Life Expectancy and Economic Growth: Local or Global GDP?

Preliminary

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Abstract

This paper documents that global economic growth is responsible for the majority of worldwide life expectancy gains. We emphasize the role of the economic frontier by exploiting time-series variation in global GDP. First, we show that innovations to global GDP are associated with substantial reductions in mortality throughout the world. Our estimates imply that doubling global GDP avoids 6 deaths per 1,000 individuals. These estimates are three times larger than the effect of innovations to local, country-specific growth, which have been the focus of the literature. Second, we instrument global GDP with monetary policy shocks in the United States to account for unobserved confounders and find quantitatively similar results. Third, we embed these global longevity spillovers in a standard growth model with finite lifespans and health production. Global spillovers account for over 70% of life expectancy improvements between 1960 and 2019, compared to around 10% for local growth. When internalizing spillovers, aggregate output further rises by more than 10%, life expectancy by 2 years and welfare by 6%.

JEL classification: I15, O11, E32, F41

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1 Introduction

Does wealth improve health? Income and life expectancy have risen dramatically over the last century. Higher incomes may improve longevity and reduce mortality, for instance because of better sanitation, nutrition or health care. Despite the multitude of possible channels, the evidence linking economic growth to mortality remains conflicted. Existing estimates imply that rising incomes at the country level account for less than a fifth of mortality reductions (Preston, 1975), or even increase mortality (Ruhm, 2000). Does wealth, then, not improve health? Or do existing estimates not account for the full effect of worldwide economic growth on mortality?

In this paper, we show that the effect of economic growth on mortality is an order of magnitude larger than previously estimated by emphasizing the role of the economic frontier. We rely on panel local projections to estimate the impact of global Gross Domestic Product (GDP) changes on mortality around the world. We exploit unconditional fluctuations in global GDP, as well as an instrumental variable approach relying on monetary policy surprises in the United States that is plausibly unrelated to confounders such as conflicts and epidemics. Our estimates imply that permanently doubling global GDP lowers mortality by 6 deaths per 1,000, nearly half of baseline mortality. This relationship together with observed economic growth accounts for 72% of the longevity improvements over the last century. This impact is three times larger than the effect of changes in local, country-level GDP, and suggests that expansions in the economic frontier have important spillovers on life expectancy around the world. We incorporate this mechanism in a standard growth model with mortality. Internalizing longevity spillovers increases aggregate output by 11%, life expectancy by 2 years and welfare by 6%.

In the first part of the paper, we develop our empirical approach. We assemble a dataset with economic and mortality information at the country and year level. We obtain macroeconomic data, including GDP and population, from the Penn World Tables and the World Bank's World Development Indicators. For mortality, we draw primarily on data from the Human Mortality Database and the World Health Organization. The Human Mortality Database provides high-quality mortality data for high-income countries, while the World Health Organization database substantially expands coverage to Latin America, Asia, the Middle East, and parts of Africa. We harmonize these sources to construct consistent mortality measures across countries. Because these international databases provide limited early coverage for China and India—two of the most populous

countries in the world during much of our sample—we complement them with national mortality statistics for both countries. Our sample includes nearly 100 countries, representing up to 80% of the world's population.

Identifying the impact of income on mortality is complicated by their jointly trending behavior. We thus construct global and local (country-level) GDP shocks: innovations to GDP per capita processes that are orthogonal to their long-run trends using the approach in Hamilton (2018). We use this approach to highlight the identifying variation, although it is equivalent to controlling for the appropriate number of lags in a single local projection.

Global GDP per capita fluctuates for many reasons that are plausibly unrelated to mortality shocks: unexpectedly fast technological progress, financial crises, political shocks, and so on. Therefore, the local projection of mortality on GDP per capita fluctuations is likely informative about the underlying relationship between income and longevity.

However, important omitted variables may lead global GDP per capita and mortality to co-move: epidemics or conflicts tend to depress economic activity and increase mortality, but are unrelated to our causal effect of interest. In addition, positive health improvements may lead to reverse causality if healthier individuals are more productive. To pose a threat to identification, qualitatively, omitted variables have to be large enough to affect worldwide economic activity at the same time as affecting mortality throughout the world. Reverse causality would have to operate at the high frequencies that we consider—of course a possibility, if not a particularly likely one. In both cases, to be quantitatively meaningful, these shocks have to dominate other sources of global GDP fluctuations.

We address these potential omitted variable and reverse causality problems with an instrumental-variable approach. We leverage that monetary policy in the United States affects worldwide GDP through international trade, financial linkages and currency pegs. We measure U.S. monetary policy shocks as quarterly unexpected deviations from Federal Reserve forecasts following Romer and Romer (2004), that we aggregate at the annual frequency.

Our identification assumption is that Federal Reserve forecasts incorporate all information relevant to United States GDP about confounders such as conflicts and epidemics. Greenbook forecasts are finalized two to five days before Federal Open Market Committee (FOMC) meetings, ensuring that the likelihood of a major confounder occurring in

between is low. In addition, the mandate of the Federal Reserve is to stabilize prices and economic activity in the United States. Thus, even if information available to the Federal Reserve did not include all possible confounders, monetary policy surprises should not respond directly to overseas mortality events that may impact economic activity overseas but not in the United States. Under these conditions, monetary policy surprises are unlikely to be related to global mortality shocks and thus constitute a valid instrument for global GDP fluctuations.

We map out the dynamic effects of global GDP per capita shocks on country-level mortality using local projections. In our initial exercise, a global GDP shock scaled to 1% on impact leads to a hump-shaped reduction in mortality that peaks at 0.07 deaths per 1,000 individuals six years out. The effect is statistically different from zero at the 95% level at horizons three to ten. The 95% confidence interval for the peak effect at horizon six is (-0.09,-0.04) deaths per 1,000 individuals. We do not detect any statistically significant nor economically meaningful pre-trends. The results are similar irrespectively of whether we include or exclude India and China from the mortality sample. They also remain unchanged for alternative de-trending approaches, such as a one-sided Hodrick-Prescott filter, taking simple first differences, or estimating our effect of interest in a single local projection without isolating the identifying variation first.

Importantly, global GDP shocks have a partially persistent effect on the level of global GDP itself, reflecting autocorrelation in the GDP process. Hence, the mortality effects six years out reflect the accumulated effects of persistently elevated global GDP itself, but are less than the implied effect of a permanent global GDP increase.

We convert these estimates identified from partially transitory global GDP shocks to the effect of a permanent increase in global GDP by taking the ratio of the cumulative impulse responses of mortality and global GDP. Our reduced-form estimates imply that a permanent rise in global GDP per capita scaled to 100% leads to a mortality reduction of 6 deaths per 1,000 individuals.

Although global growth occurs in a sequence of small increments, this conversion relies on the conditions that the relationship between GDP and mortality holds beyond the range of GDP shocks observed in sample, and that it holds for expected permanent GDP changes in addition to medium-run unexpected GDP changes.

Next, we assess the robustness of our baseline results to omitted variables in a series of exercises. Our baseline specification includes a comprehensive set of controls: we

control for four lags of the mortality rate and two lags of the shock, global GDP growth and local GDP growth, the U.S. real effective exchange rate, as well as subregion-specific linear trends and dummies for the Great Recession. We then show that the results are largely insensitive to the set of controls included. In particular, the estimates are robust to augmenting the specification with controls for conflicts and epidemics as well as demographic variables. We also obtain very similar results when excluding the real effective exchange rate, the Great Recession dummy, or the region-specific trends. Visualizing the entire identifying variation indicates that our estimates are driven by the full sample rather than by a few outliers. A jackknife exercise in which we drop each year in turn also yields very similar estimates.

We then turn to our instrumental variable strategy. We run two local projections, corresponding to the first stage and the reduced form. First, we estimate the effect of United States monetary policy surprises on GDP per capita around the world. We augment our specification with standard monetary controls to capture the global financial cycle: changes in the one year U.S. treasury rate, the ten year spread, and commodity price inflation. Consistent with well-established results, GDP per capita rise around the world following a surprise monetary expansion in the United States. The estimated responses are precisely estimated and robust to dropping controls or excluding years one at a time.

Second, we estimate the effect of monetary policy surprises on mortality. An expansionary surprise that increases world GDP by 1% at peak causes mortality to fall by 0.05 deaths per 1,000 individuals after three years, and remains persistently low thereafter. These estimates are somewhat less precise than our baseline effects, statistically significant at the 95% level in years three and eight to ten. When we estimate a pooled effect after the expansionary shock, we obtain a strongly significant estimate. We do not detect any statistically significant nor economically meaningful pre-trends. Our results hold when we drop our controls one at a time, when we drop years from the sample one a time, and for alternative detrending approaches.

We combine these first stage and reduced form instrumental variable estimates to gauge the causal effect of a permanent increase in global GDP. We take the ratio of the cumulative impulse responses of mortality and global GDP, each to the monetary policy surprise. The resulting estimate implies that a permanent rise in global GDP per capita scaled to 100% leads to a mortality reduction of 8 deaths per 1,000 individuals. While this point estimate is somewhat larger than our ordinary least square estimate, they are not

statistically distinguishable from each other.

Our estimate of the impact of GDP shocks on mortality contrasts with existing work. Preston (1975) uses cross-sectional comparisons to infer that doubling GDP lowers mortality by 1 to 2 deaths per 1,000, and can account for less than one fifth of observed improvements in life expectancy. Since then, panel-based approaches in Summers and Pritchett (1996), Ruhm (2000), Ruhm (2003), and Ruhm (2007), Baird et al. (2011), Cutler et al. (2016), Ma et al. (2022), and Finkelstein et al. (2025), all find that increases in GDP lead to estimates of similarly modest magnitudes, sometimes even negative. Why do we find effects that are positive and an order of magnitude larger?

We focus on a different source of variation. Changes in global GDP capture the comprehensive impact of expansions at the economic frontier. By contrast, previous work exploits changes in country-level, local GDP. When we estimate the impact of local GDP shocks on country-level mortality with the same empirical specification, we find similar magnitudes to previous studies. Our estimates imply that doubling local GDP reduces mortality by a statistically significant 2 excess deaths per 1,000, three times less than for global GDP.

Why, then, do global GDP expansions reduce mortality more than local GDP expansions? Our results suggest that there are important spillovers from growth at the economic frontier toward longevity in the rest of the world. Qualitatively, these longevity spillovers are consistent with two broad mechanisms. First, they are consistent with the influential line of work that finds beneficial health effects of public health interventions in lower-income regions (Miguel and Kremer, 2004; Bendavid et al., 2012; Pryce et al., 2018), often partly funded by institutions in higher-income regions such as the World Bank, the World Health Organization, the Global Fund to Fight AIDS, Tuberculosis and Malaria, non-governmental organizations, USAID or PEPFAR (Schieber et al., 2006; Hanson et al., 2022). Our analysis suggests that economic growth at the frontier may enable these interventions to take place. Second, longevity spillovers are also qualitatively consistent with international pharmaceutical or food price reductions, although the latter would have to be large enough not to be captured by local GDP estimates.[1]

We investigate these mechanisms empirically, although we do not find conclusive evidence in favor of any. First, while we find an association between global expansions and

[1]They are in principle also consistent with international diffusion of medical knowledge fueled by growth at the frontier, although the long time lags in medical research are difficult to reconcile with our high frequency analysis.

foreign aid flows, as well as between foreign aid and mortality, the combination of both accounts only for a moderate fraction of our direct estimate, perhaps due to the limited coverage of broadly available foreign aid data. Second, food prices tend to increase more than one-for-one in global expansions rather than fall. Pharmaceutical price and trade changes can account only for a negligible share of longevity gains. While our analysis is not definitive given the data limitations that we face, the lack of a clear explanation constitutes an important puzzle for future research.

In the second part of the paper, we explore the consequences of global spillovers from the economic frontier to worldwide longevity in a simple model of growth with finite lifespans. We start from the neoclassical growth model, in which households face stochastic lifespans as in Blanchard (1985). At the end of life, households experience a one-time disutility and transfer their capital to their single offspring. These assumptions ensure that investment decisions are undistorted absent other externalities. In addition to investing, households can spend their income on final consumption or on health, which lowers their mortality risk according to a health production function. Mortality also depends on aggregate GDP, which is neither internalized by households nor by firms. This spillover implies that investment decisions are dynamically inefficient: investment is too low as households fail to internalize that higher GDP in the future lowers their mortality risk.

We then quantify our framework. We estimate the elasticities of mortality to private health expenditures and to aggregate GDP by matching our reduced-form impulse response functions in the data. Consistent with our reduced-form estimates, we find that the elasticity of mortality to aggregate GDP is five times larger than to private health expenditures. The Value of Statistical Life (VSL) identifies the end-of-life disutility and thus affects the magnitude of the dynamic inefficiency. The health spending share identifies the shifter of the health production function.

Equipped with our estimated framework, we ask how the world economy would have changed between 1960 and 2019 without global longevity spillovers. While output growth would have remained virtually identical, we find that global mortality, life expectancy and welfare would have changed substantially.

Global GDP growth accounts for 72% of observed life expectancy improvements from 1960 to 2019. By contrast, private health spending changes due to local GDP growth account for 9%, with the remaining 19% unexplained by our estimated model. Absent

longevity spillovers, mortality would have fallen by only 0.8 death per 1,000 instead of 4.6. Life expectancy would have increased by only 2 years instead of 16. Welfare gains from growth would have been nearly halved.

Given the importance of global longevity spillovers for understanding historical changes in worldwide life expectancy, we then ask how today's global economy might look like if these spillovers were internalized. We find that output would be 11% higher to harness the longevity improvements from spillovers, leading to over 2 years of additional life expectancy and 6% in welfare gains. These results suggest that global longevity spillovers may represent an important margin to improve well-being around the globe.

Related literature. Our paper contributes to an influential literature measuring whether rising income causes mortality reductions. The seminal work of Preston (1975) points out that while richer countries exhibit lower mortality rates, movements along that cross-sectional relationship—the 'Preston curve'—can account only for a modest fraction of overall declines in mortality around the globe. Consistent with this insight and using panel designs across countries, many papers find that rising local GDP lowers mortality with a comparable magnitude to the cross-sectional correlation (Summers and Pritchett, 1996; Baird et al., 2011; Cutler et al., 2016; Shapira et al., 2021; Ma et al., 2022). Our approach focuses on a fundamentally different explanatory variable: we emphasize the role of global GDP and the economic frontier, which we show leads to quantitatively important international spillovers. Econometrically, it represents the missing intercept in the panel approach, which we show has significant positive and normative implications.

