The Macroeconomics of Improving Aging

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Abstract

A combination of market failures and misaligned incentives result in lower investments in medical treatments that could prevent age-related health decline. Age-related health issues directly affect mortality, labor productivity, and fertility and therefore present significant economic costs. We use a dynamic general equilibrium model to quantify the economic effects of delaying biological aging. Our results suggest that therapeutics with marginal effects on the biology of aging could increase US GDP by $3 trillion in the long run, or about $68 billion per year in the near term. Delaying aging by one full year could result in a gain of roughly $840 billion per year to the US economy.

keywords: Aging, health, demographics, general equilibrium, productivity

JEL classification: C68, E24, E37, I15, J11, J17, J24

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1 Introduction

Healthcare is often centered around the patient as a profit center. This has both risks and benefits. Market forces, bolstered by public funds and scientific talent, have fostered innovation and led to vast improvements in the treatment of many diseases, raising life expectancy by several years in the 20th century alone (Murphy and Topel, 2006). Yet a number of market failures and misaligned incentives have created a system more focused on treating illness than preventing it. Low R&D investment into extending the human healthspan in the absence of disease or injury may help perpetuate a system that overlooks the value of health and longevity in favor of treating late-stage or acute conditions.[1]

Individuals also face misaligned incentives toward reducing the effects of aging. Well-documented biases in behavioral economics explain why people are often more willing to pay for a cure than for prevention (Kremer and Snyder, 2003). Insurers, hospitals, and patients face a tragedy of the commons whereby all parties would be better off prioritizing long-term health, but the incentives for individual agents to do so remain misaligned. For instance, because US citizens are free to change their health insurance at any time, few insurers invest in future patients' health. And because government programs like Medicare can subsidize age-related health decline, individuals have fewer incentives to invest in lifestyle choices that increase their healthspan.

Government investment toward R&D targeting aging is also under-provided due to a generational misalignment. Because it can take years for R&D to translate into breakthroughs, the current population of older adults risks bearing all the up-front research costs without living to receive most benefits. This age group is more likely to vote and, thereby, to exert a greater influence on politics. All of these individual, commercial, and government incentives represent potential obstructions in the way of market provision of the needed R&D to map and target the biology of aging.

Today, in the United States, nearly 80% of all deaths can be attributed to diseases of

[1] Clemens and Gottlieb (2014) find that changes in compensation to doctors for treatments can illicit strong supply responses. However, while these incentives increase the number of treatments and technology, there is little improvement in overall health. This is consistent with a broad number of studies that find changes in financial incentives can affect technology adoption (Finkelstein 2007, Acemoglu and Finkelstein 2008, and number of patient visits Manning et al. 1987), but have do not have substantial impacts on preventative care or health outcomes.

aging.[2] A lack of human-relevant results in longevity research is partly a function of scientific challenges. We suggest that it is also a function of misaligned incentives. The National Institute on Aging, for instance, devotes more than seven times the funding to late-stage neurodegenerative diseases than to the biology of aging.[3]

Recent work by Goldman et al. (2013) and others suggests that research on delaying biological aging may have higher returns than research on cancer, heart disease, or any single chronic condition or illness. For example, Murphy and Topel (2006) find that eliminating all cancers would add roughly 3 years to the human life expectancy, and add about $50 trillion to the US economy in net present value. Eliminating cancer would have significant positive economic effects and would be a major step forward in public health. But the effects of aging are felt by every member of the population. Our results show that the amelioration of the effects of aging could have an even greater economic impact on many of the current medical R&D focus areas. Biological aging is also a neglected factor in global pandemics, since older adults are at higher risk of serious illness and death from communicable diseases.

