.t. no-disease	Young	26.94	26.51	26.14	25.58	22.99
	Old	4.92	5.04	5.12	5.19	5.49
% Deaths averted, rel. to no-lockdown	Young	94.60	94.98	95.31	95.82	98.36
	Old	94.97	95.12	95.25	95.49	97.40
	All	94.60	94.99	95.30	95.73	97.58
% Change in GDP, rel. to no-lockdown		-5.06	-5.43	-5.76	-6.33	-11.39
Panel C. Welfare						
CEV rel. to no-lockdown (%)	Young	2.27	2.17	2.07	1.90	0.35
	Old	-0.11	0.71	1.48	2.81	16.97

Note: The different columns change the age gradient of the case fatality rate (CFR) while keeping the population-weighted average CFR constant and equal to that of Covid.

The effects on the young remain similar, though slightly less strong, in Columns (2)-(4) where the CFR ratio between the age groups is less severe, for the same reasons described in the no-lockdown equilibrium. For the old, time outside relative to no-lockdown increases as their chances of dying increase (going from Column 1 to Column 4), and they enjoy more time outside than under no-lockdown when they die twice as often as the young (Column 4). Interestingly, when roles are reversed and the young die twice as often as the old (Column 2) the young do not get extra time outside relative to no-lockdown. This is driven by the difference in overall time outside: the old already spend less time outside and therefore their externality on others is lower. So the overall GDP and welfare effects that the planner achieves relative to no policy is substantially affected by the age gradient. This also materializes in the welfare numbers. While under Covid the optimal policy delivers large welfare gains for the old and small gains for the young, this is reversed when the odds of dying are flipped as this reverses who enacts externalities on whom. In fact, the optimal policy in Column (1) comes with a welfare loss for the old because they have to stay at home so much more without any noticeable mortality gains for themselves. Since policy in all scenarios brings prevalence to very low levels (i.e., constitute "no-disease" policies), deaths

remain relatively low.[49]

7.4 Summary of Lessons Learned

Four general lessons follow from the analyses above. First, lockdowns should be strict when R_0 is high, and less so when only the CFR is high. Second, the age gradient matters. When the CFR is high for the young (a large and active group), then less additional restrictions are needed because more voluntary precautions are taken. Third, the optimal lockdown depends on economic conditions. If the size of the old population is small, life expectancy is low, or teleworking is easy, then the optimal policy is less restrictive. Finally, the optimal policy does not always avoid all deaths and the welfare benefits from the optimal policy can be very unevenly distributed across the two age groups.

8 Conclusions

This paper provides a comprehensive economic framework to address the challenges of formulating pandemic policies, recognizing the unique attributes of infectious diseases and their varying impacts across different age groups. The analysis underscores the importance of considering age-specific behaviors, the role of testing strategies, and lockdown policies. These seem to be first order in the spread of infections and the deadliness of infectious diseases.

We first calibrate our model using data from the Covid-19 pandemic in the US in the 2020s. The implemented lockdown together with voluntary precautionary behavior combine to decrease the death toll by 80% relative to an epidemiological world with no behavioral changes. The optimal lockdown, however, would have been even stricter. The optimal policy would particularly lock down the young, as this is a large and more active group that, due to the lower risk they face, do not engage in enough protective behavior and thus impose externalities onto others.

By modeling the uncertainty about an individual's health status, testing becomes a valuable component in the pandemic response toolkit. With testing, it is possible to isolate the positive cases in a targeted fashion. Hence, testing enables a less restrictive optimal lockdown, minimizes GDP losses, and facilitates a swifter easing of restric-

[49] Because all policies are essentially no-disease scenarios, the time tax is not much affected by the age-gradient, because there is no actual threat of the disease under the optimal policy and the externalities from one agent onto the others depend on R_0 and the average CFR in this case. This is very different if substantial Covid-risk remains in the optimal policy as the planner would only need to add taxes to correct the parts that are not internalized by the agents' own interest for self-protection. For example, in the case of the synthetic developing country introduced in column 10 in Table 11 a similar exercise reveals that taxes for the old increase by 11 percentage points as their infection risk drops.

tions. A social planner would optimally choose a faster build-up of testing capacity relative to what was observed in the US during the Covid pandemic.

The framework is general enough to be applied to other diseases. We then calibrate the model to the Spanish flu of the 1910s and find that the optimal lockdown then would have been less strict than that of Covid. The study of different diseases implies that lockdowns should be strict when infectiousness is high and less so when only the deadliness is high. Moreover, the age gradient of the disease matters: when the young are more at risk, this larger group engages in more protective behavior. Hence, there is less need for intervention.

In sum, this paper provides a comprehensive understanding of the interplay between age-specific behaviors, testing strategies, and policies in the context of infectious diseases. The model is richer than many existing counterparts, but remains sufficiently tractable to build upon in future work. By encompassing different groups, the framework is able to capture heterogeneity in risk across individuals. Age is a first-order driver of risk for many infectious diseases, including Covid. We therefore endow our risk groups with income and life expectancy that capture working age individuals and the elderly. However, our analysis could be adapted to capture other groups with different risks by adjusting the necessary remaining features. For example, explicitly adding children as a third age group with close to zero mortality risk would be a promising avenue to pursue. Our setup could also be used to study other dimensions of heterogeneity such as education, sectors, and gender.[50] Another form of heterogeneity may be in the beliefs about the severity of a disease. If some people have wrong beliefs about the risk of dying, they have a lower incentive to protect themselves (see Greenwood et al. (2019) for an analysis along these lines in the context of HIV and Eichenbaum et al. (2024) for the Covid pandemic in Portugal). The existence of such a group of people will have implications for optimal policy. These extensions are part of a broader set of topics that warrant attention in future research.

Data Availability Statement

The data and code underlying this article are available in the replication package at https://doi.org/10.5281/zenodo.20538371.

References

Acemoglu, Daron, Victor Chernozhukov, Ivan Werning, and Michael D. Whinston. 2021. "Optimal Targeted Lockdowns in a Multigroup SIR Model." *American Eco-*

[50] Education and income are highly correlated with the ability to work from home (Adams-Prassl et al. 2022) and women are hit more by employment losses (Alon et al. 2020a).

nomic Review: Insights 3 (4): 487–502.

Acemoglu, Daron, Azarakhsh Malekian, and Asu Ozdaglar. 2016. "Network security and contagion." *Journal of Economic Theory* 166:536 – 585.

Adams-Prassl, Abi, Teodora Boneva, Marta Golin, and Christopher Rauh. 2022. "Work That Can Be Done from Home: Evidence on Variation within and across Occupations and Industries." *Labour Economics* 74, no. 102083.

Akbarpour, Mohammad, Eric Budish, Piotr Dworczak, and Scott Duke Kominers. 2023. "An Economic Framework for Vaccine Prioritization." *The Quarterly Journal of Economics*, 06, qjad022.

Almås, Ingvild, Tessa Bold, Tillmann von Carnap, Selene Ghisolfi, and Justin Sandefur. 2023. "The macroeconomics of pandemics around the world: Lives versus livelihoods revisited." *Journal of Development Economics* 163:103099.

Alon, Titan, Matthias Doepke, Jane Olmstead-Rumsey, and Michèle Tertilt. 2020a, August. "This Time It's Different: The Role of Women's Employment in a Pandemic Recession." NBER Working Paper 27660.

Alon, Titan, Minki Kim, David Lagakos, and Mitchell VanVuren. 2020b. "How should policy responses to the Covid-19 pandemic differ in the developing world?" *Covid Economics* 22:1–46.

Althaus, Christian L. 2014. "Estimating the Reproduction Number of Ebola Virus (EBOV) During the 2014 Outbreak in West Africa." *PLoS currents*, vol. 6.

Altindag, Onur, Bilge Erten, and Pinar Keskin. 2022. "Mental Health Costs of Lockdowns: Evidence from Age-Specific Curfews in Turkey." *American Economic Journal: Applied Economics* 14 (2): 320–43 (April).

Alvarez, Fernando, David Argente, and Francesco Lippi. 2021. "A Simple Planning Problem for COVID-19 Lock-down, Testing, and Tracing." *American Economic Review: Insights* 3 (3): 367–82 (September).

Aum, Sangmin, Sang Yoon (Tim) Lee, and Yongseok Shin. 2022. "Who Should Work from Home during a Pandemic? The Wage-Infection Trade-off." Technical Report 104(2), Federal Reserve Bank of St. Louis Review.

Barro, Robert J., José F. Ursúa, and Joanna Weng. 2022. "Macroeconomics of the Great Influenza Pandemic, 1918-1920." *Research in Economics* 76 (1): 21–29.

Barry, John M. 2005. *The Great Influenza: The Story of the Deadliest Pandemic in History*. New York: Penguin Books.

Belot, Michele, Syngjoo Choi, Julian Jamison, Nicholas Papageorge, Egon Tripodi, and Eline van den Broek-Altenburg. 2020. Six-country Survey on Covid-19. https://osf.io/aubkc. Accessed: 2023-09-10.

Berger, David W, Kyle F Herkenhoff, Chengdai Huang, and Simon Mongey. 2020. "Testing and Reopening in an SEIR Model." *Review of Economic Dynamics* 43:1–21.

Bick, Alexander, Adam Blandin, and Karel Mertens. 2023. "Work from Home After the COVID-19 Outbreak." *American Economic Journal: Macroeconomics* 15 (4): 1–39.

Biggs, Andrew G., and Glenn R. Springstead. 2008. "Alternate Measures of Replacement Rates for Social Security Benefits and Retirement Income." *Social Security Bulletin* 68, no. 2.

Bognanni, Mark, Douglas Hanley, Daniel Kolliner, and Kurt Mitman. 2020, September. "Economics and Epidemics: Evidence from an Estimated Spatial Econ-SIR Model." Finance and economics discussion series 2020-091, Board of Governors of the Federal Reserve System.

Boppart, Timo, Karl Harmenberg, John Hassler, Per Krusell, and Jonna Olsson. 2025. "Integrated Epi-Econ Assessment: Quantitative Theory." *Quantitative Economics* 16 (1): 89–131.

Boppart, Timo, Karl Harmenberg, Per Krusell, and Jonna Olsson. 2022. "Integrated epi-econ assessment of vaccination." *Journal of Economic Dynamics and Control* 140:104308.

Breban, Romulus, Julien Riou, and Arnaud Fontanet. 2013. "Interhuman transmissibility of Middle East respiratory syndrome coronavirus: estimation of pandemic risk." *Lancet* 382:694–9.

Brotherhood, Luiz, Tiago Cavalcanti, Daniel Da Mata, and Cezar Santos. 2022. "Slums and pandemics." *Journal of Development Economics* 157:102882.

Brotherhood, Luiz, Philipp Kircher, Cezar Santos, and Michèle Tertilt. 2020, May. "An economic model of the Covid-19 epidemic: The importance of testing and age-specific policies." CEPR discussion paper No. DP14695.

Brotherhood, Luiz, Philipp Kircher, Michele Tertilt, and Cezar Santos. 2021. "An Economic Model of the Covid-19 pandemic with young and old agents: Behavior, testing and policies." *LIDAM Discussion Paper CORE 2021/34*.

———. 2023. "Optimal Age-based Policies for Pandemics: An Economic Analysis of Covid-19 and Beyond." *IDB Working Paper Series IDB-WP-1557*.

Brotherhood, Luiz, and Cezar Santos. 2022. "Vaccines and variants: A comment on 'Optimal age-based vaccination and economic mitigation policies for the second phase of the Covid-19 pandemic'." *Journal of Economic Dynamics and Control* 140:104303.

Butler, Rachel, Mauricio Monsalve, Geb W. Thomas, Ted Herman, Alberto M. Segre, Philip M. Polgreen, and Manish Suneja. 2018. "Estimating Time Physicians and Other Health Care Workers Spend with Patients in an Intensive Care Unit Using a Sensor Network." *The American Journal of Medicine* 131 (8): 972.e9–972.e15.

CDC, Centers for Disease Control. 2021. Estimated Disease Burden of Covid-19. https://www.cdc.gov/TemplatePackage/contrib/widgets/micrositeCollectionViewerMed/in Updates-from-the-CDC&csearch=&chash=! Accessed: 2023-11-29.

———. 2022. Flu Season. https://www.cdc.gov/flu/about/season/. Accessed: 2023-07-21.

———. 2023. "Isolation and Precautions for People with COVID-19." Report.

Centers for Disease Control and Prevention. 2021. COVID-19 Pandemic Planning Scenarios. https://www.cdc.gov/coronavirus/2019-ncov/hcp/planning-scenarios.html. Accessed May 12, 2026.

Chan, Tat, Barton Hamilton, and Nicholas Papageorge. 2016. "Health, Risky Behavior and the Value of Medical Innovation for Infectious Disease." *Review of Economic Studies* 83 (3): 1737–1755.

Chari, Varadarajan V, Patrick J Kehoe, and Ellen R McGrattan. 2007. "Business cycle accounting." *Econometrica* 75 (3): 781–836.

Chen, Frederick. 2012. "A Mathematical Analysis of Public Avoidance Behavior During Epidemics Using Game Theory." *Journal of Theoretical Biology* 302:18–28.

Choi, S., E. Jung, B.Y. Choi, Y.J. Hur, and M. Ki. 2018. "High reproduction number of Middle East respiratory syndrome coronavirus in nosocomial outbreaks: mathematical modelling in Saudi Arabia and South Korea." *Journal of Hospital Infection* 99 (2): 162–168.

Dizioli, Allan, and Roberto Pinheiro. 2021. "Information and Inequality in the Time of a Pandemic." *Journal of Economic Dynamics and Control* 130:104202.

Eichenbaum, Martin, Miguel Godinho de Matos, Francisco Lima, Sergio Rebelo, and Mathias Trabandt. 2024. "Expectations, Infections, and Economic Activity." *Journal of Political Economy* 132 (8): 2571–2611.

Eichenbaum, Martin S, Sergio Rebelo, and Mathias Trabandt. 2021. "The Macroeconomics of Epidemics." *The Review of Financial Studies* 34 (11): 5149–5187.

———. 2022. "The Macroeconomics of Testing and Quarantining." *Journal of Economic Dynamics and Control* 138, no. 104227.

Fajgelbaum, Pablo D., Amit Khandelwal, Wookun Kim, Cristiano Mantovani, and Edouard Schaal. 2021. "Optimal Lockdown in a Commuting Network." *American Economic Review: Insights* 3 (4): 503–22 (December).

Farboodi, Maryam, Gregor Jarosch, and Robert Shimer. 2021. "Internal and External Effects of Social Distancing in a Pandemic." *Journal of Economic Theory* 196, no. 105293.