A parallel literature assesses whether unemployment changes affect mortality within the United States or the United Kingdom. This strand of work sometimes find the opposite sign but with a comparable absolute magnitude to local GDP shocks. These papers focus on business cycle correlations or panel variation across regions (Brenner, 1971; Brenner, 1979; Ruhm, 2000; Ruhm, 2003; Ruhm, 2007; Dehejia and Lleras-Muney, 2004; Stevens et al., 2015; Drautzburg et al., 2024; Finkelstein et al., 2025). Our analysis emphasizes global GDP and international spillovers, which we find to be an order of magnitude larger and significant for life expectancy around the world.[2]

Our paper also relates broadly to the literature that evaluates welfare gains from observed life expectancy improvements (Becker et al., 2005; Murphy and Topel, 2006; Hall

[2]In addition, we find that local hours worked and local GDP have opposite effects on mortality, proposing a possible resolution to differing signs when focusing on GDP or unemployment.

and Jones, 2007; Jones and Klenow, 2016; Escosura, 2023; Brouillette et al., 2025). Our empirical contribution is to estimate the causal effect of global growth on mortality and life expectancy. Our framework is a simple extension of those in this line of work augmented with global longevity spillovers.

The rest of this paper is organized as follows. Section 2 describes the data. Section 3 constructs our baseline and instrumental variable estimates of the impact of global GDP on mortality. Section 4 compares the effect of global GDP to the effect of local GDP. Section 5 presents our framework and counterfactuals. Section 6 concludes.

2 Data and Empirical Approach

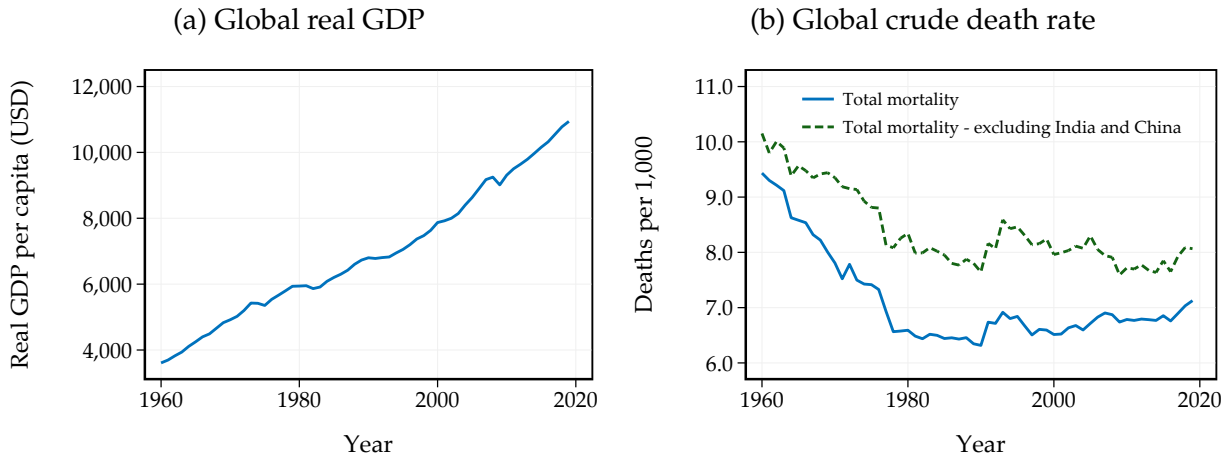
2.1 A New Mortality Panel

Our goal is to estimate the impact of economic growth on mortality. To that end, we assemble a new country-year panel combining comprehensive mortality and macroeconomic data.

For mortality, we draw primarily on the Human Mortality Database (HMD) and the World Health Organization (WHO) Mortality Database. The HMD provides long and internally consistent series for high-income countries. The WHO database substantially expands coverage to Latin America, Asia, the Middle East, and parts of Africa. Coverage for Africa is more limited and often shorter in duration. We harmonize these sources and construct mortality rates by dividing death counts by population, expressed as deaths per 1,000 individuals. When necessary, we use growth rates from auxiliary sources to interpolate or extend missing segments, as detailed in Appendix A. Because international databases provide limited early coverage for India and China, we incorporate official national sources for these countries. As mortality data for China varies across sources, we first conservatively report results without China in our sample, and next describe how including it affects our results.

The United Nations World Population Prospects (UNWPP) provides broader geographic coverage, particularly for African countries. However, these series are heavily interpolated and reflect model-based demographic reconstructions rather than actual death counts. Because our empirical strategy exploits time-series variation in mortality, we do not use UNWPP mortality rates in the baseline specification. In sensitivity checks, we

Figure 1: Global Output and Mortality Since 1960



Notes: Panel (a): evolution of global real GDP per capita (PWT, chain-weighted) since 1960. Panel (b): evolution of global crude death rate (deaths per 1,000 population) computed from HMD, WHO, and national statistics, weighted by country population.

complement our main dataset with UNWPP data to assess robustness to broader coverage of African countries.

We combine these mortality data with macroeconomic information from the Penn World Tables (PWT) and the World Bank's World Development Indicators (WDI). We obtain GDP and population data for a broad set of countries over 1960–2019. In addition, we include a set of global and U.S.-specific variables—including commodity prices, U.S. treasury yields, andthe real exchange rate—drawn from Global Financial Data and the TRADHIST database.

Our baseline sample includes just over 50 countries in 1960, expanding to nearly 100 countries by 2019. Throughout 1960–2019, the sample represents a relatively stable share of the world population, fluctuating between 60 and 80%.

Figure 2 shows the evolution of global GDP and mortality over our sample. Global real GDP per capita rises steadily from 1960 onward, more than doubling over the period. Over the same decades, the crude death rate declines sharply, particularly between 1960 and the early 1980s, before flattening somewhat thereafter. Excluding India and China yields a similar pattern, though at a higher level. Overall, the figure indicates that global income and mortality trend in opposite directions. While suggestive, these patterns are only descriptive.

2.2 Global GDP Shocks

The descriptive patterns in Figure 2 raise two concerns. First, global GDP and mortality are both trending over our sample. Regressions in levels may therefore reflect spurious correlations in the spirit of Granger and Newbold (1974). Second, common shocks—such as wars, global recessions, or pandemics—may affect both economic activity and mortality, confounding the interpretation of simple correlations.

To address the first concern, we isolate innovations to global GDP following the procedure proposed by Hamilton (2018). We project global real GDP per capita on its own lags as of period $t - h$ and define the GDP shock as the corresponding innovation:

$$\widehat{y_t^{\text{shock}}} = y_t - (\widehat{\alpha} + \widehat{\beta}_1 y_{t-h} + \ldots + \widehat{\beta}_{p+1} y_{t-h-p}), \tag{1}$$

where $\widehat{\beta}_i$ denotes the coefficient estimates of the regression of GDP on its lag $j = (h + i - 1)$ and $\widehat{\alpha}$ is the estimated intercept. This procedure removes the low-frequency trend component and isolates deviations that persist for approximately h periods. We set the horizon to $h = 1$ and the number of lags to $p = 2$. Varying these parameters leaves the results largely unchanged. The resulting GDP shocks fluctuate around zero and display limited serial correlation.

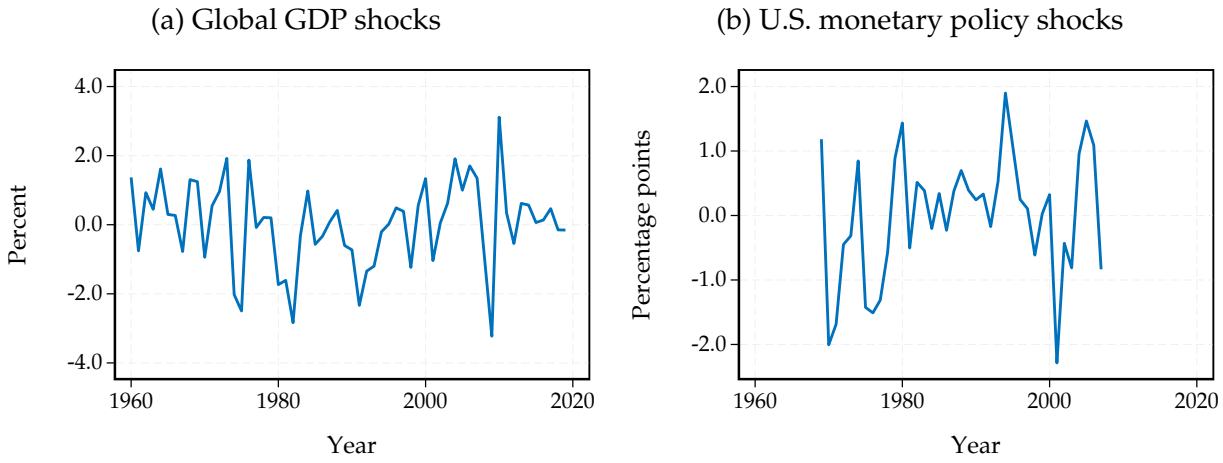
Figure 2(a) plots the resulting global GDP shocks over our sample. The series fluctuates around zero and captures short-run deviations from trend growth. Most shocks are moderate in magnitude, typically within $\pm 2\%$, though several larger contractions are visible, including during the mid-1970s, early 1980s, and the Global Financial Crisis. Positive and negative shocks are roughly balanced over time, and there is no visible low-frequency drift.

2.3 Monetary Policy Surprises as an Instrument for Global GDP

While isolating global GDP shocks mitigates concerns about trending behavior, it does not by itself resolve the second concern of omitted variable bias. Although the GDP shock series in Panel (a) does not exhibit obvious co-movement with major mortality events, GDP innovations may still reflect underlying forces that directly influence mortality. To further mitigate these concerns, we also present results based on an instrumental-variable strategy.

Specifically, we instrument global GDP with exogenous U.S. monetary policy shocks.

Figure 2: Global Output and Mortality Since 1960



Notes: Panel (a): global GDP shocks for 1960-2019, constructed using the Hamilton (2018) filter with $h = 1$ and $p = 2$. Panel (b): U.S. monetary policy shocks from Romer and Romer (2004), aggregated to the annual frequency.

Monetary policy shocks in the United States propagate internationally through trade, financial linkages, capital flows, and exchange rate regimes, and are a key driver of the global financial cycle (Kim, 2001; Miranda-Agrippino and Rey, 2020; Camara et al., 2025). As a result, unexpected U.S. policy tightenings generate broad-based fluctuations in global economic activity, making them a powerful and relevant source of external variation in global GDP.

We measure monetary policy shocks using the narrative approach of Romer and Romer (2004). Following their procedure, we regress changes in the intended federal funds rate on the Federal Reserve's internal Greenbook forecasts and other information available at the time of the policy decision:

$$\Delta \text{ffr}_m = \alpha + \beta \text{ffr}_{m-1} + \boldsymbol{X}_m' \boldsymbol{\gamma} + \varepsilon_m^{RR} \tag{2}$$

where ffr_m is the FOMC's federal funds rate target, and $\boldsymbol{X}_m$ contains economic forecasts that the FOMC has at its disposal at the time of meeting m. ε_m^{RR} captures the unsystematic component of policy changes, orthogonal to the Fed's real-time assessment of macroeconomic conditions.[3] We aggregate these shocks to an annual frequency ε_t^{RR} by summing over all the FOMC meetings in a given year t.

We use these shocks ε_t^{RR} as an instrument for global GDP innovations. U.S. monetary

[3]We use the updated Romer-Romer shock measure by Miranda-Agrippino, spanning the period 1969-2007.

policy is not set in response to mortality developments, let alone mortality conditions outside the United States. Moreover, to the extent that mortality in the U.S. affects GDP or inflation, such effects are incorporated in the Federal Reserve's own forecasts formed a few days before FOMC meetings. Because the shocks are identified as deviations from those forecasts, they are orthogonal to the current macroeconomic outlook and therefore plausibly exogenous to mortality shocks at home and abroad.

Figure 2(b) shows the time series of identified U.S. monetary policy shocks, expressed in percentage points. The series fluctuates around zero and features several pronounced episodes. In the 1970s, sizeable negative shocks are followed by a large positive innovation in the early 1980s associated with the Volcker disinflation. The mid-1990s display another notable positive shock, driven by a sequence of unexpected monetary tightenings during the 1994–95 tightening cycle. Finally, a large negative shock appears in 2001 following the collapse of the dot-com bubble. Overall, the series exhibits sharp, episodic movements that provide meaningful and plausibly exogenous variation in U.S. monetary policy.

2.4 Empirical Specification

The mortality effects of income fluctuations may take time to materialize. Therefore, we estimate the dynamic effects of global GDP shocks up to 10 years following the initial shock. We do so using panel local projections (Jordà, 2005; Jordà et al., 2020), which allow us to estimate medium-run effects directly without extrapolating from short-term dynamics.[4]

For each horizon $h = 0, \ldots, 10$, we estimate the panel regression

$$m_{i,t+h} - m_{i,t-1} = \alpha_{i,h} + \theta_h z_t + \mathbf{x}_t' \boldsymbol{\beta}_h + \mathbf{x}_{i,t}' \boldsymbol{\gamma}_h + \varepsilon_{i,t+h}, \tag{3}$$

where $m_{i,t}$ denotes the mortality rate (deaths per 1,000 individuals) in country i and year t, and $z_t \in \{\widehat{y_t^{\text{shock}}}, \widehat{\varepsilon}_t^{RR}\}$ is either the global GDP shock or the U.S. monetary policy shock. The coefficient θ_h captures the effect of a global income shock on mortality at horizon h. When z_t is the global GDP shock, we interpret the impulse responses as dynamic correlations. When z_t is the U.S. monetary policy shock, the estimates admit a more credible causal

[4]We use local projections rather than VARs or distributed lag models because they tend to be more robust at longer horizons (Montiel Olea et al., 2024). Local projections estimate the effects of interest separately at each horizon and allow for flexible control specifications.

interpretation.

The country fixed effects $\alpha_{i,h}$ absorb time-invariant cross-country differences in mortality, such as structural health system characteristics or demographic composition. The vector $\mathbf{x}_t$ contains global controls, while $\mathbf{x}_{i,t}$ includes country-specific controls. The error term $\varepsilon_{i,t+h}$ captures residual mortality fluctuations.