This paper provides an economic analysis of delaying the onset of age-related decline. Treatments that target biological aging directly, rather than acute aging-related conditions, are a casualty of the market failures described above. Using a dynamic general equilibrium model calibrated to the US economy, we simulate counterfactual scenarios where therapeutics and biotechnologies designed to improve biological aging affect population dynamics and labor productivity. Our analysis begins by estimating the economic effects of what may be the first clinical trial to measure geroprotective effects in humans, the Targeting Aging with Metformin (TAME) trial.[4] This first analysis provides a benchmark for what may be on the horizon in the very near term, given imperfect but existing therapeutics with very marginal effects on the biology of aging. We find the endpoints of the TAME trial, if successfully achieved, would increase US GDP by about $3 trillion in net present value terms, due to

[2]See the CDC leading causes of death at https://www.cdc.gov/nchs/fastats/leading-causes-of-death.htm.

[3]The total budget amount for the National Institute on Aging (NIA) under then US National Institutes of Health in the President's budget for fiscal year 2023 was $4.011 billion (see NIA, 2023). The total funding for the Division of Neuroscience was $2.414 billion, which was predominately dementia and Alzheimer's research. The total funding for the Division of Aging Biology was $337 million (see table, "Budget Authority by Activity"). See also Ostapchuk (2022).

[4]See Padki and Stambler (2021).

increases in life expectancy and labor productivity. We note that metformin is not an ideal aging drug, but it can still be useful in the collection of better aging biomarkers and surrogate endpoints in a clinical trial setting.

We then move to a stylized example of a one-year reduction in biological age for the population of US adults aged 40 and older. In this case, we consider separately the roles of three different levers through which a reduction in biological age can affect the economy: reduced mortality, higher labor productivity, and higher fertility. Our findings demonstrate the importance of productivity and population dynamics on economic growth, highlighting aging science as an R&D area with the unique potential to affect all three channels at once (mortality, productivity and fertility). We find that a one-year reduction in biological age—effectively making all adults over the age of 40 live, work, give birth and die at the rates of adults one year younger—would increase GDP by about $50 trillion. About 40% of these effects come from increases in productivity, with decreases in mortality and increases in fertility each accounting for about 30% of the total effect.

In the analysis that follows, we abstract from the cost of R&D investment in our main net present value calculations for two reasons. First, only sparse and tenuous evidence exists on the relationship between R&D investment and the probability and size of breakthroughs.[5] Second, the magnitude of the benefits are likely to be multiple orders of magnitude larger than the investment costs. Thus, our approach to estimating the value of aging research will not rely on assumptions about the rate of scientific breakthroughs per dollar of investment in research. Instead, we focus our simulations on the value of successful breakthroughs. From

[5]Qu et al. (2023) attempt to identify the most successful research strategies for US patents in the electric communication industry from 1976 to 2021. Baba and Walsh (2010) use the development of statin drugs to focus on the role of a firm's social network on how quickly breakthroughs are introduced into the market. Angori et al. (2024) look at publicly funded research patents at universities between 1978 and 2015 and compare the university dynamics to those with industry patents. Briggs and Buehler (2018) show that a nonlinear relationship exists between the degree of radical thinking or "thinking outside the box" and innovation breakthroughs. However, none of these studies speak directly to the critical parameter: what is the percentage increase in successful R&D per dollar spent. Murphy and Topel (2006) provide break-even probabilities such that if a 1 percent decrease in mortality results from a given breakthrough, and if the necessary R&D investment is $100 million, then what is the probability of success beyond which this investment has a positive ROI. But again, these are not empirical estimates of the actual success rates or their sensitivity to additional investment. Budish et al. (2015) find evidence for reductions in therapeutic commercialization lags (a kind of investment) increasing survival rates in cancer patients. Gullo et al. (2025) estimate the broad effect of government R&D investment on productivity and simulate its effect on the broad macroeconomy.

this, the reader can apply her own risk/return assumptions to see what ranges of values might generate a positive expected return on investments. In short, we suggest any plausible investment amount would be dwarfed by returns on investment.