Favero, Carlo, Andrea Ichino, and Aldo Rustichini. 2020. "Restarting the economy while saving lives under Covid-19." CEPR Discussion Paper DP14664.

Ferguson, Neil M., Derek A. T. Cummings, Christophe Fraser, James C. Cajka, Philip C. Cooley, and Donald S. Burke. 2006. "Strategies for mitigating an influenza pandemic." *Nature* 442:448–452.

Ferguson, Neil M., Daniel Laydon, Gemma Nedjati-Gilani, Natsuko Imai, Kylie Ainslie, Marc Baguelin, ..., and Azra C Ghani. 2020, 19 March. "Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand." Technical Report, Imperial College.

Fernández-Villaverde, Jesús, and Charles I. Jones. 2022. "Estimating and simulating a SIRD Model of COVID-19 for many countries, states, and cities." *Journal of Economic Dynamics and Control* 140:104318.

Galeotti, Andrea, and Brian R. Rogers. 2012. "Immunization and Group Structure." *American Economic Journal-Microeconomics, 5(2): 1-32.* 5 (2): 1–32.

Garibaldi, Pietro, Espen R. Moen, and Christopher A. Pissarides. 2020. "Modelling contacts and transitions in the SIR epidemics model." *Covid Economics* 5 (April): 1–20.

Garibaldi, Pietro, Espen R Moen, and Christopher A Pissarides. 2023. "Static and dynamic inefficiencies in an optimizing model of epidemics." *Economic Theory*, pp. 1–40.

Garriga, Carlos, Rody Manuelli, and Siddhartha Sanghi. 2022. "Optimal management of an epidemic: Lockdown, vaccine and value of life." *Journal of Economic Dynamics and Control* 140:104351.

Giagheddu, Marta, and Andrea Papetti. 2023. "The macroeconomics of age-varying epidemics." *European Economic Review* 151:104346.

Giannitsarou, Chryssi, Stephen Kissler, and Flavio Toxvaerd. 2021. "Waning Immunity and the Second Wave: Some Projections for SARS-CoV-2." *American Economic Review: Insights* 3 (3): 321–38 (September).

Glover, Andrew, Jonathan Heathcote, and Dirk Krueger. 2022. "Optimal age-Based vaccination and economic mitigation policies for the second phase of the covid-19 pandemic." *Journal of Economic Dynamics and Control* 140:104306. Covid-19 Economics.

Glover, Andrew, Jonathan Heathcote, Dirk Krueger, and José-Víctor Ríos-Rull. 2023. "Health versus wealth: On the distributional effects of controlling a pandemic." *Journal of Monetary Economics* 140:34–59.

Glynn, Judith R, and Paul A H Moss. 2020. "Systematic analysis of infectious disease outcomes by age shows lowest severity in school-age children." *Scientific data* 7 (1): 329.

Gollier, Christian. 2020a. "Cost-benefit analysis of age-specific deconfinement strategies." *Journal of Public Economic Theory* 22 (6): 1746–1771.

———. 2020b. "If the Objective is Herd Immunity, on Whom Should it be Built?" *Environmental and Resource Economics* 76:671–683.

Google LLC. 2022. COVID-19 Community Mobility Reports. https://www.google.com/covid19/mobility/. Accessed May 12, 2026.

Greenwood, Jeremy, Philipp Kircher, Cezar Santos, and Michèle Tertilt. 2013, March. "An Equilibrium Model of the African HIV/AIDS Epidemic." Working paper 43, National Bureau of Economic Research.

———. 2017. "The Role of Marriage in Fighting HIV: A Quantitative Evaluation for Malawi." *American Economic Review* 117 (5): 158–162 (May).

———. 2019. "An Equilibrium Model of the African HIV/AIDS Epidemic." *Econometrica* 87 (4): 1081–1113 (July).

Groningen Growth and Development Centre. 2020. Maddison Project Database 2020. https://www.rug.nl/ggdc/historicaldevelopment/maddison/releases/maddison-project-database-2020. Accessed May 12, 2026.

Health System Tracker. 2020. Prices for COVID-19 Testing. https://www.healthsystemtracker.org/brief/prices-for-covid-19-testing/. Accessed May 12, 2026.

Heikkinen, Terho, and Asko Järvinen. 2003. "The common cold." *The Lancet* 361 (9351): 51–59.

Hoogeveen, Martijn J., and Ellen K. Hoogeveen. 2021. "Comparable seasonal pattern for COVID-19 and flu-like illnesses." *One Health* 13:100277.

Hsieh, Chang-Tai, and Peter J Klenow. 2009. "Misallocation and manufacturing TFP in China and India." *The Quarterly journal of economics* 124 (4): 1403–1448.

Hur, Sewon. 2023. "The Distributional Effects of Covid-19 and Optimal Mitigation Policies." *International Economic Review* 64 (1): 261–294.

Kaplan, Greg, Benjamin Moll, and Giovanni L. Violante. 2020. "The Great Lockdown and the Big Stimulus: Tracing the Pandemic Possibility Frontier for the U.S." *NBER Working Paper Series 27794*.

Keppo, Jussi, Elena Quercioli, Marianna Kudlyak, Lones Smith, and Andrea Wilson. 2021. "The Behavioral SIR Model, with Applications to the Swine Flu and COVID-19 Pandemics." Unpublished Manuscript, Wisconsin.

Kermack, W. O., and A. G. McKendrick. 1927. "A Contribution to the Mathematical Theory of Epidemics." *Proceedings of the Royal Society A* 114 (772): 700–721.

Kopecky, Karen A. 2011. "The trend in retirement." *International Economic Review* 52 (2): 287–316.

Kremer, Michael. 1996. "Integrating Behavioral Choice into Epidemiological Models of AIDS." *The Quarterly Journal of Economics* 111 (2): 549–573 (05).

Krupnick, Alan, Anna Alberini, Maureen Cropper, Nathalie Simon, Bernie O'Brien, Ron Goeree, and Martin Heintzelman. 2002. "Age, Health and the Willingness to Pay for Mortality Risk Reductions: A Contingent Valuation Survey of Ontario Residents." *Journal of Risk and Uncertainty* 24:161–186.

Ma, Y., C.R. Horsburgh Jr, L.F. White, and H.E. Jenkins. 2018. "Quantifying TB transmission: a systematic review of reproduction number and serial interval estimates for tuberculosis." *Epidemiology and infection* 146:1478–1494.

Maloney, William, and Temel Taskin. 2020. "Determinants of Social Distancing and Economic Activity during COVID-19: A Global View." *Covid Economics*, no. 13:156–176.

Markel, Howard, Harvey B. Lipman, J. Alexander Navarro, Alexandra Sloan, Joseph R. Michalsen, Alexandra Minna Stern, and Martin S. Cetron. 2007. "Nonpharmaceutical Interventions Implemented by US Cities During the 1918-1919 Influenza Pandemic." *JAMA* 298 (6): 644–654 (08).

McAdams, David. 2020. "Nash SIR: An Economic-Epidemiological Model of Strategic Behavior During a Viral Epidemic." *Covid Economics* 16:115–134.

Melosi, Leonardo, and Matthias Rottner. 2023. "Pandemic Recessions and Contact Tracing." *Journal of the European Economic Association* 21 (6): 2485–2517.

Mills, Christina E., James M. Robins, and Marc Lipsitch. 2004. "Transmissibility of 1918 pandemic influenza." *Nature* 432:904–906.

Mossong, Joel, Niel Hens, Mark Jit, Philippe Beutels, Kari Auranen, Rafael Mikolajczyk, et al. 2008. "Social Contacts and Mixing Patterns Relevant to the Spread of Infectious Diseases." *PLoS Med* 5 (3): e74.

National Center for Health Statistics. 2026. Provisional COVID-19 Deaths by Week, Sex, and Age. Centers for Disease Control and Prevention, https://data.cdc.gov/NCHS/Provisional-COVID-19-Deaths-by-Week-Sex-and-Age/vsak-wrfu. Accessed May 12, 2026.

Opportunity Insights. 2026. Track the Recovery. https://tracktherecovery.org/. Accessed May 12, 2026.

Our World in Data. 2022. COVID-19 Testing. https://ourworldindata.org/coronavirus-testing. Accessed May 12, 2026.

Oxford COVID-19 Government Response Tracker. 2020. Coronavirus Government Response Tracker. https://www.bsg.ox.ac.uk/research/covid-19-government-response-tracker. Accessed May 12, 2026.

Piguillem, Facundo, and Liyan Shi. 2022. "The Optimal COVID-19 Quarantine and Testing Policies." *The Economic Journal* 132 (647): 2534–2562.

Quercioli, Elena, and Lones Smith. 2006. "Contagious Matching Games." Unpublished Manuscript, University of Michigan.

Rizzo, Caterina, Massimo Fabiani, Richard Amlôt, Ian Hall, Thomas Finnie, G James Rubin, Radu Cucuiu, Adriana Pistol, Florin Popovici, Rodica Popescu, et al. 2013. "Survey on the likely behavioural changes of the general public in four European countries during the 2009/2010 pandemic." *Modeling the interplay between human behavior and the spread of infectious diseases*, pp. 23–41.

Roser, Max. 2021. What Is the COVID-19 Stringency Index? Our World in Data, http://ourworldindata.org/covid-stringency-index. Accessed May 12, 2026.

Social Security Administration. 2019. Social Security Actuarial Life Table. https://www.ssa.gov/oact/STATS/table4c6.html. Accessed May 12, 2026.

Stein, Caroline, Hasan Nassereldine, Reed JD Sorensen, Joanne O Amlag, Catherine Bisignano, Sam Byrne, Emma Castro, Kaleb Coberly, James K Collins, Jeremy Dalos, et al. 2023. "Past SARS-CoV-2 infection protection against re-infection: a systematic review and meta-analysis." *The Lancet* 401 (10379): 833–842.

Straetemans, Masja, Philippe Glaziou, Ana L. Bierrenbach, Charalambos Sismanidis, and Marieke J.van der Werf. 2011. "Assessing tuberculosis case fatality ratio: a meta-analysis." *PloS one* 6, no. 6.

Taubenberger, Jeffery K., and David M. Morens. 2006. "Influenza: the mother of all pandemics." *Emerging Infectious Diseases* 12:15–22.

Toxvaerd, Flavio. 2019. "Rational Disinhibition and Externalities in Prevention." *International Economic Review* 60 (4): 1737–1755 (1).

U.S. Bureau of Labor Statistics. 2018. American Time Use Survey: Time Spent in Detailed Primary Activities and Percent of the Civilian Population Engaging in Each Activity, Averages per Day by Sex, 2018 Annual Averages. https://www.bls.gov/tus/tables/a1-2018.pdf. Accessed May 12, 2026.

———. 2020. Consumer Expenditure Survey, Table 1300, Age of Reference Person: Annual Expenditure Means, Shares, Standard Errors, and Coefficients of Variation, 2018. https://www.bls.gov/cex/tables/calendar-year/mean-item-share-average-standard-error/reference-person-age-ranges-2018.pdf. Accessed May 12, 2026.

———. 2021. American Time Use Survey, 2019 and 2020. https://www.bls.gov/tus/. Accessed May 12, 2026.

———. 2026a. BLS Data Finder. https://data.bls.gov/cgi-bin/srgate. Accessed May 12, 2026.

———. 2026b. Employment-Population Ratio [EMRATIO]. Retrieved from FRED, Federal Reserve Bank of St. Louis, https://fred.stlouisfed.org/series/EMRATIO. Accessed May 12, 2026.

U.S. Census Bureau. 2018. Annual Estimates of the Resident Population for Selected Age Groups by Sex for the United States, States, Counties, and Puerto Rico Commonwealth and Municipios: April 1, 2010 to July 1, 2018. https://www2.census.gov/programs-surveys/popest/tables/2010-2018/national/asrh/PEPAGESEX.pdf. Accessed May 12, 2026.

U.S. Department of Health and Human Services. 2020. COVID-19 Testing Expenditures in 2020. https://case.house.gov/uploadedfiles/covid19_bill_4_summary.pdf. Summary of COVID-19 testing-related appropriations.

Vandenbroucke, Guillaume. 2021. "The Mechanics of Individually- and Socially-Optimal Decisions during an Epidemic." *The B.E. Journal of Macroeconomics* 22:131 – 158.

Verity, Robert, Lucy C Okell, Ilaria Dorigatti, Peter Winskill, Charles Whittaker, Natsuko Imai, ..., and Neil M Ferguson. 2020. "Estimates of the severity of coronavirus disease 2019: a model-based analysis." *The Lancet Infectious Diseases*.

von Thadden, Ernst-Ludwig. 2020. "A simple, non-recursive model of the spread of Covid-19 with applications to policy." *Covid Economics* 10 (April): 24–43.

WHO. 2022. History of the Influenza Vaccine. https://www.who.int/news-room/spotlight/history-of-vaccination/history-of-influenza-vaccination. Accessed: 2022-09-10.

World Health Organization. 2026. WHO COVID-19 Dashboard. https://covid19.who.int/. Accessed May 12, 2026.

Online Appendix - Not for Publication

Optimal Age-based Policies for Pandemics:
An Economic Analysis of Covid-19 and Beyond

by Luiz Brotherhood, Philipp Kircher, Cezar Santos and Michèle Tertilt

A Laws of Motion and Aggregation

To define the laws of motion, denote the measure of agents of each type j of age a in period t by $M_t(j,a)$. Let $\mathcal{M}_t$ be the set of these for all j and a. Further, let $n_t(j,a,\mathcal{V}_t)$ and $\ell_t(j,a,\mathcal{V}_t)$ denote their times spent outside the house in equilibrium, which depends on infection state and age as well as implicitly on whether a vaccine is available at the beginning of period t (denoted by $\mathcal{V}_t \in \{0,1\}$). For some transmissions it also depends on whether a vaccine arrives between periods t and $t+1$, which follows a Markov chain with probability $\chi(0)$. Let $\mathcal{N}_t$ be the set of these equilibrium time allocations in period t for all j and a. The law of motion is a mapping from the state vector and equilibrium actions and the infection rates in period t into the number of agents of each type $\mathcal{M}_{t+1}$ in the next period. Call this map Ω, so that

$$\mathcal{M}_{t+1} = \Omega(\mathcal{M}_t(\cdot), \mathcal{N}_t(\cdot), \Pi_t(\cdot), T_t(\cdot), \xi_t(\cdot), \mathcal{V}_{t+1}, \mathcal{V}_t). \tag{14}$$

As mentioned in the main text, it simplifies the accounting to introduce two separate sub-states of the fever state: $j = f_s$ for those with fever who are susceptible (called fever-susceptible) and $j = f_i$ for those who are infected with Covid-19 (called fever-infected). Agents do not know their sub-state, obviously, and therefore act identically in both states. We continue to denote by state $j = f$ all agents who have a fever, which encompasses those in f_i and f_s.