Our global income shocks are common across countries. Consequently, year fixed effects would absorb the shock and cannot be included. Instead, we control flexibly for global confounders that may correlate with both economic activity and mortality.

In all specifications, we control for four lags of the mortality rate and two lags of the shock, global GDP growth and local GDP growth, the (log) U.S. real effective exchange rate, as well as subregion-specific linear trends and dummies for the Great Recession. In the monetary policy shock specification, we additionally control for two lags the 1-year treasury yield change and the spread between the 1 and the 10-year treasury yield. Regressions are weighted by country population in period $t-1$ so that larger countries contribute proportionally more to global mortality outcomes.

Because global GDP shocks are common across countries, residuals may exhibit serial and cross-sectional dependence. We therefore compute Driscoll and Kraay (1998) standard errors, which are robust to general forms of serial and cross-sectional dependence.

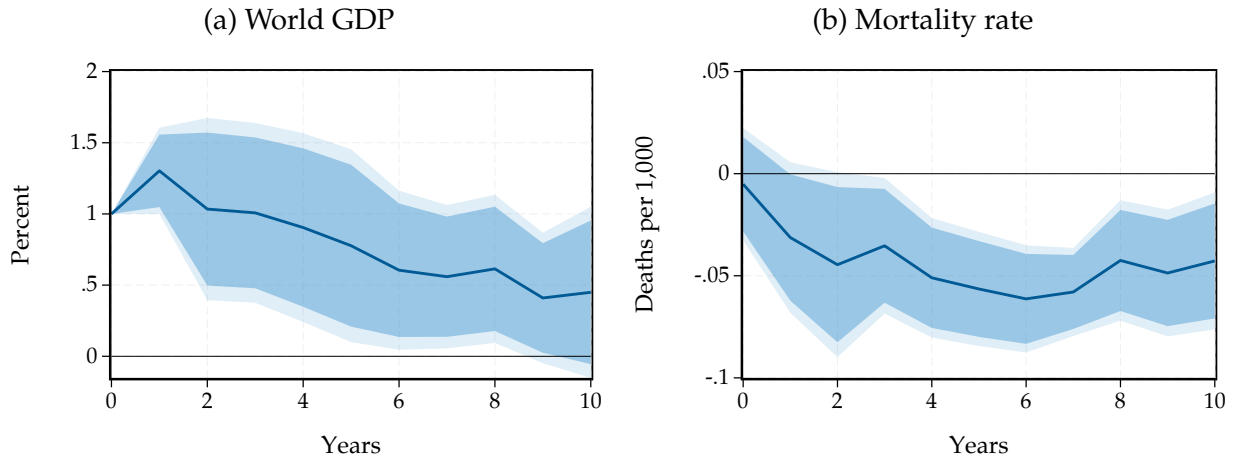
3 Global Growth and Mortality

We now present our main empirical results on the relationship between global income fluctuations and mortality. We begin by estimating the dynamic response of mortality to global GDP shocks, before turning to U.S. monetary policy shocks, which provide plausibly exogenous variation in global economic activity. For both approaches, we conduct a range of robustness checks to address potential confounding and specification concerns.

3.1 Mortality Responses to Global GDP Shocks

Figure 3 reports the dynamic responses of world real GDP per capita and mortality to a global GDP shock, normalized to increase world real GDP by 1% on impact. A positive GDP shock is associated with a substantial decline in mortality. On impact, mortality falls by around 0.02 deaths per 1,000 individuals, and the response builds gradually over the following years, reaching a peak reduction of more than 0.06 deaths per 1,000 six years

Figure 3: The Effect of Global GDP Shocks on GDP and Mortality



Notes: Impulse responses of global GDP per capita and mortality rate (deaths per 1,000 individuals) to a 1% GDP per capita shock, estimated using panel local projections in equation (3). Sample: 1960-2019. Controls: four lags of mortality rate; two lags of the shock, global GDP growth, local GDP growth, log U.S. real effective exchange rate; Great Recession indicator, and subregion-specific linear trends. Shaded areas: 90% and 95% confidence bands based on Driscoll-Kraay standard errors.

after the shock. The effect remains negative for several years and is statistically different from zero at the 95% confidence levels at horizons three through ten.

Importantly, global GDP shocks have a persistent effect on global output itself: world real GDP rises on impact and remains elevated for several years. Although the GDP shock is transitory, it produces a persistent increase in the level of GDP, indicating that temporary income innovations have lasting effects on economic activity. As a result, the mortality responses several years after the shock reflect the accumulated effects of persistently elevated global income rather than the impact of a purely temporary income fluctuation. In other words, the estimated responses capture the health consequences of medium-run income fluctuations. These effects are economically meaningful but smaller than the implied effect of a permanent change in the global income level.

To convert these estimates identified from partially transitory global GDP shocks into the effect of a permanent increase in global GDP, we compute the ratio of the cumulative impulse responses of mortality to the cumulative response of global GDP. Our estimates imply that a permanent rise in global GDP per capita scaled to 100% reduces mortality by approximately 6 deaths per 1,000 individuals in the long run.

Although global growth typically occurs through a sequence of small increments, this conversion relies on the assumption that the relationship between GDP and mortality extends beyond the range of GDP shocks observed in our sample, and that it applies not

only to medium-run unexpected income fluctuations but also to anticipated permanent changes in global GDP.

We assess the robustness of these results along several dimensions. Figure 4(a) shows that the mortality response is robust to the controls included. Specifically, we find very similar effects when we control for conflicts and epidemics as well as demographic factors. We also obtain similar results when excluding the real effective exchange rate, the Great Recession dummy, or the region-specific trends. Across specifications, there are also no statistically significant pre-trends, suggesting that the estimated responses are not driven by differential trends in mortality in the years preceding the income shock.

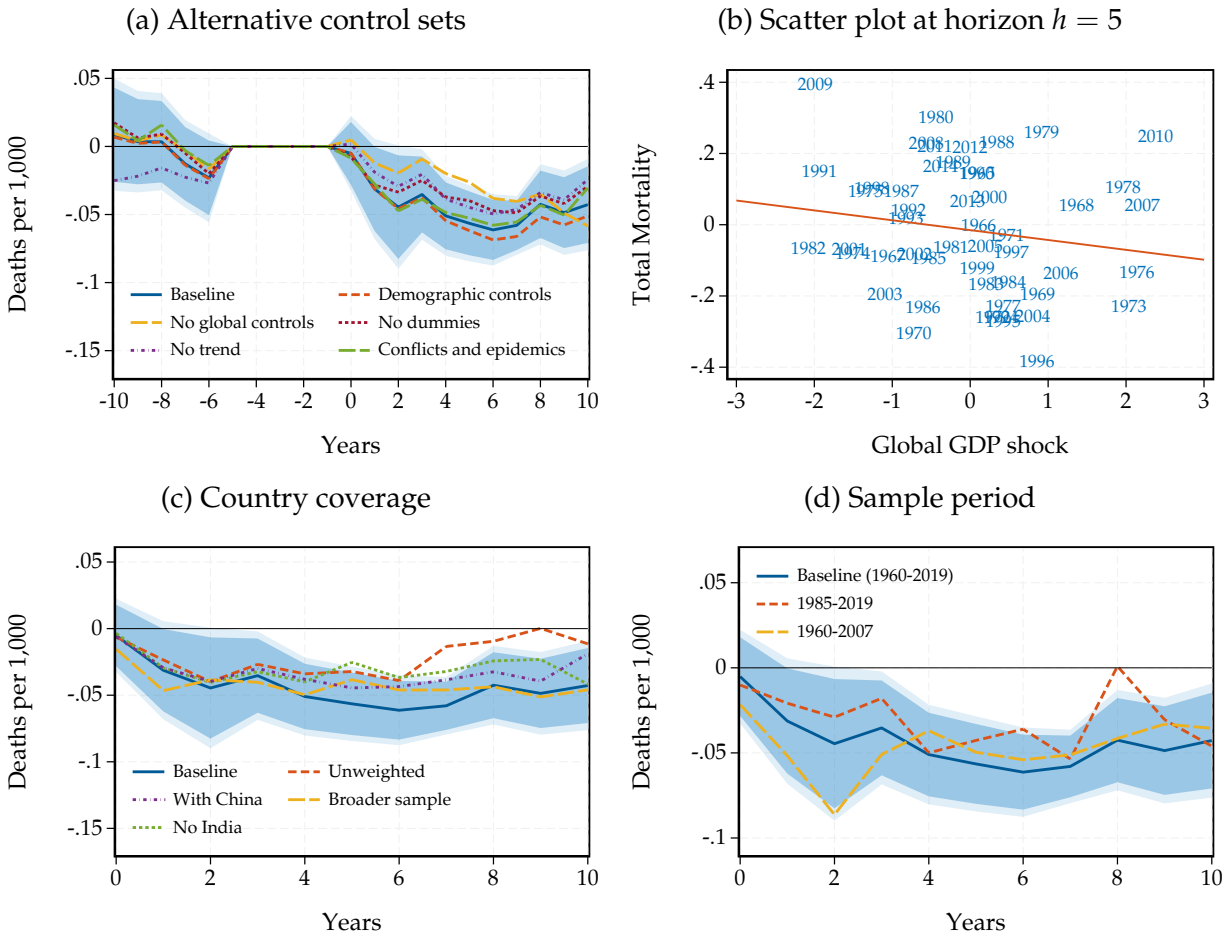
Panel (b) illustrates that the mortality response is not driven by a small number of outliers by highlighting the identifying variation in a scatter plot. We show the global GDP shock against the cumulative change in mortality five years out, corresponding to the midpoint of the horizons we consider and close to the peak effect, both residualized from our set of global and local controls. The negative relationship appears to be driven by all years in the sample rather than by a restricted set of outliers.

Panel (c) indicates that the mortality response is not driven by a particular set of countries. We compare the impulse response function after excluding India or including China and find similar effects. We also include countries in Africa covered by the UNWPP data and find comparable estimates. The unweighted regression—assigning equal weight to large and small countries—uncovers estimates in the same range but somewhat less persistent, suggesting that populous countries partly determine the persistence of our results.

Panel (d) demonstrates that the results continue to hold in shorter time periods: in the early (1960–1989) and later (1990–2019) sub-samples, although the impulse response functions are necessarily noisier in these highly demanding specifications with less than 40 years of data.

In Appendix B, we further perform a broad range of robustness exercises. Figure B.1(a) reports the results from a jackknife exercise in which we sequentially exclude each year from the sample. The resulting impulse responses are robust to the specific shocks included in the estimation. Figure B.1(b) demonstrates that the results are robust to alternative detrending approaches, including using a one-sided Hodrick-Prescott filter, simple first differences, or estimating the effect of interest directly in a single local projection without first isolating the identifying GDP innovations. Overall, these exercises point to a strong, robust negative association between global income fluctuations and mortality.

Figure 4: Sensitivity to Control Variables and Specification Choices



Notes: Sensitivity to controls, sample and other specification choices. Panel (a): impulse responses under alternative control specifications. Baseline: four lags of mortality rate; two lags of the shock, global GDP growth, local GDP growth, log U.S. real effective exchange rate; Great Recession indicator, and subregion-specific linear trends. Demographic controls: share of individuals over 65 years of age. No global controls: excludes global GDP growth and log U.S. real effective exchange rate. No dummies: excludes Recession indicator. No trend: excludes linear trends. Conflicts and epidemics: controls for indicators of the onset year of major epidemics and the number of conflicts a country is involved in in a given year. Panel (b): scatter plot of global GDP shocks against the cumulative change in total mortality 5 years out, both after residualizing our set of controls and averaging across countries. Panel (c): responses under alternative selections of countries and weighting schemes. Panel (d): responses based on alternative sample periods.

Global GDP per capita fluctuates for many reasons that are plausibly unrelated to mortality shocks, including unexpectedly rapid technological progress, fiscal policy, financial crises, or political events that affect economic activity across countries. As a result, the dynamic relationship between global GDP fluctuations and mortality is informative about the underlying connection between income and longevity.

At the same time, potential omitted variables could lead global GDP and mortality to co-move. For example, large epidemics or global conflicts may simultaneously depress economic activity and increase mortality. In addition, positive health improvements may

lead to reverse causality if healthier individuals are more productive. We mitigate these concerns by controlling for such confounding factors to the extent possible in the data. To pose a threat to identification, qualitatively, omitted variables have to be large enough to affect worldwide economic activity at the same time as affecting mortality throughout the world. Similarly, reverse causality would have to operate at the high frequencies that we consider—of course a possibility, if not a particularly likely one. In both cases, to be quantitatively meaningful, these shocks have to dominate other sources of global GDP fluctuations. To strengthen the causal interpretation of our findings, we next exploit U.S. monetary policy shocks as an external source of variation in global economic activity.

3.2 Mortality Responses to U.S. Monetary Policy Shocks

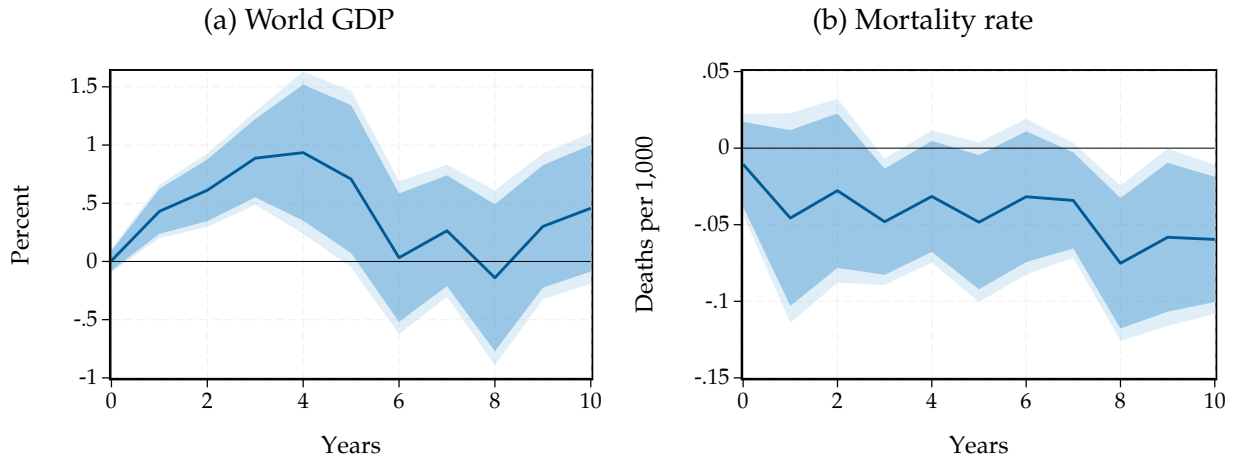
To identify the causal effect of global GDP on mortality, we need to isolate variation in global GDP that is unrelated to mortality shocks or other confounding factors that simultaneously affect economic activity and health outcomes. To address this concern, we exploit U.S. monetary policy surprises as an external source of variation in global economic activity.