Related studies aimed at quantifying the value of increasing healthspan and lifespan include Murphy and Topel (2006) as well as Scott et al. (2021). Both use a partial equilibrium lifecycle model and use the value of a statistical life to infer the willingness to pay for improvements in health and longevity. Murphy and Topel (2006) apply the model to estimate the value of decreases in US mortality from 1970 to 2000, which they find have a value of $3.2 trillion per year to the year 2000 US population. Extrapolating from this, they find that curing all cancers would be worth $50 trillion in net present value. If we transform their values to 2025 dollars, their estimate becomes about $80 trillion, which is roughly 60% larger than the result in our model when delaying aging by one year. Scott et al. (2021) use the model of Murphy and Topel (2006) to quantify the impacts of hypothetical increases in healthspan and lifespan in the US. In their calibrated model, Scott et al. (2021) find that increasing life expectancy by one year would be worth $37.6 trillion in net present value terms.

In another related study, Budish et al. (2015) consider the costs of market failures leading to under-investment in long-term health projects, with a focus on cancer. Budish et al. (2015) use a Quality Adjusted Life Year (QALY) approach in which they apply a model of firm decisions and patient cohorts to estimate the change in the number of life years from a treatment. To quantify the economic costs of under-investment, they follow Cutler (2004) and value each life year at $100,000. Budish et al. (2015) estimate the lost value in the US due to market failures in cancer research at $89 billion per year with a net present value of $2.2 trillion.

We use a dynamic general equilibrium (GE) macroeconomic model to quantify the value and effects of breakthroughs in aging science.[6] Our GE model incorporates the life cycle model of the individual, similar to Murphy and Topel (2006), but in an overlapping generations, general equilibrium (GE) framework. This allows us to capture the effects of

[6]We use the open-source OG-USA large scale overlapping generations model of US fiscal policy https://pslmodels.github.io/OG-USA. All of our code and output for replicating the analyses in this paper are available in the GitHub repository https://github.com/OpenRG/OG-Lifespan.

individuals' behavioral responses across their lifespan to the effects of medical breakthroughs of reduced mortality rates, increased productivity, and increased fertility rates (leading to population growth). But beyond the partial equilibrium approach, the GE approach models these heterogeneous agents of different ages in each period interacting with each other and influencing prices and aggregate outcomes. The GE effects can be significant in the case of simulating increases in healthspan, since there are large aggregate effects on the population distribution.

The overlapping generations structure of the model we use captures rich demographic dynamics driven by mortality, fertility, and immigration rates. The transition path in the model, from the current state to the long-run steady state, simulates the economy over hundreds of years, which is valuable because demographic transitions can take more than 100 years to reach a new steady state. These long timelines become important when our experiments involve medical breakthroughs that alter mortality rates and fertility rates whose effects can take decades to fully materialize in the population distribution. In the case of age-related infertility treatments, for instance, it takes one generation for therapeutics which improve the reproductive healthspan to translate into a larger labor force. In short, the approach we take provides an internally consistent model of individual choices, population dynamics, and market outcomes and allows us to model the economic transition to a world with improved healthspan and lifespan.

Health and economic research on aging are both active areas of academic and policy work.[7] Actual medical trials on aging therapeutics are only now beginning (see Barzilai et al., 2016a). Our hope in this article is to provide conservative quantitative estimates of the benefits from breakthroughs in aging therapies. These net benefit estimates, in turn, will highlight the economic returns for private industry and government at stake in overcoming the currently misaligned incentives and market failures standing in the way of R&D investment in improving the biology aging.

The remainder of the paper proceeds as follows. In Section 2, we outline the mechanics

[7]The National Bureau of Economic Research (NBER) has an Economics of Aging program (https://www.nber.org/programs-projects/programs-working-groups/economics-aging) with 152 affiliated scholars and an annual meeting. The *Journal of the Economics of Ageing* (https://www.sciencedirect.com/journal/the-journal-of-the-economics-of-ageing) was established in 2013.

of our macroeconomic model in detail, with particular focus on how age is reflected in model parameters and how those parameters affect agents' decisions. We then present results from simulations of counterfactual futures with varying assumptions about aging improvements in Section 3. We use Section 4 to discuss potential limitations of our analysis, and Section 5 for a concluding overview of our results and methods.