Consider the number of susceptible agents next period if no vaccine is available by the end of the period ($\mathcal{V}_{t+1} = \mathcal{V}_t = 0$). This law of motion is given by

$$\begin{aligned} &M_{t+1}(s,a) \\ &= M_t(s,a)\Delta(a)\left[1 - \pi_f(n_t(s,a,\mathcal{V}_t) + \ell_t(s,a,\mathcal{V}_t), \Pi_t, \Pi_t^*, \mathcal{V}_t) + \pi_t^*(n_t(s,a,\mathcal{V}_t) + \ell_t(s,a,\mathcal{V}_t), \Pi_t^*)\xi_t(a)\right] \\ &+ M_t(f_s,a)\Delta(a)\left[1 - \pi_f(n_t(f,a,\mathcal{V}_t) + \ell_t(f,a,\mathcal{V}_t), \Pi_t, \Pi_t^*, \mathcal{V}_t) + \pi_t^*(n_t(f,a,\mathcal{V}_t) + \ell_t(f,a,\mathcal{V}_t), \Pi_t^*)\xi_t(a)\right] \end{aligned} \tag{15}$$

where the second line captures all situations in which susceptible individuals from last period remain alive and susceptible, as explained in connection to value function (4). The third line resembles the second except that it uses the time allocations of those in the fever state. It accounts for those who entered the period fever-susceptible and continue to remain susceptible during this period. The right hand side of (15) gives the map Ω_s for the susceptible agents when no vaccine is available by the end

of the period. If a vaccine is or becomes available ($\mathcal{V}_{t+1} = 1$), trivially all susceptible and fever-susceptible individuals continue as susceptible if they do not die of natural causes.

The following provides the analogous laws of motions Ω_j for agents in the other states $j = f_s, f_i, f, i, h, r$. For completeness it also accounts for Covid deaths and new infections. The aggregate mapping Ω is then the vector of the Ω_j for all states j and ages a depending on the vaccine availability in t and $t+1$.

In the absence of a vaccine ($\mathcal{V}_{t+1} = \mathcal{V}_t = 0$) the number of fever-susceptible agents who have a fever and are not tested but are truly Covid-negative and susceptible is given by

$$
\begin{aligned}
&M_{t+1}(f_s, a) \\
&= M_t(s,a)\Delta(a)(1-\xi_t(a))\pi_f(n_t(s,a,\mathcal{V}_t) + \ell_t(s,a,\mathcal{V}_t), \Pi_t, \Pi_t^*, \mathcal{V}_t)\frac{\Pi_t^*}{\Pi_t + \Pi_t^*} \\
&+ M_t(f_s, a)\Delta(a)(1-\xi_t(a))\pi_f(n_t(f,a,\mathcal{V}_t) + \ell_t(f,a,\mathcal{V}_t), \Pi_t, \Pi_t^*, \mathcal{V}_t)\frac{\Pi_t^*}{\Pi_t + \Pi_t^*}.
\end{aligned} \tag{16}
$$

It includes in the first line susceptible people from last period who got fever but were not tested, and are truly Covid-negative and susceptible. The second line again accounts for those in the fever-susceptible state, as they can again catch another fever while truly remaining susceptible. If a vaccine is available or arrives, transmissions are suppressed and we label individuals as susceptible as they are not confused even if they get a cold.

A similar logic applies to those in the fever-infected state in the absence of a vaccine arrival:

$$
\begin{aligned}
&M_{t+1}(f_i, a) \\
&= M_t(s,a)\Delta(a)(1-\xi_t(a))\pi_f(n_t(s,a,\mathcal{V}_t) + \ell_t(s,a,\mathcal{V}_t), \Pi_t, \Pi_t^*, \mathcal{V}_t)\frac{\Pi_t}{\Pi_t + \Pi_t^*} \\
&+ M_t(f_s, a)\Delta(a)(1-\xi_t(a))\pi_f(n_t(f,a,\mathcal{V}_t) + \ell_t(f,a,\mathcal{V}_t), \Pi_t, \Pi_t^*, \mathcal{V}_t)\frac{\Pi_t}{\Pi_t + \Pi_t^*}.
\end{aligned} \tag{17}
$$

The total number of individuals in the fever state is then

$$
M_{t+1}(f, a) = M_{t+1}(f_s, a) + M_{t+1}(f_i, a) \tag{18}
$$

To account for infected people in the absence of a vaccine arrival, one counts those who started last period susceptible or fever-susceptible and get infected and tested this period, and also those who started last period infected or fever-infected who neither

required hospitalization nor recovered:

$$\begin{aligned} M_{t+1}(i,a) = & M_t(s,a)\Delta(a)\xi_t(a)\pi(n_t(s,a,\mathcal{V}_t)+\ell_t(s,a,\mathcal{V}_t),\Pi_t,\mathcal{V}_t) \\ & + M_t(f_s,a)\Delta(a)\xi_t(a)\pi(n_t(f,a,\mathcal{V}_t)+\ell_t(f,a,\mathcal{V}_t),\Pi_t,\mathcal{V}_t) \\ & + \left[M_t(f_i,a)+M_t(i,a)\right]\Delta(a)(1-\phi(0,a))(1-\alpha(a)) \end{aligned} \tag{19}$$

If a vaccine was already present or arrives this period, the equation remains unchanged but the first two terms are set to zero.

People in hospitals comprise those who entered last period infected or fever-infected and do not recover but instead require hospitalization, as well as those individuals that were already hospitalized in the previous period who neither die nor recover:

$$\begin{aligned} M_{t+1}(h,a) = & \left[M_t(f_i,a)+M_t(i,a)\right]\Delta(a)(1-\phi(0,a))\alpha(a) \\ & + M_t(h,a)\Delta(a)(1-\delta(a))(1-\phi(1,a)) \end{aligned} \tag{20}$$

The total number of individuals hospitalized is then

$$M_t(h) = \sum_a M_t(h,a) \tag{21}$$

Recovered and therefore resistant individuals comprise those who were infected or fever-infected and recover, those hospitalized who do not die but recover, and resistant individuals from the previous period:

$$\begin{aligned} M_{t+1}(r,a) = & \left[M_t(f_i,a)+M_t(i,a)\right]\Delta(a)\phi(0,a) \\ & + M_t(h,a)\Delta(a)\phi(1,a)+\Delta(a)M_t(r,a) \end{aligned} \tag{22}$$

The right hand sides of equations (16) to (22) give the map Ω_j for states $j = f_s, f_i, f, i, h, r$ under vaccine availability $\mathcal{V}_t$.

For accounting purposes, the measure of deceased agents as a result of Covid-19 is given by new Covid deaths and those who died of it in previous periods:

$$M_{t+1}(deceased,a) = M_t(deceased,a) + (1-\phi(1,a))\delta(a)M_t(h,a)\Delta(a),$$

while the number of newly infected people is given by susceptible or fever-susceptible agents who get infected

$$\begin{aligned} N_{t+1}(i,a) \quad = \quad & M_t(s,a)\Delta(a)\pi(n_t(s,a,\mathcal{V}_t)+\ell_t(s,a,\mathcal{V}_t),\Pi_t,\mathcal{V}_t) \\ & + M_t(f_s,a)\Delta(a)\pi(n_t(f,a,\mathcal{V}_t)+\ell_t(f,a,\mathcal{V}_t),\Pi_t,\mathcal{V}_t). \end{aligned}$$

Aggregation for output in the economy in a given period is given by the time young

individuals spend at outside work or telework multiplied by the corresponding wage rate:

$$Q_t = \sum_j w[n_t(j, y, \mathcal{V}_t) + \iota(v_t(j, y, \mathcal{V}_t))v_t(j, y, \mathcal{V}_t)]M_t(j, y). \tag{23}$$

For many of the exercises we aggregate these weekly output measures to get overall GDP measures for longer time periods (e.g., year).

B Extra Tables

Table B1: Robustness with respect to ρ (no-lockdown equilibria)

		$\rho = -4.45$ (elast. of subst. = 0.183) (1)	$\rho = -1.72$ (elast. of subst. = 0.368) (2)	$\rho = -0.36$ (elast. of subst. = 0.735) (3)
Panel A. Realized statistics				
Weeks to peak infections	Young	9	10	10
	Old	10	10	11
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	7.13	6.87	6.61
	Old	9.18	14.47	19.67
Dead p/ 1,000, first year	Young	1.03	0.98	0.93
	Old	16.76	16.31	15.99
	All	4.39	4.26	4.15
Dead p/ 1,000 (by vaccine arrival)	Young	1.07	1.02	0.98
	Old	17.38	17.08	16.93
	All	4.56	4.46	4.39
Recovered , % (by vaccine arrival)	Young	46.81	44.84	42.95
	Old	18.18	17.89	17.77
	All	40.68	39.07	37.56
GDP 1 year, % change w.r.t. no-disease		-5.99	-5.77	-5.55
Panel B. Expected statistics				
Hrs @ home, diff. w.r.t. no-disease	Young	6.42	6.10	5.80
	Old	7.57	11.87	16.11
Dead p/ 1,000	Young	0.85	0.81	0.77
	Old	13.64	13.30	13.09
	All	3.59	3.48	3.41
GDP, % change w.r.t. no-disease		-5.93	-5.67	-5.43

Note: Vaccine arrival: 1.5 year.

Table B2: Benchmark, No Lockdown, No Isolation, No Testing

		Benchmark (1)	No lockdown Test and isolation (2)	No lockdown Test and no isolation (3)	No lockdown No testing (4)
Panel A. Realized statistics					
Weeks to peak infections	Young	44	10	10	10
	Old	45	10	11	11
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	17.02	6.87	7.70	7.42
	Old	11.56	14.47	15.46	15.32
Dead p/ 1,000, first year	Young	0.37	0.98	1.21	1.15
	Old	7.40	16.31	21.10	19.75
	All	1.87	4.26	5.47	5.13
Dead p/ 1,000 (by vaccine arrival)	Young	0.50	1.02	1.28	1.23
	Old	9.76	17.08	22.40	21.16
	All	2.48	4.46	5.80	5.49
Recovered , % (by vaccine arrival)	Young	23.17	44.84	56.38	53.88
	Old	10.63	17.89	23.52	22.23
	All	20.48	39.07	49.35	47.11
GDP 1 year, % change w.r.t. no-disease		-12.96	-5.77	-6.38	-6.16
Panel B. Expected statistics					
Hrs @ home, diff. w.r.t. no-disease	Young	14.03	6.10	6.43	6.33
	Old	10.28	11.87	12.48	12.62
Dead p/ 1,000	Young	0.40	0.81	1	0.97
	Old	7.57	13.30	17.07	16.29
	All	1.94	3.48	4.44	4.24
GDP, % change w.r.t. no-disease		-11.33	-5.67	-5.93	-5.84

Note: Vaccine arrival: 1.5 year.

Table B3: A different value for the value of a statistical life (VSL): no-lockdown equilibrium

		VSL = 9.3M (1)	VSL = 5M (2)
Panel A. Realized statistics			
Weeks to peak infections	Young	10	10
	Old	10	10
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	6.87	4
	Old	14.47	10.30
Dead p/ 1,000, first year	Young	0.98	1.13
	Old	16.31	19.68
	All	4.26	5.10
Dead p/ 1,000 (by vaccine arrival)	Young	1.02	1.15
	Old	17.08	20.01
	All	4.46	5.19
Recovered , % (by vaccine arrival)	Young	44.84	50.33
	Old	17.89	20.79
	All	39.07	44.01
GDP 1 year, % change w.r.t. no-disease		-5.77	-3.92
Panel B. Expected statistics			
Hrs @ home, diff. w.r.t. no-disease	Young	6.10	4
	Old	11.87	9.22
Dead p/ 1,000	Young	0.81	0.97
	Old	13.30	16.45
	All	3.48	4.28
GDP, % change w.r.t. no-disease		-5.67	-4.29

Table B4: A different value for the value of a statistical life (VSL): optimal policies

		VSL = 9.3M (1)	VSL = 5M (2)
Panel A. Realized statistics			
Avg tax., first year	Young	0.26	0.26
	Old	0.31	0.30
Hrs @ home - avg. first year, diff. w.r.t. no-lockdown	Young	15.76	18.25
	Old	-9.16	-5.11
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	22.63	22.25
	Old	5.30	5.19
% Deaths averted, first year (rel. to no-lockdown)	Young	98.93	98.57
	Old	98.19	97.66
	All	98.32	97.82
% Change in GDP rel. to no-lockdown, 1 year		-12.44	-13.74
Panel B. Expected statistics (3 year horizon)			
Hrs @ home - diff. w.r.t. no-lockdown	Young	16.89	18.32
	Old	-6.38	-3.95
Hrs @ home - diff. w.r.t. no-disease	Young	22.99	22.32
	Old	5.49	5.27
% Deaths averted, rel. to no-lockdown	Young	98.36	97.75
	Old	97.40	96.61
	All	97.58	96.82
% Change in GDP, rel. to no-lockdown		-11.39	-12.07
Panel C. Welfare			
CEV rel. to no-lockdown (%)	Young	0.35	0.05
	Old	16.97	11.12

Table B5: Unconstrained Planner

		Constrained optimal (1)	Unconstrained optimal (2)
Panel A. Realized statistics			
Avg. tax, first year, susceptible	Young	0.26	0.02
	Old	0.31	0.03
Avg. tax, first year, fever	Young	0.26	0.43
	Old	0.31	0.51
Avg. tax, first year, recovered	Young	0.26	0
	Old	0.31	0
Avg. tax, first year, infected	Young	0.38	0.93
	Old	0.38	0.91
Hrs @ home - avg. first year, diff. w.r.t. no-lockdown	Young	15.76	-3.26
	Old	-9.16	-13.47
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	22.63	3.61
	Old	5.3	1
% Deaths averted, first year (rel. to no-lockdown)	Young	98.93	99.16
	Old	98.19	98.88
	All	98.32	98.93
% Change in GDP rel. to no-lockdown, 1 year		-12.44	2.26
Panel B. Expected statistics (3 year horizon)			
Hrs @ home - diff. w.r.t. no-lockdown	Young	16.89	-2.24
	Old	-6.38	-10.58
Hrs @ home - diff. w.r.t. no-disease	Young	22.99	3.87
	Old	5.49	1.28
% Deaths averted, rel. to no-lockdown	Young	98.36	99
	Old	97.4	98.66
	All	97.58	98.72
% Change in GDP, rel. to no-lockdown		-11.39	1.16
Panel C. Welfare			
CEV rel. to no-lockdown	Young	0.35	0.87
	Old	16.97	17.38

Note: "Constrained optimal" refers to a planner who chooses a time-varying age-specific lockdown policy that applies equally to both "susceptible" and "fever" individuals, and lockdown for "infected" individuals remains constant at the calibrated level. "Unconstrained optimal" refers to an omnipotent planner who can set time-varying age-specific lockdown policy separately for "susceptible", "fever", and "infected" individuals. Both have the same objective function: to maximize weighted ex-ante welfare.