Monetary policy shocks in the United States propagate internationally through trade linkages, global financial markets, and exchange rate regimes (Kim, 2001; Miranda-Agrippino and Rey, 2020; Camara et al., 2025). Moreover, the Federal Reserve sets policy to stabilize U.S. economic conditions, making it unlikely that these surprises respond directly to mortality developments or other shocks affecting economic activity outside the United States. Thus, U.S. monetary policy surprises are a promising instrument for global GDP fluctuations.

Figure 5 reports the responses of global GDP per capita and mortality to U.S. monetary policy shocks. Panel (a) shows that expansionary monetary policy surprises in the United States increase GDP per capita around the world. This first-stage relationship is statistically significant and consistent with the well-established international transmission of U.S. monetary policy. The strength of the response indicates that the instrument generates meaningful variation in global economic activity.

Panel (b) shows that these shocks also affect mortality. An expansionary monetary policy surprise that increases global GDP by roughly one percent at its peak leads to a fall in mortality of approximately 0.05 deaths per 1,000 individuals after three years. The effect is statistically significant at conventional confidence levels at horizons three

Figure 5: The Effect of Global Monetary Policy Shocks on GDP and Mortality



Notes: Impulse responses of GDP per capita and mortality rate (deaths per 1,000 individuals) to a 1 percentage point expansionary monetary policy shock estimated using panel local projections in equation (3). Sample: 1969-2007. Controls: four lags of mortality rate; two lags of the shock, global GDP growth, local GDP growth, log U.S. real effective exchange rate, change in one-year U.S. treasury yield, spread between one and 10 year U.S. treasury yields; Great Recession indicator, and subregion-specific linear trends. Shaded areas: 90% and 95% confidence bands based on Driscoll-Kraay standard errors.

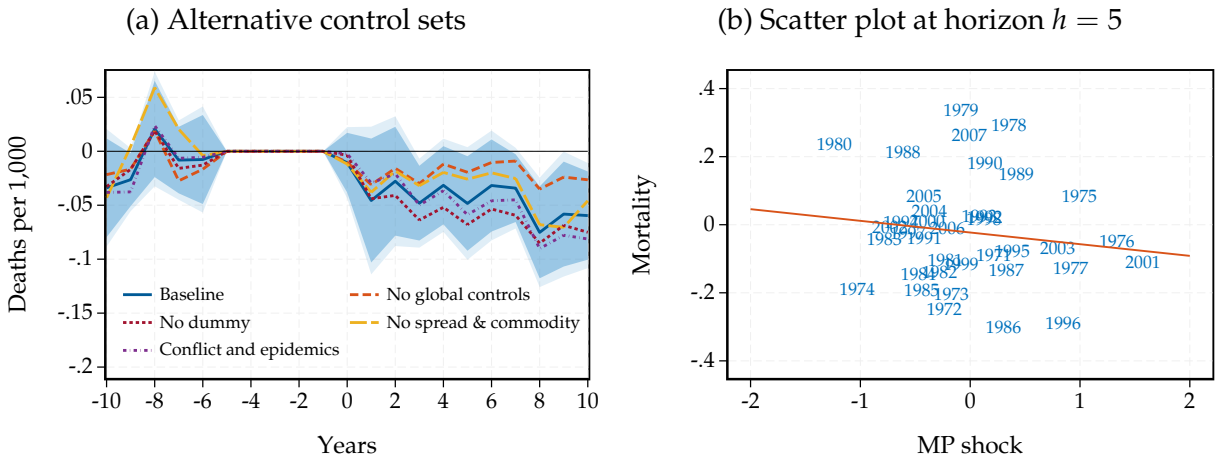
and eight through ten. While these estimates are less precise than those in Section 3.1, estimating a pooled effect across all horizons following the monetary policy shocks leads to an estimate that is statistically significant at the 95% level.

Because monetary policy shocks have only partially persistent effects on global GDP, the estimated responses reflect the mortality consequences of a persistent but transitory income fluctuation. To translate these estimates into the effect of a permanent increase in global GDP, we take the ratio of the cumulative impulse responses of mortality and global GDP to the monetary policy shock. Combining the first-stage and reduced-form responses allows us to recover the causal, two-stage least squares effect of global income on mortality.

Our results imply that a permanent increase in global GDP per capita scaled to 100% reduces mortality by 8 deaths per 1,000 individuals in the long run. While this point estimate is somewhat larger than the corresponding estimate based on global GDP shocks, the two magnitudes are quantitatively close and are not statistically distinguishable from each other at the 95% level.

As for global GDP shocks, we assess the robustness of our results based on U.S. monetary policy shocks. Figure 6(a) shows that the mortality response is robust to alternative control specifications. Dropping global controls, local controls, or financial controls one at a time does not materially affect the estimates. As in the baseline specification, we

Figure 6: Sensitivity to Control Variables and Specification Choices



Notes: Panel (a): impulse responses under alternative control specifications. Baseline: four lags of mortality rate; two lags of the shock, global GDP growth, local GDP growth, log U.S. real effective exchange rate, change in one-year U.S. treasury yield, spread between one and 10 year U.S. treasury yields; Great Recession indicator, and subregion-specific linear trends. No global controls: excludes global GDP growth and log U.S. real effective exchange rate. No dummies: excludes Recession indicator. No spread & commodity: excludes U.S. treasury spread and commodity price inflation. Conflicts and epidemics: controls for indicators of the onset year of major epidemics and the number of conflicts a country is involved in in a given year. Panel (b): scatter plot of monetary policy shocks against the cumulative change in total mortality 3 years out, both after residualizing our set of controls and averaging across countries.

also do not detect any statistically significant pre-trends, suggesting that the estimated responses are not driven by differential mortality dynamics prior to the monetary policy shock. Panel (b) again illustrates the identifying variation in a scatter plot. We plot monetary policy shocks against the cumulative change in mortality three years out, corresponding to the peak response, both residualized from the set of controls and averaged across countries. The positive relationship is not driven by a small number of influential observations—with the possible exception of the 2001 monetary shock which, if anything, attenuates our results—indicating that the results reflect broad variation in the data rather than a few outliers.

4 Global and Local Growth

4.1 Global GDP or Local GDP?

Our estimated impact of global GDP shocks on mortality differs markedly from most estimates in the existing literature. Early work by Preston (1975) and subsequent studies exploiting country-level variation in income—including Summers and Pritchett (1996), Ruhm (2000), Ruhm (2003), and Ruhm (2007), Baird et al. (2011), Cutler et al. (2016), Ma

et al. (2022), and Finkelstein et al. (2025)—typically find more modest effects of income on mortality, and in some cases even negative effects. These studies exploit fluctuations in country-level or even more granular economic activity variables, whereas our baseline estimates rely on variation in global income. This difference in the source of variation may account for the substantially larger effects we estimate.

We investigate whether the change in the explanatory variable accounts for the difference in impacts holding the sample and the econometric specification fixed. We estimate the effects of local, country-level GDP shocks as opposed to global GDP shocks. Specifically, for each horizon $h = 0, \ldots, 10$, we estimate the panel regression

$$m_{i,t+h} - m_{i,t-1} = \alpha_{i,h} + \delta_{t,h} + \theta_h \widehat{y_{i,t}^{\text{shock}}} + \mathbf{x}_{i,t}' \gamma_h + \varepsilon_{i,t+h}, \tag{4}$$

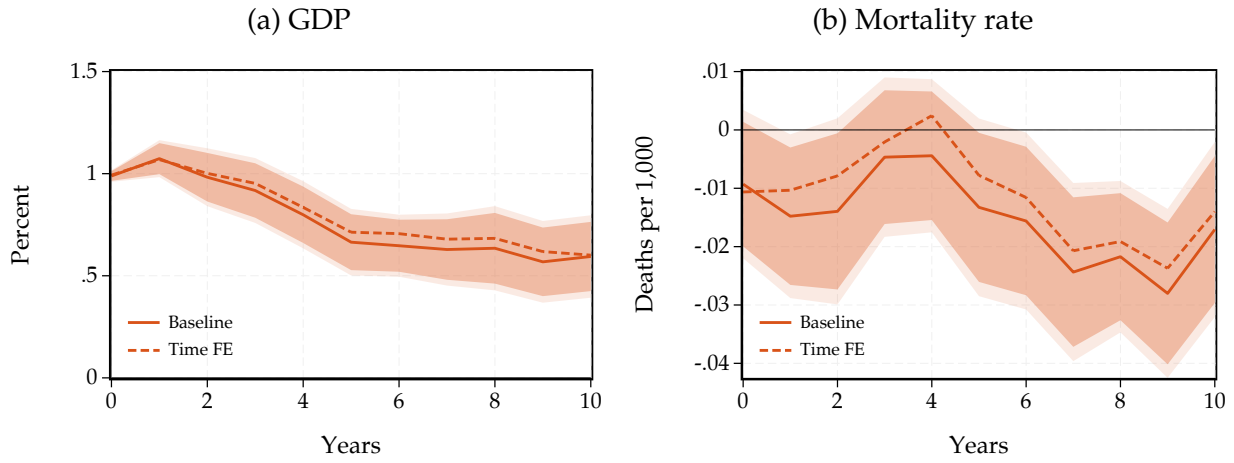
where $\widehat{y_{i,t}^{\text{shock}}}$ denotes the local GDP shock in country i and year t, constructed using the Hamilton filter, analogous to our construction of global GDP shocks. Relative to our baseline specification, this approach exploits within-country variation in economic activity rather than common global fluctuations. Because the identifying variation now varies across countries within a given year, we can additionally include year fixed effects $\delta_{t,h}$ to absorb global shocks that affect mortality across all countries simultaneously. All other controls and estimation choices remain identical to the baseline specification.

Figure 7 reports the resulting impulse responses. Panel (a) shows that a local GDP shock generates a persistent increase in country-level GDP per capita, similar in magnitude to the global GDP responses reported earlier. Panel (b) shows that local GDP expansions are associated with reductions in mortality, but the magnitude of the effect is noticeably smaller than for global GDP shocks. The mortality response is gradual and remains modest even at longer horizons.

Quantitatively, the implied relationship between income and mortality is smaller than in our baseline results. Our estimates imply that doubling local GDP reduces mortality by 2 deaths per 1,000 individuals in the long run, three times smaller than the effect implied by global GDP shocks. While the effect is statistically significant, the estimated responses also display some evidence of pre-trends despite controlling for multiple lags of mortality. Overall, these estimates are broadly consistent with the existing literature, which typically finds relatively modest effects of domestic income fluctuations on mortality.

To directly contrast the effect of global and local GDP, we estimate a specification that simultaneously includes both global and local GDP shocks. Figure 8 displays the re-

Figure 7: The Effect of Local GDP Shocks on GDP and Mortality



Notes: Impulse responses of local GDP per capita and mortality rate (deaths per 1,000 individuals) to a 1% local GDP per capita shock, estimated using panel local projections in equation (4). Sample: 1960-2019. Controls: four lags of mortality rate; two lags of the shock, global GDP growth, local GDP growth, log U.S. real effective exchange rate; Great Recession indicator, and subregion-specific linear trends. Shaded areas: 90% and 95% confidence bands based on Driscoll-Kraay standard errors.

sults. The estimated response to global GDP shocks remains virtually identical to our baseline specification, indicating that the global effects we document are not driven by omitted local economic fluctuations. By contrast, the responses to local GDP shocks become, if anything, somewhat less pronounced when both shocks are included jointly. This pattern underscores the distinct role of global economic conditions in shaping mortality outcomes.

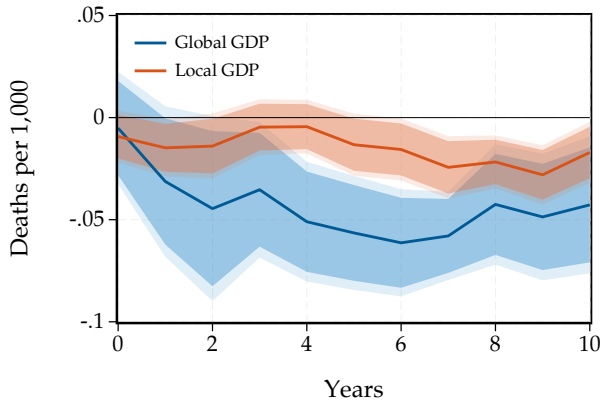
Overall, the results consistently point to a substantially stronger mortality response to global GDP shocks than to local GDP shocks. While fluctuations in domestic economic activity appear to have limited effects on mortality, changes in global economic conditions—driven primarily by movements at the economic frontier—are associated with sizable and persistent reductions in mortality rates worldwide. These results suggest that the mechanisms linking economic activity to health outcomes operate largely through global channels rather than through country-specific fluctuations.

4.2 Mechanisms

Why do global GDP expansions appear to reduce mortality more strongly than local GDP expansions? Growth at the global economic frontier appears to generate spillovers that improve health outcomes in the rest of the world.

Qualitatively, these longevity spillovers are consistent with two broad mechanisms.

Figure 8: Local vs. Global GDP Shocks



Notes: Impulse responses of local vs. global GDP shocks on mortality rate (deaths per 1,000 individuals), estimated jointly based on the same local projection specification. Sample: 1960-2019. Controls: four lags of mortality rate, growth rate of local GDP, USD real effective exchange rate, growth of US exports; two lags of global GDP shock, great recession indicator, dummy for China after 2005, and subregion-specific linear trends. Shaded areas: 90% and 95% confidence bands based on Driscoll-Kraay standard errors.

First, they are consistent with the influential line of work that finds beneficial health effects of public health interventions in lower-income regions (Miguel and Kremer, 2004; Bendavid et al., 2012; Pryce et al., 2018), often partly funded by institutions in higher-income regions such as the World Bank, the World Health Organization, the Global Fund to Fight AIDS, Tuberculosis and Malaria, non-governmental organizations, USAID or PEPFAR (Schieber et al., 2006; Hanson et al., 2022). Our analysis suggests that economic growth at the frontier may enable these interventions to take place.