Table B6: Optimal Testing Capacity Build-up and Isolation

		Optimal lockdown Optimal test build-up (1)	Optimal lockdown Optimal test build-up Optimal isolation (2)	Optimal lockdown Optimal test build-up Optimal test usage Optimal isolation (3)
Panel A. Realized statistics				
Avg tax., first year	Young	0.20	0.06	0.06
	Old	0.26	0.07	0.07
Hrs @ home - avg. first year, diff. w.r.t. laissez-faire	Young	9.58	-2.53	-2.54
	Old	-10.75	-13.87	-13.90
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	17	4.89	4.88
	Old	4.57	1.45	1.41
% Deaths averted, 1 year (rel. to laissez-faire)	Young	99.06	98.99	98.98
	Old	98.50	98.51	98.50
	All	98.60	98.59	98.59
% Change in GDP rel. to laissez-faire, 1 year		-7.28	1.66	1.67
Panel B. Expected statistics				
Hrs @ home - diff. w.r.t. laissez-faire	Young	12.18	1.63	1.62
	Old	-7.69	-10.32	-10.30
Hrs @ home - diff. w.r.t. no-disease	Young	18.51	7.96	7.94
	Old	4.93	2.31	2.32
% Deaths averted, expectation 3 yrs (rel. to laissez-faire)	Young	98.51	98.85	98.85
	Old	97.82	98.29	98.28
	All	97.95	98.39	98.38
% Change in GDP, expectation 3 yrs (rel. to laissez-faire)		-7.09	0.75	0.75
Panel C. Welfare				
CEV rel. to laissez-faire (%)	Young	0.69	0.95	0.95
	Old	21.25	21.47	21.47
Panel D. Testing				
Maximum test capacity (% of population)		5.5	4.35	4.35
Week when max. test capacity is reached		19	12	12

Note: The laissez-faire comparison scenario refers to an equilibrium in which there is no government intervention: no tests and no lockdowns.

Table B7: Optimal Policy: No Telework, Younger Population

		Covid (1)	No telework, fewer old (2)
Panel A. Realized statistics			
Avg. tax, first year	Young	0.26	0.36
	Old	0.31	0.42
Hrs @ home - avg. first year, diff. w.r.t. no-lockdown	Young	15.76	11.51
	Old	-9.16	4.66
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	22.63	13.85
	Old	5.3	13.7
% Deaths averted, first year (rel. to no-lockdown)	Young	98.93	33.97
	Old	98.19	32.35
	All	98.32	33.04
% Change in GDP rel. to no-lockdown, 1 year		-12.44	-20.9
Panel B. Expected statistics (3 year horizon)			
Hrs @ home - diff. w.r.t. no-lockdown	Young	16.89	9.28
	Old	-6.38	2.81
Hrs @ home - diff. w.r.t. no-disease	Young	22.99	11.82
	Old	5.49	12.08
% Deaths averted, rel. to no-lockdown	Young	98.36	35.53
	Old	97.4	33.31
	All	97.58	34.26
% Change in GDP, rel. to no-lockdown		-11.39	-15.35
Panel C. Welfare			
CEV rel. to no-lockdown	Young	0.35	0.02
	Old	16.97	6.79

Note: Column 2 refers to an optimal policy when individuals cannot telework and the age composition of the population is that of the 1910s, during the Spanish flu pandemic.

Table B8: Synthetic diseases: No-lockdown Equilibrium

		Covid (1)	0.5 × Covid CFR (2)	0.5 × Covid R_0 (3)	0.5 × Covid R_0 & CFR (4)	1.5 × Covid CFR (5)	0.5 × Covid R_0 (6)	1.5 × Covid R_0 (7)	0.5 × Covid CFR 1.5 × Covid R_0 (8)	1.5 × Covid CFR 0.5 × Covid R_0 (9)
Panel A. Realized statistics										
Weeks to peak infections	Young	10	9	1	1	10	7	7	7	1
	Old	10	10	1	1	10	8	8	8	1
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	6.87	3.67	0.32	0.19	9.20	8.50	12.23	4.11	0.41
	Old	14.47	9.36	1.65	1.05	16.37	14.36	16.60	8.75	2.07
Dead p/ 1,000, first year	Young	0.98	0.57	0.04	0.03	1.24	1.41	1.88	0.77	0.06
	Old	16.31	10.01	0.85	0.52	20	27.68	35.73	15.72	1.09
	All	4.26	2.59	0.21	0.13	5.25	7.03	9.13	3.97	0.28
Dead p/ 1,000 (by vaccine arrival)	Young	1.02	0.58	0.04	0.03	1.34	1.45	2	0.78	0.06
	Old	17.08	10.14	0.85	0.52	21.77	28.51	38.09	15.83	1.10
	All	4.46	2.63	0.22	0.13	5.72	7.24	9.72	4	0.28
Recovered , % (by vaccine arrival)	Young	44.84	50.79	1.86	2.21	39.45	63.43	58.51	68.10	1.64
	Old	17.89	22.07	0.88	1.12	14.56	29.67	25.32	34.27	0.72
	All	39.07	44.65	1.65	1.97	34.13	56.20	51.41	60.86	1.44
GDP 1 year, % change w.r.t. no-disease		-5.77	-3.67	-1.43	-1.36	-7.35	-7.38	-10.13	-4.21	-1.49
Panel B. Expected statistics										
Hrs @ home, diff. w.r.t. no-disease	Young	6.10	3.77	0.42	0.24	7.73	8.48	11.25	4.84	0.56
	Old	11.87	8.67	2.14	1.33	13.67	12.37	14.59	8.78	2.71
Dead p/ 1,000	Young	0.81	0.49	0.04	0.02	1.04	1.21	1.61	0.69	0.05
	Old	13.30	8.43	0.75	0.45	16.59	23.36	30.24	13.78	0.97
	All	3.48	2.19	0.19	0.11	4.37	5.95	7.73	3.49	0.25
GDP, % change w.r.t. no-disease		-5.67	-4.10	-1.86	-1.76	-6.78	-7.83	-9.90	-5.12	-1.95

Note: The different columns change the R_0 and/or the case fatality rate (CFR) to a multiple of the values of Covid. Vaccine arrival = 1.5 year.

Table B9: Synthetic diseases: Optimal Policy

		Covid (1)	0.5 × Covid CFR (2)	0.5 × Covid R_0 (3)	0.5 × Covid R_0 & CFR (4)	1.5 × Covid CFR (5)	1.5 × Covid R_0 (6)	1.5 × Covid R_0 & CFR (7)	0.5 × Covid CFR 1.5 × Covid R_0 (8)	1.5 × Covid CFR 0.5 × Covid R_0 (9)
Panel A. Realized statistics										
Avg tax., first year	Young	0.26	0.25	0.03	0.02	0.27	0.37	0.38	0.37	0.03
	Old	0.31	0.29	0.03	0.02	0.32	0.47	0.48	0.48	0.03
Hrs @ home - avg. first year, diff. w.r.t. no-lockdown	Young	15.76	18.36	2.17	1.89	13.88	23.50	20.54	27.73	2.46
	Old	-9.16	-4.38	-0.93	-0.46	-10.78	-5.96	-7.95	-0.12	-1.22
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	22.63	22.03	2.49	2.08	23.08	32	32.77	31.85	2.88
	Old	5.30	4.98	0.72	0.59	5.59	8.40	8.64	8.62	0.85
% Deaths averted, first year (rel. to no-lockdown)	Young	98.93	98.54	78.14	74.99	98.93	99.20	99.23	98.84	78.85
	Old	98.19	97.60	74.75	72.34	98.13	98.71	98.74	98.20	74.89
	All	98.32	97.77	75.28	72.74	98.28	98.79	98.82	98.30	75.51
% Change in GDP rel. to no-lockdown, 1 year		-12.44	-13.77	-1.40	-1.18	-11.36	-20.67	-19.16	-23.06	-1.61
Panel B. Expected statistics										
Hrs @ home - diff. w.r.t. no-lockdown	Young	16.89	18.30	3.77	3.15	15.78	24.38	22.08	27.42	4.16
	Old	-6.38	-3.62	-0.91	-0.36	-7.82	-3.54	-5.47	0.04	-1.26
Hrs @ home - diff. w.r.t. no-disease	Young	22.99	22.07	4.19	3.39	23.52	32.86	33.32	32.26	4.72
	Old	5.49	5.05	1.23	0.97	5.85	8.83	9.12	8.81	1.45
% Deaths averted, rel. to no-lockdown	Young	98.36	97.70	76.68	73.48	98.43	98.70	98.84	97.96	77.35
	Old	97.40	96.55	72.93	70.55	97.45	98.11	98.25	97.21	72.96
	All	97.58	96.75	73.51	70.99	97.63	98.20	98.34	97.33	73.66
% Change in GDP, rel. to no-lockdown		-11.39	-12.04	-1.04	-0.85	-10.79	-20.92	-19.91	-22.16	-1.17
Panel C. Welfare										
CEV rel. to no-lockdown (%)	Young	0.35	0.02	0.01	0	0.59	-0.04	0.41	-0.59	0.01
	Old	16.97	10.10	0.57	0.33	21.85	31.11	43.18	16.29	0.75

Note: The different columns change the R_0 and/or the case fatality rate (CFR) to a multiple of the values of Covid. Vaccine arrival = 1.5 year.

Table B10: Varying the ratio of the CFR by age while keeping the time-outside-weighted average CFR constant, using the time outside in a non-Covid world: no lockdown equilibria (robustness exercise for Table 15)

		CFR old/yng 1/39 (1)	CFR old/yng 1/2 (2)	CFR old/yng 1/1 (3)	CFR old/yng 2/1 (4)	CFR old/yng 39/1 (5)
Panel A. Realized statistics						
Weeks to peak infections	Young	11	11	11	11	10
	Old	10	10	10	10	10
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	18.71	18.19	17.71	16.88	6.87
	Old	0.10	1.67	3.04	5.19	14.47
Dead p/ 1,000, first year	Young	2.10	2.06	2.03	1.96	0.98
	Old	0.04	0.80	1.51	2.70	16.31
	All	1.66	1.79	1.92	2.12	4.26
Dead p/ 1,000 (by vaccine arrival)	Young	2.73	2.67	2.61	2.50	1.02
	Old	0.05	0.99	1.86	3.33	17.08
	All	2.16	2.31	2.45	2.67	4.46
Recovered , % (by vaccine arrival)	Young	22.16	22.83	23.52	24.85	44.84
	Old	17.81	17.42	17.11	16.72	17.89
	All	21.23	21.67	22.15	23.11	39.07
GDP 1 year, % change w.r.t. no-disease		-14.53	-14.10	-13.71	-13.05	-5.77
Panel B. Expected statistics						
Hrs @ home, diff. w.r.t. no-disease	Young	15.68	15.23	14.81	14.08	6.10
	Old	0.08	1.38	2.53	4.35	11.87
Dead p/ 1,000	Young	2.09	2.04	1.99	1.90	0.81
	Old	0.04	0.74	1.39	2.49	13.30
	All	1.65	1.76	1.86	2.03	3.48
GDP, % change w.r.t. no-disease		-12.62	-12.26	-11.93	-11.37	-5.67

Note: Expected vaccine arrival = 1.5 year.

Table B11: Varying the ratio of the CFR by age while keeping the time-outside-weighted average CFR constant, using the time outside in a non-Covid world: optimal policy (robustness exercise for Table 16)

		CFR old/yng 1/39 (1)	CFR old/yng 1/2 (2)	CFR old/yng 1/1 (3)	CFR old/yng 2/1 (4)	CFR old/yng 39/1 (5)
Panel A. Realized statistics						
Avg tax., first year	Young	0.28	0.27	0.27	0.27	0.26
	Old	0.30	0.29	0.29	0.30	0.31
Hrs @ home - avg. first year, diff. w.r.t. no-lockdown	Young	5.54	5.85	6.37	7.04	15.76
	Old	4.63	3.07	1.74	-0.30	-9.16
Hrs @ home, avg. first year, diff. w.r.t. no-disease	Young	24.25	24.04	24.08	23.91	22.63
	Old	4.73	4.74	4.78	4.89	5.30
% Deaths averted, first year (rel. to no-lockdown)	Young	96.73	96.85	97.01	97.24	98.93
	Old	96.98	96.96	96.96	96.99	98.19
	All	96.73	96.86	97	97.17	98.32
% Change in GDP rel. to no-lockdown, 1 year		-5.10	-5.40	-5.82	-6.37	-12.44
Panel B. Expected statistics						
Hrs @ home - diff. w.r.t. no-lockdown	Young	8.78	9.06	9.49	10.07	16.89
	Old	4.66	3.40	2.29	0.61	-6.38
Hrs @ home - diff. w.r.t. no-disease	Young	24.46	24.29	24.30	24.14	22.99
	Old	4.74	4.78	4.82	4.96	5.49
% Deaths averted, rel. to no-lockdown	Young	95.93	96.09	96.21	96.46	98.36
	Old	96.28	96.27	96.25	96.28	97.40
	All	95.93	96.11	96.22	96.41	97.58
% Change in GDP, rel. to no-lockdown		-6.35	-6.56	-6.87	-7.27	-11.39
Panel C. Welfare						
CEV rel. to no-lockdown (%)	Young	1.81	1.74	1.68	1.58	0.35
	Old	-0.11	0.57	1.23	2.40	16.97

Note: Expected vaccine arrival = 1.5 years.

C The Possibility of Reinfection

This section describes the changes to the baseline model and main results when we allow for the possibility that an individual can catch Covid multiple times.

C.1 Theory with Reinfection

The main conceptual change to the model is that, after an individual recovers from Covid, there is a chance that they will become truly recovered and immune to future infections while there is also a chance that they will end up back in the susceptible pool and may catch the disease again. Denote by ζ the probability that an infected agent will become susceptible again.[51]

We will first describe the required changes in the value functions. The main idea is that, whenever the continuation value was given by that of a recovered agent in the baseline formulation, now there needs to be an expected value to account for the possibility that this person becomes susceptible again.