Second, longevity spillovers are also qualitatively consistent with international price reductions that affect mortality. Pharmaceutical prices are one candidate. Food price reductions that may improve nutrition are another candidate. To operate through global rather than local GDP, these international price reductions would have to disproportionally scale with global GDP changes.

Finally, longevity spillovers are in principle also consistent with international diffusion of medical knowledge fueled by growth at the frontier. However, the long time lags in medical research are difficult to reconcile with our high frequency analysis.

We investigate these mechanisms empirically, although we do not find conclusive evidence in favor of any. First, we investigate whether foreign aid may be responsible for global longevity spillovers. We obtain data on foreign aid from the U.S. and approved by the federal government. This data has the advantage of being harmonized across possible destinations but is unlikely to be representative of all foreign aid. While we find an

association between global expansions and foreign aid flows, as well as between foreign aid flows and mortality, the combination of both accounts for no more than a tenth of our direct estimate, perhaps because of the limited coverage of our data. We plan to add figures describing these results in future iterations of this draft.

Second, we consider whether pharmaceutical price changes and trade may account for longevity spillovers. We find limited evidence of price and quantity responses to global GDP shocks, as well as a limited association between these international variables and mortality. The combination results in accounting for a negligible fraction of our direct estimate.

International food prices tend to increase more than one-for-one in global expansions rather than fall. As a result, they do not appear to be a plausible candidate to account for longevity spillovers.

Taken together, these findings leave the mechanisms behind the strong relationship between global GDP growth and mortality largely unresolved. While data limitations prevent us from reaching definitive conclusions, the absence of a clearly identified mechanism constitutes an important avenue for future research. Nevertheless, we now explore the macroeconomic implications of the global longevity spillovers of growth suggested by our results.

5 A Model of Global Wealth and Global Health

Our framework closely follows the standard neoclassical growth model. We augment it in two ways. First, we introduce internal health improvements that are produced similarly to final goods and can be purchased by households in a health service market, which reduces mortality. Second, we allow for external health improvements that directly reduce mortality but are not produced or purchased in markets.

5.1 Setup

Preferences. Time is continuous and runs forever. There is a unit continuum of infinitely-lived identical households who populate the world economy. Households have constant relative risk aversion flow preferences over a consumption good which we use as the numeraire: $U(C) = \frac{C^{1-\gamma}-1}{1-\gamma}$. Labor supply is exogenous and set to $L_t = 1$. The pure rate of time preference of households is ρ.

Households face stochastic lifespans τ as in Blanchard (1985). When they die, they receive a terminal value reduction $\overline{V}$ and give rise to a single offspring with respect to which they are fully altruistic. Households thus have the following dynamic preferences:[5]

$$V_0(K_0) = \mathbb{E}_0 \left[\int_0^\tau e^{-\rho t} U(C_t) dt + e^{-\rho \tau} \left(V(K_\tau) - \overline{V} \right) \right].$$

Lifespans end stochastically according to a Poisson process with time-varying intensity M_t.

Final good production. Final good firms produce the final consumption good with a Cobb-Douglas production function in capital K_t and labor L_t with time-dependent Total Factor Productivity (TFP) Z_t: $Y_t = Z_t K_t^\alpha L_t^{1-\alpha}$. They hire labor at wage w_t and rent capital from households at net interest rate r_t in competitive factor markets. Capital depreciates at rate δ, which is constant over time and covered by firms. The path of productivity Z_t is perfectly foreseen.

Health and mortality. Households can spend final goods on health. We denote their health spending by H_t. Healthcare purchases reduce mortality. Mortality reductions also occur externally to healthcare expenditures due to income growth. The mortality rate M_t follows:

$$M_t = \overline{M} H_t^{-\mu} Y_t^{-\nu}.$$

The parameter μ governs the effect of healthcare expenditures on mortality, while ν governs the effect of aggregate income on mortality.

Household budget constraint. Firms make zero profits given constant returns to scale, so we omit profits in the budget constraint of the household, which writes: $C_t + H_t + \dot{K}_t = w_t + r_t K_t$. Households are endowed with an initial capital stock K_0.

Equilibrium. A competitive equilibrium of our economy is a collection of sequences $\{C_t, K_t, r_t, w_t\}_{t=0}^\infty$ such that households optimize given prices and aggregate output

[5] We use this dynastic formulation rather than annuities to focus on efficiency properties of investment due to healthcare improvements and avoid other sources of dynamic inefficiency. The consequence is that disutility from death $\overline{V}$ must be introduced as a one-time cost rather than as a flow shift in the utility function as in Hall and Jones (2007) and Jones and Klenow (2016), in which case it would become neutral.

$\{r_t, w_t, Y_t\}_{t=0}^{\infty}$:

$$\max_{\{C_t, K_t, H_t\}_t} \mathbb{E}_0 \left[\int_0^{\tau} e^{-\rho t} U(C_t) dt + e^{-\rho \tau} \left(V(K_\tau) - \overline{V} \right) \right],$$

subject to $C_t + H_t + \dot{K}_t = w_t + r_t K_t$, to $M_t = \overline{M} H_t^{-\mu} Y_t^{-\nu}$ and given K_0. Firms optimize given prices $\{r_t, w_t\}_t$:

$$\max_{K_t^D, L_t^D} Z_t (K_t^D)^{\alpha} (L_t^D)^{1-\alpha} - (r_t + \delta) K_t^D - w_t L_t^D.$$

Factor markets clear: $K_t = K_t^D$ and $1 = L_t^D$.

Efficient allocation. The efficient allocation maximizes the same objective subject to the resource constraint $C_t + H_t + \dot{K}_t = Y_t - \delta K_t$ taking into account that mortality M_t also depends on aggregate output Y_t.

Efficiency. We show in Appendix C that the decentralized equilibrium is inefficient for $\nu \neq 0$. Households do not internalize the health spillovers of income growth. We highlight this property with the Euler equation:

$$\gamma \frac{\dot{C}_t}{C_t} = \alpha Z_t K_t^{\alpha - 1} - \rho - \delta + \mathbf{I}^{\text{SP}} \alpha \nu \frac{M_t}{K_t u'(C_t)} \overline{V},$$

where $\mathbf{I}^{\text{SP}} = 1$ in the efficient allocation, and zero in the decentralized equilibrium.

Health spending and mortality. In the decentralized equilibrium and in the efficient allocation, health spending is: $H_t = \overline{H} C_t^{\frac{\gamma}{1+\mu}} Y_t^{-\frac{\nu}{1+\mu}}$ where $\overline{H}$ is a combination of parameters given in Appendix C. Mortality is: $M_t = \widetilde{M} C_t^{-\frac{\mu\gamma}{1+\mu}} Y_t^{-\frac{\nu}{1+\mu}}$ where $\widetilde{M}$ is a combination of parameters given in Appendix C.

Multi-country interpretation. We map our model to our empirical results as follows. We assume that there is a unit continuum of ex-ante identical countries indexed by i that start in steady-state and do not interact in factor markets. The only interaction between countries is that the aggregate health spillover depends on global aggregate output $Y_t = \int Y_{it} di$. Mortality then depends on country-specific health consumption and the aggregate health spillover: $M_{it} = \overline{M} H_{it}^{-\mu} Y_t^{-\nu}$. Since all countries are symmetric, this model collapses to the single-country framework in which the global health spillover is not internalized.

5.2 Estimation

We set a first group of parameters to standard values. The rate of time preference is $\rho = 0.02$. The intertemporal elasticity of substitution is $1/\gamma = 1$. The capital share is $\alpha = 0.3$, and the capital depreciation rate is $\delta = 0.08$.

We estimate the parameters governing mortality internally: $\overline{V}, \mu, \nu$. To estimate these parameters, we interpret our model through the lens of the decentralized equilibrium of the multi-country extension described in Section 5.1. We leverage the equilibrium relationship between mortality M_{it}, consumption C_{it} and aggregate output Y_t: $M_{it} = \widetilde{M} C_{it}^{-\frac{\mu\gamma}{1+\mu}} Y_t^{-\frac{\nu}{1+\mu}}$, where i indexes countries.

We estimate the elasticity of mortality to private health spending μ by matching the estimated elasticity of mortality to local GDP per capita. If we measured directly the long-run elasticity of mortality to health spending $\theta_{M,H}$, we could directly set μ such that: $-\frac{\mu\gamma}{1+\mu} = \theta_{M,H}$.

We face two additional practical challenges. First, we measure the elasticity of mortality to local GDP Y_{it}—which we interpret to be driven by idiosyncratic shocks in local TFP Z_{it}. We therefore use indirect inference and search for the elasticity μ that lets us match the empirical elasticity of mortality to local output in the model to the data. Second, we estimate an entire impulse response function. Thus, we target the ratio of the cumulative impulse response function, which corresponds to the long-run elasticity.

We estimate the elasticity of mortality to aggregate output ν by matching the empirical elasticity of mortality to global GDP. Denote by $\theta_{M,Y}$ the empirical long-run elasticity of mortality to global GDP, that is, the ratio of cumulative impulse responses. We then set ν such that: $\frac{\nu}{1+\mu} = -\theta_{M,Y}$.

We set the shifter of mortality to target an average global life expectancy of 52 years in 1960. Consistent with our empirical analysis. Life expectancy at birth in 2019 is 72 years.

To estimate the end-of-life disutility $\overline{V}$, we leverage that the empirical VSL satisfies $\overline{V} = u'(C)\text{VSL}$ in the model. Logarithmic utility has the advantage that this ratio is scale-independent. We thus set: $\overline{V} = \frac{\text{VSL}}{C}$ using a VSL value and annual consumption data from the United States. Scale-independence implies that the same utility shifter $\overline{V}$ applies independently from country income. Conversely, our estimating relationship implies a unit elasticity of the VSL with respect to country income.

5.3 Global Longevity Spillovers In Perspective

Our first exercise assesses the role of global longevity spillovers in accounting for life expectancy improvements since 1960. To do so, we initialize the economy in 1960. Then, we input a change in TFP that leads aggregate output to increase nearly threefold by 2019 in the decentralized equilibrium as in the data. We then compare the evolution of the economy in the decentralized equilibrium with and without the global health spillover. Figure 9 displays the results.

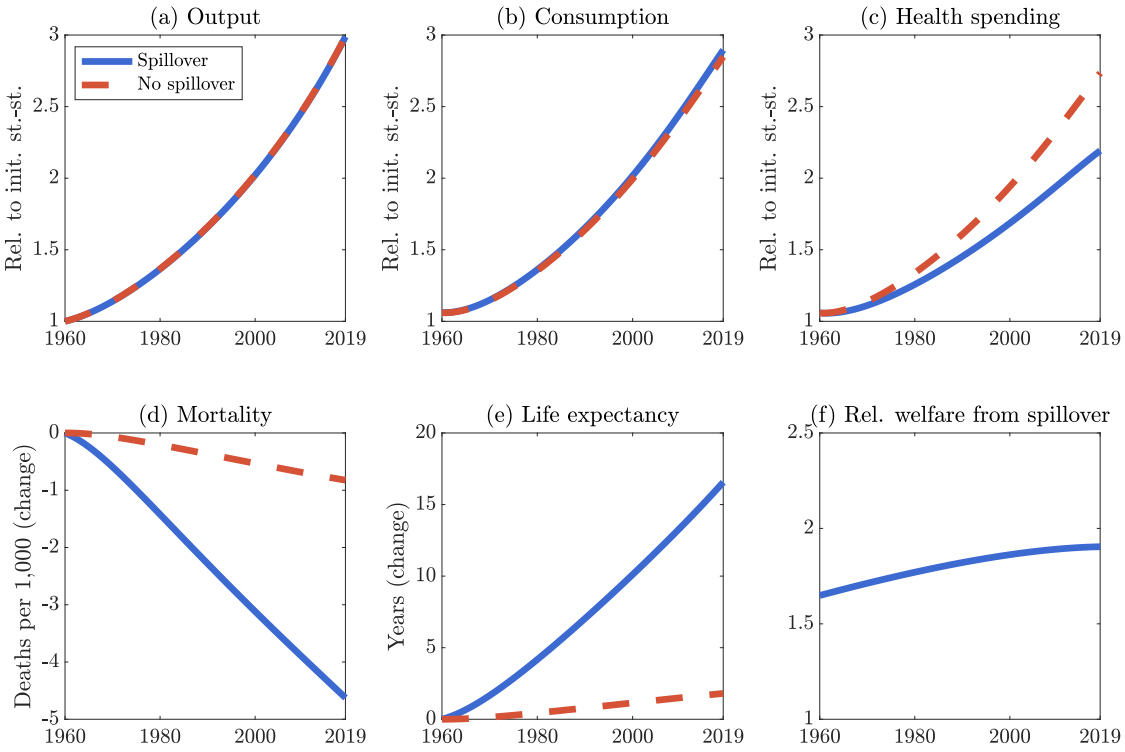
Panel (a) show that the path of output is largely unaffected by the presence of the spillover. Panels (b) and (c) indicate why. Without the global longevity spillover, health spending rises faster to compensate the lack of longevity improvements. However, health spending is a relatively small portion of overall expenditures. Thus, consumption and hence savings, capital and output remain largely unchanged.

Panel (d) reveals that mortality falls substantially less without the global longevity spillover despite substitution toward health spending. The decline stands at 0.8 death per 1,000 individuals, as opposed to 4.6 deaths per 1,000 with global spillovers.

Changes in mortality rates have substantial effects on life expectancy. Panel (e) shows that life expectancy rises by less than 2 years between 1960 and 2019 without the global longevity spillover, accounting for 9% of the increase in the data. With the spillover, the increase in life expectancy reaches 16 years, 81% of the increase in the data. The spillover thus accounts for 72% of the life expectancy increase in the data.

The differences in life expectancy trajectories have stark implications for welfare. Panel (f) shows that welfare gains from economic growth are between 1.5 times and twice as large with the global longevity spillover, due to life expectancy improvements. These relative welfare gains represent the consumption-equivalent welfare change relative to 1960 with the spillover, relative to welfare gains without the spillover. They are comparable to the magnitude of welfare gains associated with historical life expectancy improvements (Becker et al., 2005; Murphy and Topel, 2006; Hall and Jones, 2007; Jones and Klenow, 2016) because global longevity spillovers account for the majority of these historical changes. Importantly, the reference value incorporates the moderate life expectancy gains from private health spending.