Start with currently infected i agents. Their value function should be changed to:

$$\begin{aligned} V_t(i,a,\mathcal{V}) = \max_{c,x,n,v,\ell,d} & u(c,g(x,\ell),d,v) + \\ & \beta(a)\phi(0,a)\left[\zeta W_{t+1}(s,a,\mathcal{V} + (1-\zeta)W_{t+1}(r,a,\mathcal{V})\right] + \\ & \beta(a)(1-\phi(0,a))\alpha(a)W_{t+1}(h,a,\mathcal{V}) + \\ & \beta(a)(1-\phi(0,a))(1-\alpha(a))W_{t+1}(i,a,\mathcal{V}) \\ & \text{s.t. resource constraints.} \end{aligned} \tag{24}$$

Turn to currently hospitalized h agents. Their value function should now read:

$$\begin{aligned} V_t(h,a,\mathcal{V}) = \beta(a)\{ & \phi(1,a)\left[\zeta W_{t+1}(s,a,\mathcal{V}) + (1-\zeta)W_{t+1}(r,a,\mathcal{V})\right] + \\ & (1-\phi(1,a))(1-\delta(a))W_{t+1}(h,a,\mathcal{V})\} \\ & \text{s.t. resource constraints.} \end{aligned} \tag{25}$$

We also need to change the laws of motions for the distributions of agents. To do this, we must divide the measure of those that used to become recovered into those that are now truly recovered versus those that are susceptible again. Such a division should be weighted by the probability ζ. So, only two laws of motions need to be

[51] For simplicity, we assume that the individual enters the same susceptible state as a person who was never infected before. With slightly more notation one could add a second susceptible state that has a similar value function, but with possibly different probabilities of reinfection and hospitalization upon infection. Additionally, one could allow only partial immunity by introducing a probability that those in the "r" state re-enter the susceptible state with some probability. Our formulation abstracted from this to preserve mathematical and especially computational tractability.

changed: those for recovered next period, $M_{t+1}(r,a)$, and those for susceptible next period, $M_{t+1}(s,a)$.

The law of motion for the recovered r should be changed to:

$$\begin{aligned} M_{t+1}(r,a) = (1-\zeta)\left[M_t(f_i,a) + M_t(i,a)\right]\Delta(a)\phi(0,a) \\ + (1-\zeta)M_t(h,a)\Delta(a)\phi(1,a) + \Delta(a)M_t(r,a) \end{aligned} \tag{26}$$

And the law of motion for the susceptible s should now read:

$$\begin{aligned} & M_{t+1}(s,a) \\ & = M_t(s,a)\Delta(a)\left[1-\pi_f(n_t(s,a,\mathcal{V}_t)+\ell_t(s,a,\mathcal{V}_t),\Pi_t,\Pi_t^*,\mathcal{V}_t)+\pi_t^*(n_t(s,a,\mathcal{V}_t)+\ell_t(s,a,\mathcal{V}_t),\Pi_t^*)\xi_t(a)\right] \\ & + M_t(f_s,a)\Delta(a)\left[1-\pi_f(n_t(f,a,\mathcal{V}_t)+\ell_t(f,a,\mathcal{V}_t),\Pi_t,\Pi_t^*,\mathcal{V}_t)+\pi_t^*(n_t(f,a,\mathcal{V}_t)+\ell_t(f,a,\mathcal{V}_t),\Pi_t^*)\xi_t(a)\right] \\ & + \zeta\left[M_t(f_i,a)+M_t(i,a)\right]\Delta(a)\phi(0,a) + \zeta M_t(h,a)\Delta(a)\phi(1,a) \end{aligned} \tag{27}$$

C.2 Benchmark Economy with Reinfections

In the following, we will assess the impact of a positive reinfection probability ($\zeta > 0$, see model in Section C.1) on the benchmark economy, all else equal.

The main issue is how to calibrate the level of ζ. This variable represents the probability that an agent who recovers from Covid is again susceptible and therefore can catch the disease again, as opposed to becoming recovered and resistant without probability of reinfection. Stein et al. (2023) provide a meta-study on reinfection, covering 65 studies from 19 countries that examine this topic with data from the first two years of the pandemic. The paper's findings are summarized as "Mean pooled effectiveness was greater than 78% against severe disease (hospitalisation and death) for all variants, including omicron BA.1. Protection from re-infection from ancestral, alpha, and delta variants declined over time but remained at 78.6% (49.8% - 93.6%) at 40 weeks" (p. 833). That is, roughly 22% of individuals are shielded against severe disease, and for most varieties this is also the number of individuals protected against reinfection.

We therefore set $\zeta = 0.22$ as our preferred specification. Stein et al. (2023) cite protection against reinfection after 40 weeks, but the researchers clarify that they "found that protection against re-infection was high, with a mean pooled estimate greater than 82% for ancestral, alpha, beta, and delta variants" (p. 836). So their 40 weeks summary estimate also provides an averaged benchmark. So setting ζ such that roughly 20% of individuals re-enter the susceptible pool also seems reasonable to capture reinfection.

We also provide results for other values of ζ, exploring $\zeta \in \{0.1, 0.5, 0.9\}$ for robustness. The lower value of $\zeta = 0.1$ can be justified by looking mainly at protection against severe disease or hospitalization. The previous quote was a lower bound, us-

ing the few studies that investigate severe disease long after initial infection. In fact, Stein et al. (2023) note: "protection against severe disease remained high for all variants, with 90.2% [..] for ancestral, alpha, and delta variants, and 88.9% [..] for omicron BA.1 at 40 weeks." This would justify that just 10% of infected individuals return to the susceptible pool.

The value of $\zeta = 0.5$ is a better representation of reinfections under Omicron: "Pooled effectiveness against re-infection by the omicron BA.1 variant was 45.3%" (p.836). We view $\zeta = 0.9$ as a very conservative value. We picked it because "Protection against re-infection by the omicron BA.1 variant declined more rapidly and was estimated at 36.1% [..] at 40 weeks", and inspection of the most long-run estimates according to the graphs in Stein et al. (2023) is roughly 25% with a lower error bound at roughly 10% (Figure 3, p. 838). This is the most conservative number available. And it is especially conservative because even though reinfection probabilities drop over time, protection against severe disease remains above 75% even for omicron over this time.

Table C12 compares the baseline results for the economy with reinfection risk to our benchmark without reinfections. Table C13 compares the optimal policies for economies with different reinfection risks. In both tables, column (1) refers to the economy without reinfection risk, column (3) gives results for what we regard a realistic Covid reinfection rate (22%), while Column (2) provides the results for a 10% reinfection rate, and Columns (4)-(5) show what would happen under higher reinfection rates. In interpreting these results, one should also take into account that a prior Covid-19 infection does lead to partial immunity in that it reduces the severity of the disease and thereby substantially lowers the mortality rate. Analyzing this subtlety would require a model with even more health states and is left for future research.

Table C12: Benchmark results considering different reinfection rate scenarios

		Benchmark (1)	Reinfection (10%) (2)	Reinfection (22%) (3)	Reinfection (50%) (4)	Reinfection (90%) (5)
Panel A. Realized statistics						
Weeks to peak infections	Young	44	44	44	96	96
	Old	45	45	45	45	96
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	17.02	17.15	17.32	17.79	18.68
	Old	11.56	11.75	11.98	12.51	13.21
Dead p/ 1,000, first year	Young	0.37	0.38	0.40	0.44	0.48
	Old	7.40	7.63	7.91	8.54	9.20
	All	1.87	1.93	2	2.17	2.35
Dead p/ 1,000 (by vaccine arrival)	Young	0.50	0.53	0.57	0.68	0.88
	Old	9.76	10.23	10.86	12.57	15.34
	All	2.48	2.61	2.77	3.23	3.98
Recovered , % (by vaccine arrival)	Young	23.17	22.17	20.75	16.15	4.25
	Old	10.63	10.05	9.27	6.92	1.71
	All	20.48	19.57	18.30	14.17	3.71
GDP 1 year, % change w.r.t. no-disease		-12.96	-13.07	-13.20	-13.57	-14.29
Panel B. Expected statistics						
Hrs @ home, diff. w.r.t. no-disease	Young	14.03	14.14	14.30	14.78	16.12
	Old	10.28	10.49	10.74	11.40	12.77
Dead p/ 1,000	Young	0.40	0.43	0.46	0.56	0.83
	Old	7.57	7.94	8.43	9.85	13.22
	All	1.94	2.04	2.17	2.55	3.48
GDP, % change w.r.t. no-disease		-11.33	-11.41	-11.52	-11.88	-12.84

Note: This table compares the baseline results for the economy with reinfection risk to our benchmark without reinfections. Column (1) refers to the economy without reinfection risk, column (3) gives results for what we regard a re- alistic Covid reinfection rate, while columns (3)-(5) show what would happen under much higher reinfection rates.

Table C13: Optimal policy considering different reinfection rate scenarios

		Optimal lockdown (1)	Reinfection (10%) (2)	Reinfection (22%) (3)	Reinfection (50%) (4)	Reinfection (90%) (5)
Panel A. Realized statistics						
Avg tax., first year	Young	0.26	0.26	0.26	0.26	0.26
	Old	0.31	0.31	0.31	0.31	0.31
Hrs @ home - avg. first year, diff. w.r.t. no-lockdown	Young	15.76	15.20	14.40	11.86	5.77
	Old	-9.16	-9.71	-10.34	-11.75	-13.61
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	22.63	22.66	22.68	22.72	22.79
	Old	5.30	5.28	5.28	5.30	5.30
% Deaths averted, first year (rel. to no-lockdown)	Young	98.93	99.01	99.10	99.29	99.45
	Old	98.19	98.30	98.44	98.72	98.99
	All	98.32	98.43	98.56	98.83	99.08
% Change in GDP rel. to no-lockdown, 1 year		-12.44	-12.09	-11.58	-9.88	-5.24
Panel B. Expected statistics (3 year horizon)						
Hrs @ home - diff. w.r.t. no-lockdown	Young	16.89	16.56	16.08	14.50	9.78
	Old	-6.38	-6.76	-7.25	-8.78	-11.50
Hrs @ home - diff. w.r.t. no-disease	Young	22.99	23.02	23.05	23.11	23.20
	Old	5.49	5.47	5.48	5.50	5.51
% Deaths averted, rel. to no-lockdown	Young	98.36	98.47	98.61	98.92	99.34
	Old	97.40	97.56	97.76	98.23	98.82
	All	97.58	97.73	97.92	98.36	98.92
% Change in GDP, rel. to no-lockdown		-11.39	-11.19	-10.89	-9.79	-5.52
Panel C. Welfare						
CEV rel. to no-lockdown (%)	Young	0.35	0.42	0.51	0.81	1.70
	Old	16.97	18.15	19.83	25.58	40.99

Note: This table compares the optimal policy for the economy with reinfection risk to optimal policy in our baseline without reinfections. Column (1) refers to the economy without reinfection risk, column (3) gives results for what we regard a re- alistic Covid reinfection rate, while columns (3)-(5) show what would happen under much higher reinfection rates.

D Alternative Calibrations

D.1 The Spanish Flu

Table D14 lists the model parameters that are different for the Spanish flu calibration. Some parameters relate to how different the world was in the 1910s and others directly relate to the different disease. The remaining parameters coincide with those for Covid reported in Tables 1 and 2.

The fraction of the old population comes from the US Census Bureau's estimates for the resident population in July 1917. The population was younger then and only 7.7% of the adult population was 65 years old or older. The average person between 20 and 64 years old in the late 1910s was 37 years old. According to the United States Life Tables for the period, the remaining life expectancy for this person was 32.4 years. Hence, we set $\Delta(y) = 1 - 1/(32.4 \times 52) = 0.9994$. For those above 65 years old, the average person was 72 and expected to live an extra 8.7 years. Thus, $\Delta(o) = 1 - 1/(8.7 \times 52) = 0.9978$.

According to the Groningen Growth and Development Centre's Maddison Project Database 2020 (Groningen Growth and Development Centre 2020), the United States per capita GDP in 1917 was 19% of the value in 2018. We thus decrease the value of the per-unit-of-time wage w rate to match this target. We keep the same replacement rate for the old's income (60%) and adjust this group's income accordingly. We also assume that there is no telework in the 1910s and thus set the telework parameters (ι_0 and ι_1) to zero.

Turn attention now to the specifics of the Spanish flu virus. Taubenberger and Morens (2006) report age-specific case fatality rates (CFR) for the Spanish flu pandemic. Aggregating across age groups, the CFR for the young (20-64 years old) is 1.9% and the CFR for the old (65 or above) is 4.01%. We do not have information, however, on the decomposed transition rates from infection to ICU and from ICU to death. Hence, we assume that, if an infected individual goes to the ICU with serious symptoms from the Spanish flu, they die with certainty. Since there are no choices during ICU treatment, this is not a very strong assumption. So, there is no possibility of recovery from ICU, $\phi(1, y) = \phi(1, o) = 0$, and an ICU patient dies with certainty, $\delta(y) = \delta(o) = 1$. This means that the continuation value of going to the hospital is 0. We only need to determine $\phi(0, a)$ now. These are chosen to match the CFRs. With $\alpha = 1$, an infected individual recovers after one period. So, $\phi(0, a) = 1 - CFR(a)$. Hence, $\phi(0, y) = 0.981$ and $\phi(0, o) = 0.960$.

Mills, Robins, and Lipsitch (2004) estimate the basic reproduction number R_0 for the Spanish flu using data for 45 cities in the United States. The median value of their estimates is less than three. Following their estimates, we target an $R_0 = 2.7$, slightly

Table D14: Calibration – Parameters that are different under Spanish Flu

Parameter	Value	Interpretation
	0.077	Fraction of old in population
$\Delta(y)$	0.9994	Weekly survival (natural causes), young
$\Delta(o)$	0.9978	Weekly survival (natural causes), old
w	0.167	Wage per unit of time
$\overline{w}$	0.041	Retirement income
ι_0	0.0	Parameter related to telework productivity
ι_1	0.0	Parameter related to telework productivity
$\phi(0, y)$	0.981	Prob of recovering from mild Covid-19, young
$\phi(0, o)$	0.960	Prob of recovering from mild Covid-19, old
$\phi(1, y)$	0.0	Prob of recovering from hospitalization, young
$\phi(1, o)$	0.0	Prob of recovering from hospitalization, old
$\delta(y)$	1.0	Weekly death rate (among hospitalized), young
$\delta(o)$	1.0	Weekly death rate (among hospitalized), old
Π_0	6.448	Infectiousness of Spanish flu
χ_t	$1/(10\times52)$	Prob of vaccine arrival (average = 10 years)
η_0	0.0	Stringency index function
η_1	0.0	Stringency index function
ξ_t	0.0	Probability of testing (no tests)

lower than that of Covid. In the model, this data target identifies $\Pi_0 = 6.448$.

A vaccine for influenza did not appear nearly as quickly as the one for Covid. The first effective vaccine only appeared around 20 years after the Spanish flu pandemic WHO (2022). However, other vaccines were being used. So, the population probably expected a quicker turnaround. We thus assume an expected arrival for a flu vaccine in 10 years and set the weekly probability of a vaccine arrival accordingly. We set the latest possible date for the vaccine arrival at $T^* = 546$, or 10.5 years after the onset of the pandemic.

We do not have detailed information about the strictness of lockdowns that were adopted in response to the Spanish flu pandemic in the 1910s. However, we will not match the entire time series as we did for Covid. We will solve a no-policy benchmark and the optimal policy in response to the disease. Hence, we set the parameters of the benchmark lockdown stringency (η_0 and η_1) to zero. As there was no equivalent to the modern (PCR and antigent) tests, we assume there was no testing and isolation in the 1910s. So, the probability of being tested is $\xi_t = 0$.

D.2 Testing Build-up

As discussed in Section 6.1, we must discipline two parameters: κ and p. The parameter κ controls the quadratic investment cost, $f(k_t) = \kappa k_t^2$, and p is the unit cost of a test.

We choose κ to match the expenditures on testing capacity in the US in 2020. The *Oxford Coronavirus Government Response Tracker* reports data on daily tests performed in the US (Oxford COVID-19 Government Response Tracker 2020). We aggregate this to weekly frequency (see Figure 6b). At the end of 2020, 3.8% of the US population could be tested in one week. So, $\overline{K} = 0.038$. The Department of Health and Human Services (HHS) spent \$25 billion for necessary expenses to research, develop, validate, manufacture, purchase, administer, and expand capacity for COVID-19 tests (U.S. Department of Health and Human Services 2020). We need to turn this value into model units. Let ϵ denote the "exchange rate" between actual dollars and "model dollars." This rate can be found as the ratio of model output (Q_m) by data output (Q_d). Weekly output in the model consists of the young's labor supply, since the wage is normalized to 1. Weekly output in the data is approximately \$400 billion. Let $\overline{M}$ denote the total amount spent in testing capacity in the model. So, $\overline{M} = \$25 \text{ billion} \times \epsilon = 0.217$.

According to Figure 6b, the ramp-up of tests in 2020 was approximately linear. With this assumption, we have:

$$\overline{M} = \sum_{t=1}^{T} \kappa k_t^2 = \sum_{t=1}^{T} \kappa \bar{k}^2 = T\kappa \left(\frac{\overline{K}}{T}\right)^2 = \frac{\kappa \overline{K}^2}{T},$$

where $T = 40$ is the number of weeks between the start of the pandemic in early March and the end of 2020. Hence, $\kappa = (T\overline{M})/\overline{K}^2 = 590.2$.

There is no single price for a test in the data. The Health System Tracker provides a distribution of prices (Health System Tracker 2020). The 25th percentile is around \$50. We use this somewhat lower bound to discipline p, as the HHS expenditures are quite comprehensive and a portion of the HHS resources may have been used to partially offset the unit cost of each test. Using the "exchange rate" defined above, $p = \$50 \times \epsilon$.

E Computational Appendix

It is instructive to discuss the first-order conditions that characterize the agents' choices. Let's examine a simplified version of our model, excluding age heterogeneity, uncertainty, and other components. The value function for a susceptible agent is as follows:

$$V_t(s) = \max_{c,x,n,\ell,v,d} \ln(c) + \gamma \ln\left([\theta x + (1-\theta)\ell]^{1/\rho}\right) + \lambda \ln(d) + b$$
$$+ \beta\{(n+\ell)\Pi_t V_{t+1}(i) + [1-(n+\ell)\Pi_t]V_{t+1}(s)\}$$

subject to

$$c + x = w[n + \iota(v)v],$$
$$\frac{n+\ell}{1-\tau_t} + v + d = 1 + T_t.$$

To solve the maximization problem above, rewrite it as

$$\max_{x,n,\ell,v} \ln(w[n+\iota(v)v] - x) + \gamma \ln\left([\theta x + (1-\theta)\ell]^{1/\rho}\right)$$
$$+\lambda \ln\left(1 + T_t - \frac{n+\ell}{1-\tau_t} - v\right) + \beta(n+\ell)\Pi_t[V_{t+1}(i) - V_{t+1}(s)].$$

The first-order conditions are

$$[x]: -\frac{1}{w[n+\iota(v)v]-x} + \frac{\gamma\theta x^{\rho-1}}{\theta x^\rho + (1-\theta)\ell^\rho} = 0,$$
$$[n]: \frac{w}{w[n+\iota(v)v]-x} - \frac{\lambda}{1+T_t(s)-(n+\ell)/(1-\tau_t)-v}\frac{1}{1-\tau_t(s)} + \beta\Pi_t[V_{t+1}(i) - V_{t+1}(s)] = 0,$$
$$[\ell]: \frac{\gamma(1-\theta)\ell^{\rho-1}}{\theta x^\rho + (1-\theta)\ell^\rho} - \frac{\lambda}{1+T_t(s)-(n+\ell)/(1-\tau_t)-v}\frac{1}{1-\tau_t(s)} + \beta\Pi_t[V_{t+1}(i) - V_{t+1}(s)] = 0,$$
$$[v]: \frac{w[\iota'(v)v + \iota(v)]}{w[n+\iota(v)v]-x} - \frac{\lambda}{1+T_t(s)-(n+\ell)/(1-\tau_t)-v} = 0.$$

Substitute the lump-sum tax rebate $T_t = \tau_t(n+\ell)/(1-\tau_t)$ in the equations above to obtain the following system of equations in x, n, ℓ, and v:

$$[x]: -\frac{1}{w[n+\iota(v)v]-x} + \frac{\gamma\theta x^{\rho-1}}{\theta x^\rho + (1-\theta)\ell^\rho} = 0, \tag{28}$$
$$[n]: \frac{w}{w[n+\iota(v)v]-x} - \frac{\lambda}{1-n-\ell-v}\frac{1}{1-\tau_t} + \beta\Pi_t[V_{t+1}(i) - V_{t+1}(s)] = 0, \tag{29}$$
$$[\ell]: \frac{\gamma(1-\theta)\ell^{\rho-1}}{\theta x^\rho + (1-\theta)\ell^\rho} - \frac{\lambda}{1-n-\ell-v}\frac{1}{1-\tau_t} + \beta\Pi_t[V_{t+1}(i) - V_{t+1}(s)] = 0, \tag{30}$$
$$[v]: \frac{w[\iota'(v)v + \iota(v)]}{w[n+\iota(v)v]-x} - \frac{\lambda}{1-n-\ell-v} = 0. \tag{31}$$

The equations above describe the trade-offs faced by agents during the pandemic. For example, equation (29) demonstrates that an agent equalizes marginal benefits and costs associated with working outside. The first term represents the marginal utility from consumption acquired through one additional hour of work. The other two terms pertain to the marginal cost of n: the sacrifice of leisure at home and an increased probability of contracting Covid. Higher taxation (i.e., a more stringent lockdown) elevates the marginal cost of n by intensifying the sacrifice of home leisure. Additionally, a more contagious environment (a higher Π_t) or a more severe illness (a more negative $V_{t+1}(i) - V_{t+1}(s)$) amplifies the marginal cost of n.

We use the following computational algorithm to find a general equilibrium of the main model:

1. Guess the path of general equilibrium variables $\{\Pi_t, \xi_t\}_{t=0}^{T^*}$.

2. Starting from the last time period T^* and going backward in time, solve the agents' problems. For a given time period t, the agents' optimal choices can be obtained from solving systems of equations such as (28)-(31), with the general equilibrium variables and future value functions treated as given.

3. Starting from the first time period and going forward in time, obtain new distribution for all types using their laws of motion.

4. Using the optimal policies and distribution types obtained in steps 2 and 3, compute new general equilibrium variables $\{\Pi'_t, \xi'_t\}_{t=0}^{T^*}$ using (10)-(12).

5. If $\{\Pi_t, \xi_t\}_{t=0}^{T^*}$ is close enough to $\{\Pi'_t, \xi'_t\}_{t=0}^{T^*}$, the algorithm has converged. Otherwise, go back to step 2 using $\{\Pi'_t, \xi'_t\}_{t=0}^{T^*}$ as a new guess of the general equilibrium variables.

F A model with two-period uncertainty

Here we extend the base model to allow for two periods of uncertainty. If a susceptible person experiences a fever, they do not get any information after this period unless they get tested. Only at the end of the next period does this person get to know they started that period. So, in the second period, the uncertainty about the infection is resolved similar to the fever case in the base model by revealing the beginning-of-period infection status (which means that the person can continue to be worried about the health status in the subsequent period if they develop another fever). In the first period, no information is revealed unless through testing. Testing is assumed to detect the right status, including "recovered."

Unless specifically newly introduced, notation follows the main body of the paper.

F.1 Value Functions in the model with two periods of uncertainty

F.1.1 Susceptible

$$\begin{aligned} V_t(s,a,\mathcal{V}) \;=\; & \max_{c,x,n,v,\ell,d} u(c,g(x,\ell),d,v) + \\ & +\beta(a)[1-\pi_f(n+\ell,\Pi_t,\Pi_t^*,\mathcal{V})+\pi^*(n+\ell,\Pi_t^*)\xi_t(a)]W_{t+1}(s,a,\mathcal{V}|s) \\ & +\beta(a)\pi(n+\ell,\Pi_t,\mathcal{V})\xi_t(a)]W_{t+1}(i,a,\mathcal{V}|s) + \\ & \beta(a)(1-\xi_t(a))\pi_f(n+\ell,\Pi_t,\Pi_t^*,\mathcal{V})W_{t+1}(f_1,a,\mathcal{V}|s) \end{aligned}$$

with constraints (2) and (3) from the main paper, and the convention from the main body that

$$W_{t+1}(j,a,\mathcal{V}|s) = \chi(\mathcal{V})V_{t+1}(j,a,1) + (1-\chi(\mathcal{V}))V_{t+1}(s,a,0)$$

The expressions are similar to those in the main text. Note that they ensure that individuals arrive in the "confused" fever state only if a vaccine is not available by the end of the period.

F.1.2 Infected

$$\begin{aligned} V_t(i,a,\mathcal{V}) \;=\; & \max_{c,x,n,v,\ell,d} u(c,g(x,\ell),d,v) \\ & +\beta(a)\phi(0,a)W_{t+1}(r,a,\mathcal{V}) \\ & +\beta(a)(1-\phi(h,a))\alpha(a)W_{t+1}(h,a,\mathcal{V}) \\ & +\beta(a)(1-\phi(h,a))(1-\alpha(a))W_{t+1}(i,a,\mathcal{V}), \end{aligned}$$

again with constraints (2) and (3) from the main text. This is also similar to the main

text, and the convention from the main body that

$$W_{t+1}(j,a,\mathcal{V}) = \chi(\mathcal{V})V_{t+1}(j,a,1) + (1-\chi(\mathcal{V}))V_{t+1}(j,a,0).$$

F.1.3 f_1 fever people

The state "f_1" now replaces our previous fever state "f". The index "1" indicates that this is the first period of uncertainty over the health status.

In the following, "f_2" stands for having fever in the second period of uncertainty. This can be if the person is healthy (either susceptible or recovered after infection last period) but develops another cold. It can also be because the person caught Covid in one of the last two periods.

In the following, "nf_2" stands for *not* having fever in the second period of uncertainty (and also not being in hospital and not having been tested). So these include individuals face uncertainty about being susceptible or recovered.

The value function for a person in the first period of uncertainty (i.e., the first period with a fever) is

$$\begin{aligned}
V_t(f_1,a,\mathcal{V}) \quad = \quad & \max_{c,x,n,v,\ell,d} u(c,g(x,\ell),d,v) + \\
& + \frac{\Pi^*_{t-1}\beta(a)}{\Pi_{t-1}+\Pi^*_{t-1}}(1-\chi(\mathcal{V}_t)) \left[\begin{array}{c} (1-\xi_t(a))[1-\pi_f(n+\ell,\Pi_t,\Pi^*_t,0)]V_{t+1}(nf_2,a,0;n+\ell) \\ +(1-\xi_t(a))\pi_f(n+\ell,\Pi_t,\Pi^*_t,0)V_{t+1}(f_2,a,0;n+\ell) \\ +\xi_t(a)\pi(n+\ell,\Pi_t,0)V_{t+1}(i,a,0) \\ +\xi_t(a)[1-\pi(n+\ell,\Pi_t,0)]V_{t+1}(s,a,0) \end{array} \right] \\
& + \frac{\Pi^*_{t-1}\beta(a)}{\Pi_{t-1}+\Pi^*_{t-1}}\chi(\mathcal{V}_t) \left[\begin{array}{c} (1-\xi_t(a))[1-\pi_f(n+\ell,\Pi_t,\Pi^*_t,1)]V_{t+1}(nf_2,a,1;n+\ell) \\ +(1-\xi_t(a))\pi_f(n+\ell,\Pi_t,\Pi^*_t,1)V_{t+1}(f_2,a,1;n+\ell) \\ +\xi_t(a)\pi(n+\ell,\Pi_t,1)V_{t+1}(i,a,1) \\ +\xi_t(a)[1-\pi(n+\ell,\Pi_t,1)]V_{t+1}(s,a,1) \end{array} \right] \\
& + \frac{\Pi_{t-1}\beta(a)}{\Pi_{t-1}+\Pi^*_{t-1}} \left[\begin{array}{c} \phi(0,a)(1-\pi^*(n+\ell,\Pi^*_t)(1-\xi_t(a))W_{t+1}(nf_2,a,\mathcal{V};n+\ell) \\ +\phi(0,a)\pi^*(n+\ell,\Pi^*_t)(1-\xi_t(a))W_{t+1}(f_2,a,\mathcal{V};n+\ell) \\ +(1-\phi(0,a))(1-\alpha(a))(1-\xi_t(a))W_{t+1}(f_2,a,\mathcal{V};n+\ell) \\ +\phi(0,a)\xi_t(a)W_{t+1}(r,a,\mathcal{V}) \\ +(1-\phi(0,a))\alpha(a)W_{t+1}(h,a,\mathcal{V}) \end{array} \right]
\end{aligned}$$

The part multiplied by $\frac{\Pi_{t-1}\beta(a)}{\Pi_{t-1}+\Pi^*_{t-1}}(1-\chi(\mathcal{V}_t))$ captures the continuation if the individual started the period truly healthy and no vaccine is available by the end of the period. The first two lines within the associated square bracket capture the case without testing, where this individual either does not contract another fever and enters nf_2 next period, or does get a fever and enters f_2 next period. The third and fourth lines capture the case of testing, where the individual enters infected or susceptible status depending on whether they got Covid or not during this period. The simplicity of our setup is visible in the fact that the multiplier $\frac{\Pi_{t-1}}{\Pi_{t-1}+\Pi^*_{t-1}}$ does not depend on the actual choices last period. This feature is lost when going to more periods, and the continuation

value V_{t+1} discussed below depends now on the actions taken in the current period. The reason is that they influence beliefs about states in the next period, which is a continuous variable (linked to the continuous variable "time outside"). This increase in state space for the value functions makes the multi-period-uncertainty setup much less tractable.