Figure 9: The Impact of Global Longevity Spillovers, 1960-2019



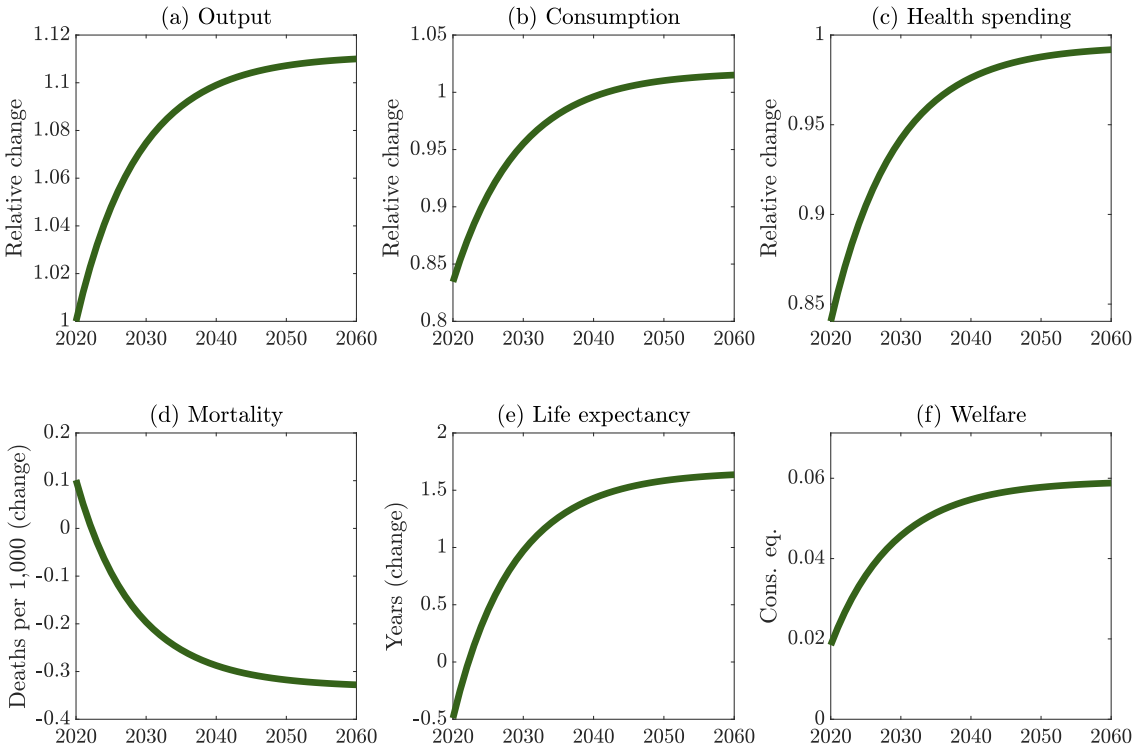
5.4 Harnessing Global Longevity Spillovers

Given the effect of global longevity spillovers on life expectancy and welfare across decentralized equilibria, our second exercise evaluates how much the efficient allocation that internalizes the spillover can improve on the decentralized equilibrium. We initialize the economy in the 2019 steady-state, and switch to efficient decisions in 2020. Figure 10 displays the resulting transitional dynamics.

Output rises gradually by up to 11%. This transition reflects increased incentives to save in productive capital once global longevity spillovers are internalized. Despite the increase in output, consumption remains below the decentralized steady-state value for over 20 years: the efficient decision is to save more to increase life expectancy instead. Private health spending also falls are remains below the initial decentralized equilibrium value, as the implied reduction in life expectancy is more than compensated by internalized spillovers.

The net effect is to lower mortality by just over 0.3 death per 1,000 individuals, leading

Figure 10: Harnessing Global Longevity Spillovers



to a life expectancy improvement of just under 2 years. Consequently, forward-looking welfare gains initially jump by over 2%, before rising by 6% in the long run.

6 Conclusion

This paper documents that global economic growth may be the main reason why life expectancy has risen over the last sixty years. Our analysis focuses on the impact of expansions at the economic frontier. To do so, we exploit time-series variation in global GDP. Our estimates imply that doubling global GDP avoids 6 deaths per 1,000 individuals. These estimates are three times larger than the effect of innovations to local, country-specific growth, that have been the focus of the literature since Preston (1975). While we control for as many confounders as possible, we further instrument global GDP with monetary policy shocks in the United States and find similar results.

When we embed these global longevity spillovers in a standard growth model with fi-

nite lifespans and health production, we find that these uninternalized spillovers account for 72% of life expectancy improvements between 1960 and 2019. Internalizing spillovers further increases aggregate output by 11% and life expectancy by 2 years. While our results must necessarily be interpreted with caution given the limited amount of identifying variation in the data, they suggest that global longevity spillovers have important implications for growth, life expectancy and welfare around the world.

Important questions remain unanswered by our analysis. Through which channels do global health spillovers operate? Are internationally funded public health interventions the main driver? International pharmaceutical or food prices? Medical knowledge diffusion? All these mechanisms likely play a role, but disentangling their respective importance is challenging. The limited nature of our data does not allow us to propose definitive answers. We hope that future research will.

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Online Appendix

Life Expectancy and Economic Growth

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A Data

A.1 Mortality Data

We construct our mortality variables by combining data from several sources, with the Human Mortality Database (HMD, 2024) serving as the primary input. The HMD is widely regarded as the gold standard for mortality data. It is based on high-quality vital registration data, complemented by census counts, birth records, and population estimates, and applies a harmonized methodology across countries. Crucially for our purposes, it relies on observed annual death counts rather than model-based imputation across years, making it well suited to high-frequency analysis that exploits year-to-year variation. The HMD covers most of Western and Northern Europe, North America, Australia, and a number of Eastern European and Asian countries. We download mortality counts `deaths.zip` and population and live-birth data, `population.zip` and `births.zip`, from `www.mortality.org/Data/ZippedDataFiles`. For each age group—total, ages 0–4, ages 5–64, and ages 65 and above—we divide death counts by population and multiply by 1,000 to obtain mortality rates per 1,000 individuals. This normalization is applied consistently across all data sources to ensure comparability.

Because HMD coverage is largely confined to high-income countries, we supplement the HMD series with data from the World Health Organization (WHO, 2024) Mortality Database to expand coverage and fill gaps. The WHO database compiles death counts reported by national statistical authorities from civil registration systems, with data disaggregated by age, sex, and cause of death and subject to varying degrees of completeness across countries and over time. Relative to the HMD, it provides broader geographic coverage, extending to Latin America and the Caribbean, the Middle East, parts of Asia, Eastern Europe, and Africa. We download mortality counts and population data from `www.who.int/data/data-collection-tools/who-mortality-database`. For countries where the entire HMD series is missing, we use the corresponding WHO mortality counts to construct our measures. For countries with partial gaps, we preserve the level of the HMD series and use the growth rates implied by the WHO data to interpolate and extrapolate missing years.

Despite the expanded coverage, two crucial countries remain missing from the baseline sources: India and China. For these countries, we therefore draw on additional sources. We obtain mortality rates for China directly from the National Bureau of Statis-

tics of China (https://data.stats.gov.cn/english/easyquery.htm?cn=C01), which reports annual crude death rates based on official statistics. For India, we obtain mortality counts from Vital Statistics India (VSI) and combine them with population data from the United Nations World Population Prospects. Starting from 2009, VSI reports are available at https://censusindia.gov.in/census.website/data/VSREPORT. For earlier years we digitize this data from University library sources. Finally, we add mortality rates for Liechtenstein from Carleton et al. (2022). Together, these sources yield a mortality panel with coverage of around 60 countries in 1960, rising to over 100 countries in the second half of our sample period.

Despite this broad coverage, reliable annual mortality data remain unavailable for many countries, particularly in Africa, where civil registration systems are often incomplete or absent. For these countries, we rely on estimates from the United Nations and Division World Population Prospects (UNWPP, 2024), accessed from population.un.org/wpp/. Unlike the HMD and WHO databases, which are based on directly observed deaths from civil registration systems, the UNWPP constructs internally consistent demographic series by combining a wide range of available sources—including population censuses, household surveys, partial registration systems, and international datasets—and applying demographic models to reconcile discrepancies and interpolate missing observations. In particular, the estimates are derived using a cohort-component framework that ensures consistency between population levels and the underlying components of change, namely fertility, mortality, and migration. While this approach provides comprehensive global coverage and is well suited for capturing long-run trends and cross-country differences, it necessarily smooths high-frequency variation and is therefore less appropriate for our empirical design, which exploits annual fluctuations in mortality. Based on this data, we expand our mortality sample. Specifically, for any country where the HMD-WHO-based series contains fewer than 30 observations, we replace it with a mortality rate constructed from UNWPP death counts and population figures. This extended variable covers up to 210 countries and is used in sensitivity checks and for the regional analysis in Africa.

A.2 Economic Data

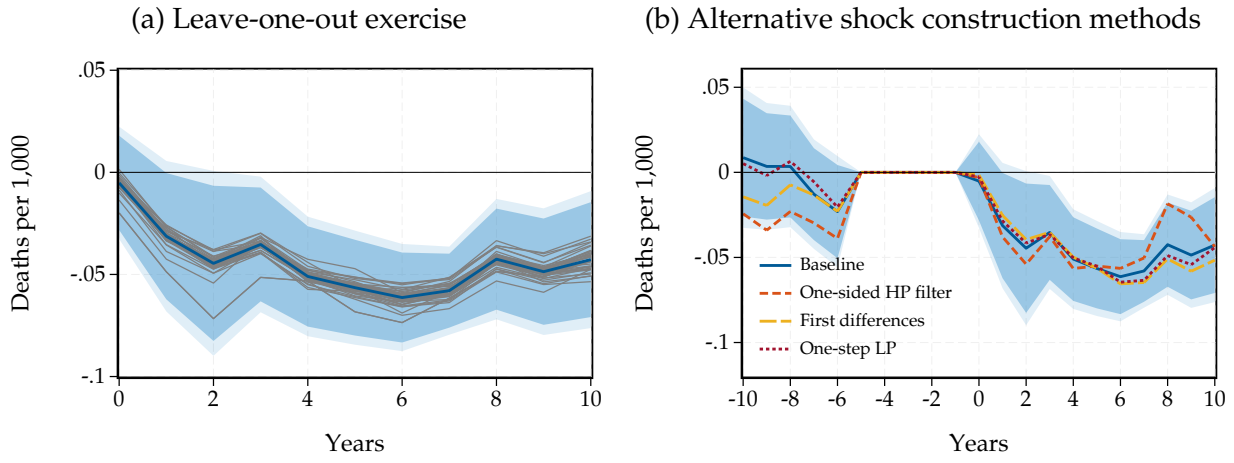
We obtain GDP and population data from the Penn World Tables (PWT; Feenstra et al., 2015), which provides comprehensive coverage for our sample countries from 1960

to 2019. Our main output measure is real GDP per capita from the national accounts (rgdpna/pop). Based on this data, we construct a measure of world real GDP, by chain-weighting country-level growth rates. We obtain a global commodity price index from Bloomberg. World recession dates are taken from the World Bank Kose et al. (2020). Data on wars are drawn from the Uppsala Conflict Data Program. Information on major epidemics and pandemics is compiled from publicly available historical sources.

B Additional Empirical Results

This appendix presents additional figures and table to supplement the results in the main text.

Figure B.1: Sensitivity to Shock Years and Shock Construction



Notes: Panel (a): Jackknife estimates setting each GDP shock to zero one at a time. The baseline estimate (solid line) lies within the range of all jackknife estimates, indicating robustness to excluding any single shock. Panel (b): Impulse responses using alternative methods for constructing GDP shocks.

C Model

C.1 Decentralized Equilibrium

C.1.1 Optimality Conditions

Firm FOCs and market clearing. We obtain:

$$\alpha Z_t K_t^{\alpha-1} = r_t + \delta \qquad\qquad r_t K_t + w_t = Y_t - \delta K_t = Z_t K_t^{\alpha} - \delta K_t.$$

Household HJB. The HJB of the households is then:

$$\rho V_t(K_t) = \max_{C_t, H_t} U(C_t) + \big(r_t K_t + w_t - C_t - H_t\big) V_{tK}(K_t) - \overline{M} H_t^{-\mu} Y_t^{-\nu} \overline{V} + \frac{\partial V_t}{\partial t}(K_t).$$

The household's first-order condition with respect to consumption C_t is:

$$U'(C_t) = V_{tK},$$

The envelope condition is

$$\rho V_{tK} = r_t V_{tK} + \frac{dK_t}{dt} V_{tKK} + \frac{dV_{tK}}{dt},$$

where crucially the household does not internalize the effect of savings on mortality improvements through aggregate output. Together with the consumption FOC, the envelop condition leads to the standard Euler equation:

$$\frac{\dot{C}_t}{C_t} = \frac{r_t - \rho}{\gamma}.$$

Substituting in the marginal product of capital:

$$\boxed{\frac{\dot{C}_t}{C_t} = \frac{\alpha Z_t K_t^{\alpha-1} - \rho - \delta}{\gamma}.}$$

Healthcare demand. The household's first-order condition with respect to health expenditure H_t is:

$$V_{tK} = \mu \frac{M_t}{H_t} \overline{V},$$

which implies:

$$\frac{H_t}{C_t^{\gamma}} = \mu M_t \overline{V}.$$

Substituting in the expression for M_t, we obtain:

$$\boxed{H_t = \overline{H} C_t^{\frac{\gamma}{1+\mu}} Y_t^{-\frac{\nu}{1+\mu}},}$$

where $\overline{H} \equiv (\mu \overline{MV})^{\frac{1}{1+\mu}}$. Substituting into mortality, we obtain:

$$\boxed{M_t = \widetilde{M} C_t^{-\frac{\mu\gamma}{1+\mu}} Y_t^{-\frac{\nu}{1+\mu}}},$$

where $\widetilde{M} = \overline{M}^{\frac{1}{1+\mu}} (\mu \overline{V})^{-\frac{\mu}{1+\mu}}$. Finally, capital accumulates according to:

$$\boxed{\dot{K}_t = Z_t K_t^{\alpha} - \delta K_t - C_t - H_t.}$$

C.1.2 Steady-State

The Euler equation implies: $\rho + \delta = \alpha \overline{Z} K_{ss}^{\alpha - 1}$, which pins down the steady-state capital stock: $\boxed{K_{ss} = \left(\frac{\alpha \overline{Z}}{\rho + \delta} \right)^{\frac{1}{1-\alpha}}}$ and steady-state output $\boxed{Y_{ss} = \alpha \overline{Z} K_{ss}^{\alpha}}$. The capital accumulation equation pins down steady-state consumption given the steady-state capital stock and given steady-state output:

$$\boxed{C_{ss} + \overline{H} Y_{ss}^{-\frac{\nu}{1+\mu}} C_{ss}^{\frac{\gamma}{1+\mu}} = \overline{Z} K_{ss}^{\alpha} - \delta K_{ss}}.$$