The part multiplied by $\frac{\Pi_{t-1}\beta(a)}{\Pi_{t-1}+\Pi^*_{t-1}}\chi(\mathcal{V}_t)$ captures the continuation for an indiviual who started the period truly healthy and a vaccine does become available during this period. The terms in the associated bracket are similar to before, but another fever only arises in the case of common cold captured through the indicator $\mathcal{V}=1$ in both π_f and π. Obviously this also affects the beliefs about the infection status in the second period of uncertainty discussed below.

The part multiplied by $\frac{\Pi_{t-1}\beta(a)}{\Pi_{t-1}+\Pi^*_{t-1}}$ captures the continuation if the individual started the period truly infected. In the associated square brackets the first three lines capture the cases without testing. The first is the case where the individual recovers and does not get another cold, in which case they enter nf_2. The second line captures recovery but the person catches a cold (but cannot again get Covid) and enters f_2. The third line is the case where the person does not recover and neither goes to hospital, in which case they enter f_2 (as a truly infected person). The fifth line captures that the individual recovers and gets tested, entering the r state. And the last line captures the transition into hospital, in which case the individual learns their health status and goes into h state. Transitions are again averaged over vaccine arrival which affects future lockdowns, with the only slight twist that for the two second-period untested states $j\in\{f_2,nf_2\}$ the continuation value also conditions on current-period actions:

$$W_{t+1}(j,a,\mathcal{V};n+f)=\chi(\mathcal{V})V_{t+1}(j,a,1;n+\ell)+(1-\chi(\mathcal{V}))V_{t+1}(j,a,0;n+\ell).$$

F.1.4 f_2 fever people in period t:

To discuss the second period of uncertainty for f_2 and nf_2 individuals, it is sometimes convenient to index the transmission probability Π_t by the vaccine availability at the end of that period, i.e., by $\mathcal{V}_{t+1}$. The presence or arrival of a vaccine suppresses further infections and is public knowledge, so we write $\Pi_t(\mathcal{V}_{t+1})$ where $\Pi_t(0)=\Pi_t$ as before and $\Pi_t(1)=0$ in the presence of a vaccine. With this convention we can also write $\pi_f(n_t+\ell_t,\Pi_t(\mathcal{V}_{t+1}))$ instead of the longer $\pi_f(n_t+\ell_t,\Pi_t(\mathcal{V}_{t+1}),\mathcal{V}_t)$ as the number is independent of the last argument. We do the same for $\pi(\cdot)$.

For fever-2 people, $\frac{\Pi^*_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}\pi_f(n_{t-1}+\ell_{t-1},\Pi_{t-1}(\mathcal{V}_t))(1-\xi_{t-1}(a))$ is the chance of entering into the fever-2 state from a healthy state, where $n_{t-1}+\ell_{t-1}$ is *the time that f_1 individuals of the same age spent outside in the previous period.* For such an individual who enter f_2 from a healthy state the following holds:

The chance of being susceptible is $\frac{\Pi^*_{t-1}}{\Pi_{t-1}(\mathcal{V}_t)+\Pi^*_{t-1}}$.

The chance of being infected is $\frac{\Pi_{t-1}(\mathcal{V}_t)}{\Pi_{t-1}(\mathcal{V}_t)+\Pi^*_{t-1}}$.

The chance of entering into the fever-2 state from the infected state is

$$\begin{cases} \frac{\Pi_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}(1-\phi(0,a))(1-\alpha(a))(1-\xi_{t-1}(a)) & \text{for someone who continues to be infected} \\ \frac{\Pi^*_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}\phi(0,a)\pi^*(n_{t-1}+\ell_{t-1},\pi^*_{t-1})(1-\xi_{t-1}(a)) & \text{for someone who recovered} \end{cases}$$

Putting this together, Bayes rule says that the chance of being susceptible conditional on fever-2 state is

$$\begin{aligned} &\Pi_{f2}(\Pi_{t-2},\Pi_{t-1}(\mathcal{V}_t),\Pi^*_{t-1},n_{t-1}+\ell_{t-1}) \\ &= \frac{\frac{\Pi^*_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}\frac{\Pi^*_{t-1}}{\Pi_{t-1}(\mathcal{V}_t)+\Pi^*_{t-1}}\pi_f(n_{t-1}+\ell_{t-1},\Pi_{t-1}(\mathcal{V}_t),\Pi^*_{t-1})}{\left[\begin{array}{c}\frac{\Pi^*_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}\pi_f(n_{t-1}+\ell_{t-1},\Pi_{t-1}(\mathcal{V}_t),\Pi^*_{t-1})+\frac{\Pi_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}(1-\phi(h,a))(1-\alpha(a)) \\ +\frac{\Pi^*_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}\phi(0,a)\pi^*(n_{t-1}+\ell_{t-1},\Pi^*_{t-1})\end{array}\right]} \end{aligned}$$

To compute these beliefs, the actions from the previous period matter. So, the state space of an individual in period t includes all possible actions taken last period, which is an infinite-dimensional object. Or framed otherwise, beliefs in the current period can take any value on a continuum and create therefore an infinite-dimensional state space, while before the beliefs were tied down by a number that depended on the relative probability of the Covid versus a common cold only.

The chance of being recovered is:

$$\begin{aligned} &R_{f2}(\Pi_{t-2},\Pi_{t-1}(\mathcal{V}_t),\Pi^*_{t-1},n_{t-1}+\ell_{t-1}) \\ &= \frac{\frac{\Pi^*_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}\phi(0,a)\pi^*(n_{t-1}+\ell_{t-1},\Pi^*_{t-1})}{\left[\begin{array}{c}\frac{\Pi^*_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}\pi_f(n_{t-1}+\ell_{t-1},\Pi_{t-1}(\mathcal{V}_t),\Pi^*_{t-1})+\frac{\Pi_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}(1-\phi(h,a))(1-\alpha(a)) \\ +\frac{\Pi^*_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}\phi(0,a)\pi^*(n_{t-1}+\ell_{t-1},\Pi^*_{t-1})\end{array}\right]} \end{aligned}$$

The chance of being infected is then the complement of these two.

Except for this belief, the value function for f_2 individuals is otherwise identical to the baseline model:

$$\begin{aligned} &V_t(f_2,a,\mathcal{V}_t;n_{t-1}+\ell_{t-1}) \\ &=\max_{c,x,n,v,\ell,d} u(c,g(x,\ell),d,v) \\ &+\Pi_{f2}(\Pi_{t-2},\Pi_{t-1}(\mathcal{V}_t),\Pi^*_{t-1},n_{t-1}+\ell_{t-1})\tilde{V}_t(c,x,n,l,d;s,a,\mathcal{V}_t) \\ &+[1-\Pi_{f2}(\Pi_{t-2},\Pi_{t-1}(\mathcal{V}_t),\Pi^*_{t-1},n_{t-1}+\ell_{t-1})-R_{f2}(\Pi_{t-2},\Pi_{t-1}(\mathcal{V}_t),\Pi^*_{t-1},n_{t-1}+\ell_{t-1})]\tilde{V}_t(c,x,n,l,d;i,a,\mathcal{V}_t) \\ &+R_{f2}(\Pi_{t-2},\Pi_{t-1}(\mathcal{V}_t),\Pi^*_{t-1},n_{t-1}+\ell_{t-1})\beta(a)W_t(r,a,\mathcal{V}_t) \end{aligned}$$

where $\tilde{V}_t(c,x,n,l,d;s,a,\mathcal{V}_t)$ represents the last three lines of the value function for sus-

ceptible and $\tilde{V}_t(c, x, n, l, d; i, a, \mathcal{V}_t)$ the last three lines of the value function of infected (who know that they are infected) above, and the last line captures the case of continuation with the knowledge of being recovered.

F.2 nf_2 no fever people in period t :

For no-fever-2 people there are again several chances. They might be susceptible or they are recovered. Let again $n_{t-1} + \ell_{t-1}$ *denote the time that* f_1 *individuals of the same age spent outside the previous period.*

The chance to arrive in nf_2 being susceptible is $\frac{\Pi^*_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}[1-\pi_f(n_{t-1}+\ell_{t-1}, \Pi_{t-1}(\mathcal{V}_t), \Pi^*_{t-1})](1-\xi_{t-1}(a))$

The chance to arrive in nf_2 being recovered is $\frac{\Pi_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}\phi(0, a)(1 - \xi_{t-1}(a))$

Putting this together, the chance of being susceptible conditional on the no-fever-2 state is

$$\Pi_{nf_2}(\Pi_{t-2}, \Pi_{t-1}(\mathcal{V}_t), \Pi^*_{t-1}, n_{t-1} + \ell_{t-1})$$
$$= \frac{\frac{\Pi^*_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}[1 - \pi_f(n_{t-1} + \ell_{t-1}, \Pi_{t-1}(\mathcal{V}_t), \Pi^*_{t-1})]}{\frac{\Pi^*_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}[1 - \pi_f(n_{t-1} + \ell_{t-1}, \Pi_{t-1}(\mathcal{V}_t), \Pi^*_{t-1})] + \frac{\Pi_{t-2}}{\Pi_{t-2}+\Pi^*_{t-2}}\phi(0, a)}$$

Then the value function becomes simply

$$\begin{aligned}
&V_t(nf_2, a, \mathcal{V}_t; n_{t-1} + \ell_{t-1}) \\
&= \max_{c,x,n,v,\ell,d} u(c, g(x, \ell), d, v) \\
&+\Pi_{nf_2}(\Pi_{t-2}, \Pi_{t-1}(\mathcal{V}_t), \Pi^*_{t-1}, n_{t-1} + \ell_{t-1})\tilde{V}_t(c, x, n, l, d; s, a, \mathcal{V}_t) \\
&+[1 - \Pi_{nf_2}(\Pi_{t-2}, \Pi_{t-1}(\mathcal{V}_t), \Pi^*_{t-1}, n_{t-1} + \ell_{t-1})]\beta(a)W_{t+1}(r, a, \mathcal{V}_t)
\end{aligned}$$

where $\tilde{V}_t(c, x, n, l, d; s, a, \mathcal{V}_t)$ is as explained before, and $W_{t+1}(r, a, \mathcal{V}_t)$ is the continuation value of being recovered and knowing it.

The value from being in hospital $V_t(h, a)$ and the value of knowingly being recovered $V_t(r, a)$ are unchanged from the baseline model in the main text.

F.3 Type Distributions in the model with two periods of uncertainty

Now we have more uncertainty types than in the baseline model. We have f_1, as well as f_2 and nf_2. The f_1 state can be split into fi_1 for "fever-infected1" and fs_1 for "fever-susceptible1". The same we can do for f_2, which can be split into fi_2 "fever-infected2" and fs_2 "fever-susceptible2" and fr_2 "fever-recovered2" (where the latter are recovered individuals who have a cold and do not know that they have recovered).

Then we also can split the nf_2 into nfs_2 for "no-fever-susceptible2" and nfr_2 for "no-fever-recovered2".

The system of transition equations is similar to before. Just where there used to be one fever state, there are now f_1, f_2 and nf_2. From the other states s, i, h, r individuals flow into the first fever state. People flow out of the second fever state and the second non-fever-state back into the other states s, i, h, r. And then we need transitions between the first and the second fever states. Note that optimal choices now not only condition on the current vaccine availability $\mathcal{V}_t$, but also sometimes on past vaccine availability $\mathcal{V}_{t-1}$. This is innocuous for $\mathcal{V}_t = 0$, as $\mathcal{V}_{t-1} = 0$ trivially in this case.

We will first discuss the case where no vaccine has arrived until $t+1$.

For the s state we now have evolution:

$$\begin{aligned}
M_{t+1}(s,a) \;=\;& M_t(s,a)\Delta(a)\left[\begin{array}{c} 1-\pi_f(n_t(s,a,\mathcal{V}_t)+\ell_t(s,a,\mathcal{V}_t),\Pi_t,\Pi_t^*,\mathcal{V}_t) \\ +\pi^*(n_t(s,a,\mathcal{V}_t)+\ell_t(s,a,\mathcal{V}_t),\Pi_t^*)\xi_t(a) \end{array}\right] \\
&+M_t(nfs_2,a)\Delta(a)\left[\begin{array}{c} 1-\pi_f(n_t(nf_2,a,\mathcal{V}_t,\mathcal{V}_{t-1})+\ell_t(nf_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}),\Pi_t,\Pi_t^*,\mathcal{V}_t) \\ +\pi^*(n_t(nf_2,a,\mathcal{V}_t,\mathcal{V}_{t-1})+\ell_t(nf_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}),\Pi_t^*)\xi_t(a) \end{array}\right] \\
&+M_t(fs_2,a)\Delta(a)\left[\begin{array}{c} 1-\pi_f(n_t(f_2,a,\mathcal{V}_t,\mathcal{V}_{t-1})+\ell_t(f_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}),\Pi_t,\Pi_t^*,\mathcal{V}_t) \\ +\pi^*(n_t(f_2,a,\mathcal{V}_t,\mathcal{V}_{t-1})+\ell_t(f_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}),\Pi_t^*)\xi_t(a) \end{array}\right] \\
&+M_t(fs_1,a)\Delta(a)[1-\pi(n_t(f_1,a,\mathcal{V}_t)+\ell_t(f_1,a,\mathcal{V}_t),\Pi_t,\mathcal{V}_t)]\xi_t(a)
\end{aligned}$$

The first line is the same as in the baseline model and describes who of the susceptible stay susceptible next period. The second line uses exactly the same terms but applies them to the second-period non-fever-susceptible people. The third line does the same for the second-period fever-susceptible. The first three lines look similar, but the actions for n and l that are taken are different. The last line is for the fever-susceptible in the first period: They can only become susceptible if they get tested. And then only if they did not catch the disease this period. (In the last line it is the function π that indicates actually contracting Covid; it is not function π_f for any type of fever or π^* for a simple cold as in the previous lines.)

The evolution for people who know that they are infected now becomes

$$\begin{aligned}
M_{t+1}(i,a) \;=\;& M_t(s,a)\Delta(a)\xi_t(a)\pi(n_t(s,a,\mathcal{V}_t)+\ell_t(s,a,\mathcal{V}_t),\Pi_t,\mathcal{V}_t) \\
&+M_t(nfs_2,a)\Delta(a)\xi_t(a)\pi(n_t(nf_2,a,\mathcal{V}_t,\mathcal{V}_{t-1})+\ell_t(nf_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}),\Pi_t,\mathcal{V}_t) \\
&+M_t(fs_2,a)\Delta(a)\xi_t(a)\pi(n_t(f_2,a,\mathcal{V}_t,\mathcal{V}_{t-1})+\ell_t(f_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}),\Pi_t,\mathcal{V}_t) \\
&+M_t(fs_1,a)\Delta(a)\xi_t(a)\pi(n_t(f_1,a,\mathcal{V}_t,\mathcal{V}_{t-1})+\ell_t(f_1,a,\mathcal{V}_t,\mathcal{V}_{t-1}),\Pi_t,\mathcal{V}_t) \\
&+[M_t(i,a)+M_t(fi_2,a)+M_t(fi_1,a)\xi_t(a)](1-\phi(0,a))(1-\alpha(a))
\end{aligned}$$

The first line remains unchanged and captures those who know that they are susceptible who get Covid this period and are tested. The next three lines have the same

expressions for agents that are susceptible but do not know their status. The last line captures all those who were already infected and who neither recover nor go to the hospital. People in the i state know their type and those in fi_2 state get to know it at the end of the period, but those in fi_1 state only get to know it if they get tested.

For those in the hospital state h:

$$\begin{aligned} M_{t+1}(h,a) &= [M_t(i,a) + M_t(fi_2,a) + M_t(fi_1,a)]\Delta(a)(1-\phi(0,a))\alpha(a) \\ &\quad + M_t(h,a)\Delta(a)(1-\delta_t(a))(1-\phi(1,a)) \end{aligned}$$

The hospital transitions remain essentially unchanged except that all infected states are included in the first line. The first line captures infected individuals who do not recover but go to hospital (where their type is revealed even if they did not know before). The second line captures those who were already in hospital and neither recovered nor died.

Those that know that they have recovered, i.e., state r:

$$\begin{aligned} M_{t+1}(r,a) &= [M_t(i,a) + M_t(fi_2,a) + M_t(fi_1,a)\xi_t(a)]\Delta(a)\phi(0,a) \\ &\quad + M_t(h,a)\Delta(a)\phi(1,a) \\ &\quad + [M_t(r,a) + M_t(nfr_2,a) + M_t(fr_2,a)]\Delta(a) \end{aligned}$$

The first line adds all infected agents that recover, but for period-one this only counts those that get tested (because untested agents in period 1 remain unsure about their type). The second line captures recovery from hospital. The third line adds all recovered agents that survive (note that there are no such individuals in the period-1-fever state). This includes the nfr_2 and the fr_2 agents, whose status is revealed at the end of the second period.

The evolution for Covid-deceased agents does not change:

$$M_{t+1}(deceased,a) = M_t(deceased,a) + (1-\phi(1,a))\delta(a)M_t(h,a)\Delta(a).$$

First period of fever:

$$\begin{aligned} M_{t+1}(fs_1,a) &= M_t(s,a)\Delta(a)(1-\xi_t(a))\pi^*(n_t(s,a,\mathcal{V}_t) + \ell_t(s,a,\mathcal{V}_t), \Pi_t^*) \\ &\quad + M_t(fs_2,a)\Delta(a)(1-\xi_t(a))\pi^*(n_t(f_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}) + \ell_t(f_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}), \Pi_t^*) \\ &\quad + M_t(nfs_2,a)\Delta(a)(1-\xi_t(a))\pi^*(n_t(nf_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}) + \ell_t(nf_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}), \Pi_t^*) \end{aligned}$$

This is identical to the expression (16) for fever-susceptible in the baseline model, only that the second line captures the fever-susceptible in the second period who can again become fever-1-susceptible through a new cold. This does not involve fever-

susceptible from period 1, as these either move to period 1 or get tested and their state revealed. (It is π^* in the formula, i.e., the chance of contracting the common cold).

Fever-infected fi_1:

$$\begin{aligned} M_{t+1}(fi_1, a) &= M_t(s,a)\Delta(a)(1-\xi_t(a))\pi(n_t(s,a,\mathcal{V}_t,\mathcal{V}_{t-1}) + \ell_t(s,a,\mathcal{V}_t,\mathcal{V}_{t-1}),\Pi_t,\mathcal{V}_t) \\ &\quad + M_t(fs_2,a)\Delta(a)(1-\xi_t(a))\pi(n_t(f_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}) + \ell_t(f_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}),\Pi_t,\mathcal{V}_t) \\ &\quad + M_t(nfs_2,a)\Delta(a)(1-\xi_t(a))\pi(n_t(nf_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}) + \ell_t(nf_2,a,\mathcal{V}_t,\mathcal{V}_{t-1}),\Pi_t,\mathcal{V}_t) \end{aligned}$$

It has the same logic as for fs_1, just that these individuals got Covid instead of the common cold. (Note that it is π in the formula.)

Second-period:

These transition probabilities have no analogue in the baseline model.

Fever-susceptible fs_2 :

$$M_{t+1}(fs_2,a) = M_t(fs_1,a)\Delta(a)(1-\xi_t(a))\pi^*(n_t(f_1,a,\mathcal{V}_t) + \ell_t(f_1,a,\mathcal{V}_t),\Pi_t^*)$$

It includes those that are fever-susceptible in period 1, do not get tested, and get another cold.

Fever-infected fi_2:

$$\begin{aligned} M_{t+1}(fi_2, a) &= M_t(fs_1,a)\Delta(a)(1-\xi_t(a))\pi(n_t(f_1,a,\mathcal{V}_t) + \ell_t(f_1,a,\mathcal{V}_t),\Pi_t,\mathcal{V}_t) \\ &\quad + M_t(fi_1,a)\Delta(a)(1-\xi_t(a))(1-\alpha(a))(1-\phi(0,a)) \end{aligned}$$

It captures the fever-susceptible from period 1 that do not get tested and now get Covid, as well as the fever-infected from period 1 that do not go to the hospital nor get healed nor tested.

Fever-recovered fr_2:

$$M_{t+1}(fr_2,a) = M_t(fi_1,a)\Delta(a)\phi(0,a)\pi^*(n_t(f_1,a,\mathcal{V}_t) + \ell_t(f_1,a,\mathcal{V}_t),\Pi_t^*)(1-\xi_t(a))$$

They comprise the first-period fever-infected who recover but get another cold and are not tested.

No-fever-recovered nfr_2:

$$M_{t+1}(nfr_2,a) = M_t(fi_1,a)\Delta(a)\phi(0,a)(1-\pi^*(n_t(f_1,a,\mathcal{V}_t) + \ell_t(f_1,a,\mathcal{V}_t),\Pi_t^*))(1-\xi_t(a))$$

The no-fever-recovered comprise the period-1-fever-infected who recover and do not get a cold nor are tested.

No-fever-susceptible nfs_2:

$$M_{t+1}(nfs_2, a) = M_t(fs_1, a)\Delta(a)(1-\xi_t(a))[1-\pi_f(n_t(f_1, a, \mathcal{V}_t) + \ell_t(f_1, a, \mathcal{V}_t), \Pi_t, \Pi_t^*, \mathcal{V}_t)]$$

They comprise the period-1-fever-susceptible who are not tested and do not develop a fever.

If a vaccine arrived by $t+1$, the equations in this subsection remain unchanged except that π_f, π and π^* need to be set to zero as no new infections and no new confusion with fever arise.

F.4 Aggregation in the model with two periods of uncertainty

One can now add up the period infection statistic as

$$\hat{\Pi}_t = \Pi_0 \sum_{\tilde{a}, j \in \{fi_1, fi_2, i, h\}} (n_t(j, \tilde{a}, \mathcal{V}_t, \mathcal{V}_{t-1}) + \ell_t(j, \tilde{a}, \mathcal{V}_t, \mathcal{V}_{t-1}))M_t(j, \tilde{a})$$

and output as

$$Q_t = \sum_j w[n_t(j, y, \mathcal{V}_t, \mathcal{V}_{t-1}) + \iota(v_t(j, y, \mathcal{V}_t, \mathcal{V}_{t-1}))v_t(j, y, \mathcal{V}_t, \mathcal{V}_{t-1})]M_t(j, y)$$

where the sum over j now includes all (including the new) states (and where the argument of the action includes $\mathcal{V}_{t-1}$ even though this is only relevant for f_2-individuals and nf_2-individuals).

And the aggregate capacity constraint for testing becomes

$$\xi_t(a) = K_t / \left[\begin{array}{c} \sum_{\tilde{a}} A_{\tilde{a}}[M_t(s, \tilde{a})\pi_f(n_t(s, \tilde{a}, \mathcal{V}_t) + \ell_t(s, \tilde{a}, \mathcal{V}_t), \Pi_t(\mathcal{V}_t))+ \\ M_t(fs_1, a) + M_t(fi_1, a)+ \\ M_t(fs_2, \tilde{a})\pi_f(n_t(f_2, \tilde{a}, \mathcal{V}_t, \mathcal{V}_{t-1}) + \ell_t(f_2, \tilde{a}, \mathcal{V}_t, \mathcal{V}_{t-1}), \Pi_t, \Pi_t^*, \mathcal{V}_t)+ \\ M_t(fr_2, \tilde{a})\pi^*(n_t(f_2, \tilde{a}, \mathcal{V}_t, \mathcal{V}_{t-1}) + \ell_t(f_2, \tilde{a}, \mathcal{V}_t, \mathcal{V}_{t-1}), \Pi_t^*)+ \\ M_t(nfs_2, \tilde{a})\pi_f(n_t(nf_2, \tilde{a}, \mathcal{V}_t, \mathcal{V}_{t-1}) + \ell_t(nf_2, \tilde{a}, \mathcal{V}_t, \mathcal{V}_{t-1}), \Pi_t, \Pi_t^*, \mathcal{V}_t)+ \\ M_t(nfr_2, \tilde{a})\pi^*(n_t(nf_2, \tilde{a}, \mathcal{V}_t, \mathcal{V}_{t-1}) + \ell_t(nf_2, \tilde{a}, \mathcal{V}_t, \mathcal{V}_{t-1}), \Pi_t^*)+ \\ M_t(r, \tilde{a})\pi^*(n_t(r, \tilde{a}, \mathcal{V}_t) + \ell_t(r, \tilde{a}, \mathcal{V}_t), \Pi_t^*)] \end{array} \right]$$

Here the susceptible and recovered might get tested if they get new symptoms and the fever-1 individuals might get tested based on their current status.

One can also create other statistics by adding up, as all agents that have some in-

fection I (whether known or not)

$$\begin{aligned} M_t(I,a) &= M_t(i,a) + M_t(fi_1,a) + M_t(fi_2,a) \\ M_t(I) &= \sum_{\tilde{a}} M_t(I,\tilde{a}) \end{aligned}$$

or that have recovered R (whether known or not)

$$\begin{aligned} M_t(R,a) &= M_t(r,a) + M_t(fr_2,a) + M_t(nfr_2,a) \\ M_t(R) &= \sum_{\tilde{a}} M_t(R,\tilde{a}). \end{aligned}$$

Table F15: The Effects of Testing with 2-period uncertainty (robustness exercise for Table 7)

		Covid R_0 No testing 1-period uncertainty (1a)	Covid R_0 No testing 2-period uncertainty (1b)	Covid R_0 Full testing 1&2-period uncertainty (2)	$0.5\times$ Covid R_0 No testing 1-period uncertainty (3a)	$0.5\times$ Covid R_0 No testing 2-period uncertainty (3b)	$0.5\times$ Covid R_0 Full testing 1&2-period uncertainty (4)
Panel A. Realized statistics							
Weeks to peak infections	Young	10	8	45	48	47	1
	Old	11	9	46	48	47	1
Hrs @ home - avg. first year, diff. w.r.t. no-disease	Young	7.42	7.44	3.86	1.05	1.26	0.10
	Old	15.32	15.11	11.68	4.83	5.72	0.54
Dead p/ 1,000, first year	Young	1.15	1.27	0.44	0.11	0.15	0.01
	Old	19.74	22.49	7.60	2.25	2.98	0.30
	All	5.13	5.81	1.97	0.57	0.76	0.07
Dead p/ 1,000 (by vaccine arrival)	Young	1.23	1.32	0.54	0.17	0.23	0.01
	Old	21.16	23.28	8.89	3.12	4.06	0.31
	All	5.49	6.02	2.33	0.80	1.05	0.08
Recovered , % (by vaccine arrival)	Young	53.88	57.75	23.84	7.53	10.17	0.65
	Old	22.23	24.34	9.47	3.35	4.36	0.32
	All	47.10	50.60	20.77	6.63	8.93	0.58
GDP 1 year, % change w.r.t. no-disease		-6.16	-6.29	-3.60	-1.86	-1.98	-1.31
Panel B. Expected statistics							
Hrs @ home, diff. w.r.t. no-disease	Young	6.33	6.54	2.56	0.78	0.83	0.15
	Old	12.61	12.12	8.74	3.83	4.24	0.74
Dead p/ 1,000	Young	0.96	1.06	0.37	0.11	0.15	0.01
	Old	16.28	18.20	6.04	2.09	2.62	0.26
	All	4.24	4.72	1.58	0.54	0.67	0.07
GDP, % change w.r.t. no-disease		-5.84	-6.11	-3.17	-2.07	-2.10	-1.70

Note: Columns (1a), (2), (3a) and (4) in this table replicate Table 7). Columns (1b), (2), (3b) and (4) in this table provide the same statistics for the model with 2 periods of uncertainty. In column (2) and (4) there is no difference between the model with 1 and 2 periods of uncertainty since full testing eliminates the uncertainty.