From there, we obtain

$$\boxed{H_{ss} = \overline{H} C_{ss}^{\frac{\gamma}{1+\mu}} Y_{ss}^{-\frac{\nu}{1+\mu}} \qquad M_{ss} = \widetilde{M} C_{ss}^{\frac{-\mu\gamma}{1+\mu}} Y_{ss}^{-\frac{\nu}{1+\mu}} \qquad I_{ss} = \delta K_{ss}}$$

C.1.3 Log-Linearized Transitional Dynamics

Equilibrium conditions. We start by noting that

$$\frac{\widehat{H}_t}{H_t} = \frac{\gamma}{1+\mu}\frac{\widehat{C}_t}{C_t} - \frac{\nu}{1+\mu}\frac{\widehat{Y}_t}{Y_t} = \frac{\gamma}{1+\mu}\frac{\widehat{C}_t}{C_t} - \frac{\alpha\nu}{1+\mu}\frac{\widehat{K}_t}{K_t}.$$

Therefore, to first order:

$$\widehat{H}_t = \frac{\gamma}{1+\mu}\frac{H}{C}\widehat{C}_t - \frac{\alpha\nu}{1+\mu}\frac{H}{K}\widehat{K}_t$$

We then linearize the resource constraint:

$$\begin{aligned}\frac{d\widehat{K}_t}{dt} &= (\alpha Z K^{\alpha-1} - \delta)\widehat{K}_t - \widehat{C}_t - \widehat{H}_t + \widehat{Z}_t K^{\alpha} \\ &= \rho\widehat{K}_t - \widehat{C}_t - \widehat{H}_t + Y\widehat{z}_t \\ &= \boldsymbol{\phi_K}\widehat{K}_t - \boldsymbol{\phi_C}\widehat{C}_t + Y\widehat{z}_t\end{aligned}$$

where we denoted $\widehat{z}_t = \widehat{Z}_t/Z$ and defined: $\boxed{\phi_K \equiv \rho + \frac{\alpha\nu}{1+\mu}\frac{H}{K}}$ and $\boxed{\phi_C \equiv 1 + \frac{\gamma}{1+\mu}\frac{H}{C}}$.

Next, we linearize the Euler equation:

$$\frac{d\widehat{C}_t}{dt} = \frac{C}{\gamma}\left(-\alpha(1-\alpha)ZK^{\alpha-2}\widehat{K}_t + \alpha K^{\alpha-1}\widehat{Z}_t\right) = \frac{C}{\gamma}\left(-\frac{(1-\alpha)r}{K}\widehat{K}_t + r\widehat{z}_t\right).$$

We define:

$$X_t = \begin{pmatrix}\widehat{K}_t \\ \widehat{C}_t\end{pmatrix}.$$

We can summarize the linearized resource constraint and Euler equation as:

$$\dot{X}_t = AX_t + S_t,$$

where:

$$A = \begin{pmatrix}\boldsymbol{\phi_K} & -\boldsymbol{\phi_C} \\ -\frac{(1-\alpha)rC}{\gamma K} & 0\end{pmatrix} \;,\; S_t = \widehat{z}_t B \;,\; B = \begin{pmatrix}Y \\ \frac{rC}{\gamma}\end{pmatrix}.$$

Consumption. We have an initial condition $\widehat{K}_0$, and a terminal condition $\widehat{C}_t \to 0$. We now apply standard Blanchard-Kahn arguments. Let $A = M^{-1}DM$, with D diagonal. For determinacy we require that parameters are such that D has a positive eigenvalue in the top left position, and a negative eigenvalue in the bottom right position. We denote by $\mathcal{X}_t = MX_t$, so that

$$\dot{\mathcal{X}}_t = D\mathcal{X}_t + MS_t.$$

We then solve explicitly for $\mathcal{X}_t$:

$$\mathcal{X}_t = e^{tD}\left[\mathcal{X}_0 + \int_0^t e^{-sD}(MS_t)ds\right].$$

Hence, long-run stability requires the top entry of the bracket to be zero as time grows. That is:

$$0 = \mathcal{X}_{0,1} + \int_0^\infty e^{-sD_1}(MS_s)_1 ds.$$

Therefore,

$$M_{1\bullet}X_0 = -\int_0^\infty e^{-sD_1}M_{1\bullet}S_s ds.$$

We can thus solve for initial consumption:

$$\widehat{C}_0 = -\frac{1}{M_{12}}\left[M_{11}\widehat{K}_0 + \int_0^\infty e^{-sD_1}M_{1\bullet}S_s ds\right].$$

We denote $\varepsilon_K = -\frac{M_{11}}{M_{12}}$, $\varepsilon_S = -\frac{1}{M_{12}}M_{1\bullet}$ and $\varepsilon_{S,s} = e^{-sD_1}\varepsilon_S$. We can write more compactly:

$$\widehat{C}_0 = \varepsilon_K\widehat{K}_0 + \int_0^\infty \varepsilon_{S,s}S_s ds.$$

Of course, this condition must hold at all times:

$$\widehat{C}_t = \varepsilon_K\widehat{K}_t + \int_0^\infty \varepsilon_{S,s}S_{t+s} ds.$$

To compute consumption somewhat more efficiently, we exploit that:

$$\begin{aligned}\widehat{C}_t &= \varepsilon_K \widehat{K}_t + \varepsilon_S B \int_0^\infty e^{-sD_1} z_{t+s} ds \\ &= \varepsilon_K \widehat{K}_t + e^{tD_1} \varepsilon_S B \int_t^\infty e^{-sD_1} z_s ds,\end{aligned}$$

after changing variables in the integral.

Capital and output. We substitute the solution for linearized consumption into the law of motion of capital:

$$\frac{d\widehat{K}_t}{dt} = (L_{11} - \varepsilon_K)\widehat{K}_t + S_{1t} - \int_0^\infty \varepsilon_{S,s} S_{t+s} ds.$$

Denote $\kappa = -(L_{11} - \varepsilon_K)$ and $\mathcal{S}_t = S_{1t} - \int_0^\infty \varepsilon_{S,s} S_{t+s} ds$ so that:

$$\frac{d\widehat{K}_t}{dt} = -\kappa \widehat{K}_t + \mathcal{S}_t.$$

We obtain:

$$\widehat{K}_t = e^{-\kappa t}\widehat{K}_0 + e^{-\kappa t} \int_0^t e^{\kappa s} \mathcal{S}_s ds.$$

In small log deviations:

$$\widehat{k}_t = e^{-\kappa t}\widehat{k}_0 + \frac{e^{-\kappa t}}{K} \int_0^t e^{\kappa s} \mathcal{S}_s ds.$$

Since (in small log deviations):

$$\widehat{y}_t = \widehat{z}_t + \alpha \widehat{k}_t,$$

we are left with calculating $\widehat{k}_t$ as a function of the sequence $\widehat{z}$. We express:

$$\begin{aligned}\int_0^t e^{\kappa s} \mathcal{S}_s ds &= \int_0^t e^{\kappa s} \left(S_{1s} - \int_0^\infty \varepsilon_{S,r} S_{s+r} dr \right) ds \\ &= \int_0^t e^{\kappa s} S_{1s} ds - \iint_0^\infty \mathbb{1}[s \leq t] \varepsilon_{S,r} S_{s+r} e^{\kappa s} ds dr \\ &= \int_0^t e^{\kappa s} S_{1s} ds - \iint_0^\infty \mathbb{1}[s \leq t] \varepsilon_S S_{s+r} e^{\kappa s - D_1 r} ds dr.\end{aligned}$$

Changing variables to $\tau = s + r$ over r, we obtain

$$\begin{aligned}\int_0^t e^{\kappa s}\mathcal{S}_s ds &= \int_0^t e^{\kappa s} S_{1s} ds - \varepsilon_S \iint_0^\infty \mathbb{1}[s \le t, s \le \tau] S_\tau e^{\kappa s - D_1(\tau - s)} ds d\tau \\ &= \int_0^t e^{\kappa s} S_{1s} ds - \varepsilon_S \int_{\tau=0}^\infty e^{-D_1\tau} S_\tau \int_{s=0}^{\min\{t,\tau\}} e^{(D_1+\kappa)s} ds d\tau \\ &\equiv \int_0^t e^{\kappa s} S_{1s} ds - \varepsilon_S \int_{\tau=0}^\infty J_{t,\tau} S_\tau d\tau,\end{aligned}$$

where we defined:

$$J_{t,\tau} = e^{-D_1\tau} \int_{s=0}^{\min\{t,\tau\}} e^{(D_1+\kappa)s} ds = e^{-D_1\tau} \frac{e^{(D_1+\kappa)\min\{t,\tau\}} - 1}{D_1 + \kappa}.$$

Thus,

$$\widehat{k}_t = e^{-\kappa t}\widehat{k}_0 + \frac{e^{-\kappa t}}{K} \int_0^\infty \left\{ B_1 \mathbb{1}[s \le t] e^{\kappa s} - (\varepsilon_S B) J_{t,s} \right\} \widehat{z}_s ds \equiv e^{-\kappa t}\widehat{k}_0 + \int_0^\infty \mathcal{K}_{t,s}\widehat{z}_s ds,$$

where we defined:

$$\mathcal{K}_{t,s} = \frac{e^{-\kappa t}}{K} \left\{ B_1 \mathbb{1}[s \le t] e^{\kappa s} - (\varepsilon_S B) J_{t,s} \right\}.$$

Hence, we have obtained:

$$\widehat{y}_t = \widehat{z}_t + \alpha \left\{ e^{-\kappa t}\widehat{k}_0 + \int_0^\infty \mathcal{K}_{t,s}\widehat{z}_s ds \right\}.$$

Welfare. To calculate welfare in the first-order approximation, we log-linearize the HJB and use the envelope condition and that net investment is zero in steady-state. We obtain:

$$\rho \widehat{V}_t + \rho V_K \widehat{K}_t = 0 + \left(\widehat{Y}_t - \delta \widehat{K}_t\right) V_K - \widehat{M}_t^C \overline{V} + \frac{\partial \widehat{V}_t}{\partial t}.$$

where note that $\widehat{V}_t$ is the change in value holding current capital fixed, and $\widehat{M}_t^C$ is the change in mortality only coming from the externality: the change from H envelops out. The total welfare change is $\widehat{W}_t = \widehat{V}_t + V_K \widehat{K}_t$.

Re-arranging:

$$\rho \widehat{V}_t - \frac{\partial \widehat{V}_t}{\partial t} = \left(\widehat{Y}_t - (\rho + \delta)\widehat{K}_t\right) V_K - \widehat{M}_t \overline{V}.$$

Integrating:

$$\widehat{V}_0 = \int_0^\infty e^{-\rho t}\Big\{u'(C)\big(\widehat{Y}_t - (\rho+\delta)\widehat{K}_t\big) - \overline{V}\widehat{M}_t\Big\}dt.$$

And total welfare is:

$$\widehat{W}_0 = u'(C)\widehat{K}_0 + \int_0^\infty e^{-\rho t}\Big\{u'(C)\big(\widehat{Y}_t - (\rho+\delta)\widehat{K}_t\big) - \overline{V}\widehat{M}_t\Big\}dt.$$

When there is no mortality, this should coincide with $u'(C)\int_0^\infty e^{-\rho t}\widehat{C}_t dt$. Without mortality, $\frac{d\widehat{K}_t}{dt} + \widehat{C}_t = \widehat{Y}_t - \delta\widehat{K}_t$. Substituting in and integrating by parts shows that these expressions indeed coincide. We now use the same calculation to express everything in terms of C and H sequences to be more interpretable.

We have $\int_0^\infty e^{-\rho t}(\widehat{Y}_t - \delta\widehat{K}_t)dt = \int_0^\infty e^{-\rho t}\left(\frac{d\widehat{K}_t}{dt} + \widehat{C}_t + \widehat{H}_t\right)dt = \int_0^\infty e^{-\rho t}(\widehat{C}_t + \widehat{H}_t)dt - \widehat{K}_0 + \rho\int_0^\infty e^{-\rho t}\widehat{K}_t dt$. Substituting in, we obtain:

$$\widehat{W}_0 = \int_0^\infty e^{-\rho t}\Big\{u'(C)(\widehat{C}_t + \widehat{H}_t) - \overline{V}\widehat{M}_t\Big\}dt.$$

If we calculate welfare relative to a scenario **in which only consumption changes**, the percent consumption-equivalent change $\widehat{\omega}$ would then satisfy: $\frac{C\omega_0 u'(C)}{\rho} = \widehat{W}_0$, so that:

$$\omega_0 = \frac{\rho\widehat{W}_0}{Cu'(C)}$$

C.2 Planner Problem

C.2.1 Optimality Conditions

Consumption and Euler equation. The only inefficient decision is the envelope condition. The planner internalizes the effects of higher capital on output. The envelope condition with the shadow interest rate (the marginal product of labor) is:

$$\rho V_{tK} = r_t V_{tK} + \frac{dK_t}{dt}V_{tKK} + \nu\frac{M_t}{Y_t}\frac{\partial Y}{\partial K}\overline{V} + \frac{dV_{tK}}{dt}.$$

Substituting in the consumption FOC and the expression for the MPK:

$$(\rho - r_t)u'(C_t) = u''(C_t)\frac{dC_t}{dt} + \nu\frac{M_t}{Y_t}(r_t + \delta)\overline{V}.$$

Re-arranging:

$$\frac{\dot{C}_t}{C_t} = \frac{r_t - \rho}{\gamma} + \nu\frac{M_t C_t^{\gamma}}{\gamma Y_t}(r_t + \delta)\overline{V}.$$

Substituting in the MPK:

$$\boxed{\frac{\dot{C}_t}{C_t} = \frac{\alpha Z_t K_t^{\alpha-1} - \rho - \delta}{\gamma} + \frac{\alpha\nu}{\gamma}\frac{C_t^{\gamma}}{K_t}M_t\overline{V}.}$$

Consumption grows faster for the planner: consumption today is lower because the planner invests more to lower mortality going forward.

Other equations. All the other equations are the same as in the decentralized equilibrium. Only the Euler equation changes.

C.2.2 Steady-State

We temporarily omit subscripts for simplicity. The only difference is in the Euler equation:

$$\begin{aligned}\rho + \delta &= \alpha Z K^{\alpha-1} + \alpha\nu\frac{C^{\gamma}}{K}M\overline{V} \\ &= \alpha Z K^{\alpha-1} + \eta Z^{-\frac{\nu}{1+\mu}} C^{\frac{\gamma}{1+\mu}} K^{-\left(1+\frac{\alpha\nu}{1+\mu}\right)},\end{aligned}$$

where $\boxed{\eta \equiv \alpha\nu\overline{V}\widetilde{M}}$. We will have to solve jointly for consumption and capital. The resource constraint delivers as in the laissez-faire:

$$C + \overline{HZ}^{-\frac{\nu}{1+\mu}} K^{-\frac{\alpha\nu}{1+\mu}} C^{\frac{\gamma}{1+\mu}} = \overline{Z}K^{\alpha} - \delta K.$$

We collect these two equations in (C, K):

$$\boxed{\begin{aligned}\rho + \delta &= \alpha Z K^{\alpha-1} + \eta Z^{-\frac{\nu}{1+\mu}} C^{\frac{\gamma}{1+\mu}} K^{-\left(1+\frac{\alpha\nu}{1+\mu}\right)} \\ ZK^{\alpha} - \delta K &= C + \overline{H}Z^{-\frac{\nu}{1+\mu}} K^{-\frac{\alpha\nu}{1+\mu}} C^{\frac{\gamma}{1+\mu}}.\end{aligned}}$$

The rest of the allocation is the same as in the laissez-faire, conditional on C_{ss}, K_{ss}.

We can try to solve these two stead-state equations with fsolve with an initial guess that corresponds to the laissez-faire. But it turns out that it fails. To help computation, we manipulate these equations a bit more. Denote by: $x = Z^{-\frac{\nu}{1+\mu}} C^{\frac{\gamma}{1+\mu}} K^{-\left(1+\frac{\alpha\nu}{1+\mu}\right)}$, and rewrite:

$$\begin{aligned} \rho + \delta &= \alpha Z K^{\alpha-1} + \eta x \\ ZK^{\alpha} - \delta K &= C + \overline{H} x K. \end{aligned}$$

Re-arranging to eliminate x, we obtain:

$$\frac{C}{K} = ZK^{\alpha-1}(1+\alpha\zeta) - \delta(1+\zeta) - \zeta\rho.$$

where $\zeta = \overline{H}/\eta$. Substituting back in x, we obtain:

$$x = Z^{-\frac{\nu}{1+\mu}} \left[ZK^{\alpha-1}(1+\alpha\zeta) - \delta(1+\zeta) - \zeta\rho\right]^{\frac{\gamma}{1+\mu}} K^{\frac{\gamma-\alpha\nu}{1+\mu}-1}$$

Substituting into the Euler equation, we obtain an equation in K only:

$$\rho + \delta = \alpha Z K^{\alpha-1} + \eta Z^{-\frac{\nu}{1+\mu}} \left[ZK^{\alpha-1}(1+\alpha\zeta) - \delta(1+\zeta) - \zeta\rho\right]^{\frac{\gamma}{1+\mu}} K^{\frac{\gamma-\alpha\nu}{1+\mu}-1}$$

The RHS is generally decreasing in K if γ is not too large. So it should have a unique solution.

C.2.3 Log-Linearized Transitional Dynamics

The planner faces a different Euler equation, but the log-linearization of the resource constraint is the same conditional on C. The Euler equation becomes to first order:

$$\frac{d\widehat{C}_t}{dt} = \left(\alpha Z_t K_t^{\alpha-1} - \rho - \delta + \widehat{\eta Z_t^{-\frac{\nu}{1+\mu}}} C_t^{\frac{\gamma}{1+\mu}} K_t^{-\left(1+\frac{\alpha\nu}{1+\mu}\right)}\right)\frac{C}{\gamma} + 0$$

and so:

$$\begin{aligned}\frac{\gamma}{C}\frac{d\widehat{C}_t}{dt} &= -\alpha(1-\alpha)ZK^{\alpha-2}\widehat{K}_t + \alpha K^{\alpha-1}\widehat{Z}_t + \frac{\alpha\nu\overline{V}C^{\gamma}M}{K}\left\{-\frac{\nu}{1+\mu}\widehat{z}_t + \frac{\gamma}{1+\mu}\frac{\widehat{C}_t}{C} - \left(1+\frac{\alpha\nu}{1+\mu}\right)\frac{\widehat{K}_t}{K}\right\}\\ &= -\frac{(1-\alpha)r}{K}\widehat{K}_t + r\widehat{z}_t - \nu\mathcal{M}\widehat{z}_t + \gamma\mathcal{M}\frac{\widehat{C}_t}{C} - (1+\mu+\alpha\nu)\mathcal{M}\frac{\widehat{K}_t}{K}\\ &= -\frac{(1+\mu+\alpha\nu)\mathcal{M}+(1-\alpha)r}{K}\widehat{K}_t + (r-\nu\mathcal{M})\widehat{z}_t + \gamma\mathcal{M}\frac{\widehat{C}_t}{C}\end{aligned}$$

where $\boxed{\mathcal{M} = \frac{\alpha\nu\overline{V}C^{\gamma}M}{(1+\mu)K}}$.

Thus, for the planner, we must simply change the A and B matrices to:

$$A = \begin{pmatrix} \boldsymbol{\phi_K} & -\boldsymbol{\phi_C} \\ -\frac{(1-\alpha)r+\boldsymbol{\psi_K}}{\gamma}\frac{C}{K} & \boldsymbol{\psi_C} \end{pmatrix} \ , \ S_t = \widehat{z}_t B \ , \ B = \begin{pmatrix} \Upsilon \\ \frac{r-\boldsymbol{\psi_z}}{\gamma}C \end{pmatrix},$$

where $\boxed{\psi_K = (1+\mu+\alpha\nu)\mathcal{M}}$, $\boxed{\psi_C = \mathcal{M}}$ and $\boxed{\psi_z = \nu\mathcal{M}}$.

D Nonlinear Transitional Dynamics

Newton algorithm. We use a Newton algorithm. We collect the equilibrium conditions:

$$\begin{aligned}\dot{C}_t &= \frac{\alpha Z_t K_t^{\alpha-1} + E_t(C_t,K_t) - \rho - \delta}{\gamma}C_t \equiv F_t(K_t,C_t)\\ \dot{K}_t &= Z_t K_t^{\alpha} - \delta K_t - C_t - \overline{H}C_t^{\frac{\gamma}{1+\mu}}(Z_t K_t^{\alpha})^{-\frac{\nu}{1+\mu}} \equiv G_t(K_t,C_t),\end{aligned}$$

where in the decentralized equilibrium $E_t \equiv 0$. For the planner, we have:

$$E_t(C_t,K_t) = \eta C_t^{\frac{\gamma}{1+\mu}} Z_t^{-\frac{\nu}{1+\mu}} K_t^{-\frac{\alpha\nu+1+\mu}{1+\mu}}.$$

Discretized, these become:

$$C_{t+1} - C_t = F_t(K_t,C_t) \qquad\qquad K_{t+1} - K_t = G_t(K_t,C_t).$$

together with initial condition $K_0 = \overline{K}_0$ given, and terminal condition $K_T = \overline{K}_T$ given. We are looking for a path of $X_t = \{C_t, K_t\}_t$ that sets to zero all these equations, which we

re-write:

$$0 = \mathcal{F}_t(K_t, C_t, C_{t+1}) \qquad\qquad 0 = \mathcal{G}_t(K_t, K_{t+1}, C_t).$$

with $\mathcal{F} = F + C_t - C_{t+1}$ and $\mathcal{G} = G + K_t - K_{t+1}$. We collect all these equations under the name $\mathcal{H}$. A Newton updating algorithm is as follows:

$$\mathbf{X}^{n+1} = \mathbf{X}^n - \mathcal{J}(\mathbf{X}^n)^{-1}\mathcal{H}(\mathbf{X}^n),$$

where $\mathcal{J}$ is the Jacobian of $\mathcal{H}$ w.r.t. X. To calculate this Jacobian consistently, we have to specify the order in which we stack the unknowns and the equations. We posit:

$$\mathbf{X}_t = \begin{pmatrix} K_1 \\ K_2 \\ \vdots \\ K_t \\ \vdots \\ K_{T-1} \\ C_0 \\ C_1 \\ \vdots \\ C_t \\ \vdots \\ C_{T-1} \end{pmatrix} \qquad \mathcal{H}(\mathbf{X}) = \begin{pmatrix} \mathcal{G}_0(\overline{K}_0, K_1, C_0) \\ \mathcal{G}_1(K_1, K_2, C_1) \\ \vdots \\ \mathcal{G}_{t-1}(K_{t-1}, K_t, C_{t-1}) \\ \vdots \\ \mathcal{G}_{T-2}(K_{T-2}, K_{T-1}, C_{T-2}) \\ \mathcal{F}_0(\overline{K}_0, C_0, C_1) \\ \mathcal{F}_1(K_1, C_1, C_2) \\ \vdots \\ \mathcal{F}_t(K_t, C_t, C_{t+1}) \\ \vdots \\ \mathcal{F}_{T-1}(K_{T-1}, C_{T-1}, \overline{C}_T) \end{pmatrix}.$$

Note that (1) there is no $\mathcal{G}_{T-1}$ condition, as this would over-determine K_T with the terminal condition, and (2) there is $\mathcal{F}_T$ condition because we already enforced $C_T = \overline{C}_T$ in the last Euler equation. The Jacobian keeps $\mathcal{H}$ on the rows, and takes $\mathbf{X}$ on the columns, is

sparse and satisfies:

$$\mathcal{J} = \begin{pmatrix}
\frac{\partial \mathcal{G}_0}{\partial K_1} & & & & & 0 & \frac{\partial \mathcal{G}_0}{\partial C_0} & & & & & & 0 \\
\frac{\partial \mathcal{G}_1}{\partial K_1} & \frac{\partial \mathcal{G}_1}{\partial K_2} & & & & & & \frac{\partial \mathcal{G}_1}{\partial C_1} & & & & & \\
 & \ddots & \ddots & & & & & & \ddots & & & & \\
 & & \frac{\partial \mathcal{G}_{t-1}}{\partial K_{t-1}} & \frac{\partial \mathcal{G}_{t-1}}{\partial K_t} & & & & & & \frac{\partial \mathcal{G}_{t-1}}{\partial C_{t-1}} & & & \\
 & & & \ddots & \ddots & & & & & & \ddots & & \\
0 & & & & \frac{\partial \mathcal{G}_{T-2}}{\partial K_{T-2}} & \frac{\partial \mathcal{G}_{T-2}}{\partial K_{T-1}} & 0 & & & & & \frac{\partial \mathcal{G}_{T-2}}{\partial C_{T-2}} & 0 \\
0 & & & & & 0 & \frac{\partial \mathcal{F}_0}{\partial C_0} & \frac{\partial \mathcal{F}_0}{\partial C_1} & & & & & \\
\frac{\partial \mathcal{F}_1}{\partial K_1} & 0 & & & & & & \frac{\partial \mathcal{F}_1}{\partial C_1} & \frac{\partial \mathcal{F}_1}{\partial C_2} & & & & \\
0 & \ddots & \ddots & & & & & & \ddots & \ddots & & & \\
 & & \frac{\partial \mathcal{F}_{t-1}}{\partial K_{t-1}} & 0 & & & & & & \frac{\partial \mathcal{F}_{t-1}}{\partial C_{t-1}} & \frac{\partial \mathcal{F}_{t-1}}{\partial C_t} & & \\
 & & & \ddots & \ddots & & & & & & \ddots & \ddots & \\
 & & & & \frac{\partial \mathcal{F}_{T-2}}{\partial K_{T-2}} & 0 & & & & & & \frac{\partial \mathcal{F}_{T-2}}{\partial C_{T-2}} & \frac{\partial \mathcal{F}_{T-2}}{\partial C_{T-1}} \\
0 & & & & & \frac{\partial \mathcal{F}_{T-1}}{\partial K_{T-1}} & 0 & & & & & & \frac{\partial \mathcal{F}_{T-1}}{\partial C_{T-1}}
\end{pmatrix}.$$

We thus have to calculate:

$$\begin{aligned}
\frac{\partial \mathcal{G}_t}{\partial K_t} &= 1 + \frac{\partial G_t}{\partial K_t} = 1 + \alpha Z_t K_t^{\alpha-1} - \delta + \frac{\alpha\nu}{1+\mu} H_t / K_t \\
\frac{\partial \mathcal{G}_t}{\partial K_{t+1}} &= -1 \\
\frac{\partial \mathcal{G}_t}{\partial C_t} &= -1 - \frac{\gamma}{1+\mu} H_t / C_t
\end{aligned}$$

$$\begin{aligned}
\frac{\partial \mathcal{F}_t}{\partial C_t} &= 1 + \frac{\alpha Z_t K_t^{\alpha-1} + E_t - \rho - \delta}{\gamma} + \frac{1}{1+\mu} E_t \\
\frac{\partial \mathcal{F}_t}{\partial C_{t+1}} &= -1 \\
\frac{\partial \mathcal{F}_t}{\partial K_t} &= \frac{\alpha(\alpha-1) Z_t K_t^{\alpha-2} - \frac{\alpha\nu+1+\mu}{1+\mu} E_t / K_t}{\gamma} C_t
\end{aligned}$$

with $H_t = \overline{H} C_t^{\frac{\gamma}{1+\mu}} Z_t^{-\frac{\nu}{1+\mu}} K_t^{-\frac{\alpha\nu}{1+\mu}}$.

Welfare. For welfare, we use steady-state welfare in the decentralized equilibrium as the baseline (for both the DE and SP cases). We have:

$$V_t + \frac{u((1+\omega_t)C_{ss}^{DE}) - u(C_{ss}^{DE})}{\rho} = V_{ss}^{DE},$$

so that:

$$u((1+\omega_t)C_{ss}^{DE}) - u(C_{ss}^{DE}) = \rho(V_t - V_{ss}^{DE}),$$

and hence:

$$\omega_t = \frac{1}{C_{ss}^{DE}} u^{-1}\left[u(C_{ss}^{DE}) + \rho(V_t - V_{ss}^{DE})\right] - 1.$$

References Appendix

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