2 An Economic Model of Aging

2.1 Model Overview

To simulate the economy in counter-factual scenarios, we use the `OG-USA` dynamic general equilibrium model.[8] This model features overlapping-generations of households who vary in their labor productivity profiles. Households make choices each period about how much to work, save, and consume. These choices are made with perfect foresight, where the only uncertainty the households face is with respect to their mortality.

In addition to households, our parameterization of the model contains a representative firm that produces output using capital and labor in a constant returns to scale production function. The government raises revenue through individual income and payroll taxes and a corporate income tax. This revenue is used to finance a public pension system (i.e., social security), other transfers, government purchases of goods and service, and interest payments on any borrowing. Gaps between revenues and outlays are financed by government debt issues, although the long run of the model must have a constant debt-to-GDP ratio to ensure that no Ponzi scheme assumption is met.

We solve this model for its long run steady state, as well as the transition from the model's initial state to the steady state. It is this transition path that will be the focus of the results presented in this paper.

The model parameters are calibrated to match a number of characteristics of the US economy. These include the capital-output ratio, the debt-to-GDP ratio, the distributions

[8]Documentation for the `OG-USA` model is online at https://pslmodels.github.io/OG-USA. Documentation for the model theory is online here https://pslmodels.github.io/OG-Core. All of our code and output for replicating the analyses in this paper are available in the GitHub repository https://github.com/OpenRG/OG-Lifespan.

of wealth and income, variations in labor productivity across the lifecycle and across skill groups, the profile of hours worked by age, average and effective marginal tax rates on households and businesses, and other salient features. We omit a discussion of each parameter value here in the interest of brevity, but refer the reader to the `OG-USA` documentation for a detailed description of the calibration and the resulting parameter values.

2.2 Aging

Age is a state variable in our lifecycle model and agents' choices naturally vary as they age due to changes in labor productivity, the accumulation of savings, and a decline in the expected life-years remaining.

Our simulations of improvements in biological aging will affect the model through three main channels: 1) labor productivity and the related disutility of labor supply, 2) mortality rates, and 3) fertility rates. By treating aging, we are assuming that cognitive and physical decline will be slowed. This will increase productivity in later life. In addition, it will reduce the direct and opportunity costs of working and therefore lower the disutilty of labor supply in the model. Aging treatments are also assumed to have a direct impact on mortality rates by reducing age-related disease and decline. Finally, we will assume that therapeutics which directly target biological aging will extend the reproductive healthspan and thus marginally increase fertility rates.

For a full description of the model, we refer the reader to DeBacker et al. (2019). Here, we present the key equations that relate the parameters of the model which are adjusted to reflect changes in labor productivity, mortality, and fertility.

2.2.1 The role of productivity and mortality in household decisions

The household in the model chooses savings, consumption, and labor supply in each period of life to maximize their discounted, expected lifetime utility subject to their budget constraint. The per period utility function is given as:

$$u(c_{j,s,t}, n_{j,s,t}, b_{j,s+1,t+1}) \equiv \frac{(c_{j,s,t})^{1-\sigma} - 1}{1-\sigma} + e^{g_y t(1-\sigma)} \chi^n_s \left(b \left[1 - \left(\frac{n_{j,s,t}}{\tilde{l}} \right)^{\upsilon} \right]^{\frac{1}{\upsilon}} \right) + \chi^b_j \rho_s \frac{(b_{j,s+1,t+1})^{1-\sigma} - 1}{1-\sigma} \quad \forall j, t \quad \text{and} \quad E+1 \leq s \leq E+S \tag{1}$$

Consumption of the household of type j at age s in time period t is given as $c_{i,s,t}$, labor supply is denoted $n_{j,s,t}$, and household financial assets at the start of period t are $b_{j,s,t}$, while the savings they leave period t with is $b_{j,s+1,t+1}$. Note that consumption is strictly positive, while labor supply must be in the set $(0, \tilde{l}]$, where $\tilde{l}$ is the maximum fraction of a model period a household can work. The first component of the utility function is a constant relative risk aversion function of consumption, with a coefficient of relative risk aversion (CRRA) given by σ. The second component of the utility function represents the disutility of labor supply. This component has a utility weight of χ^n_s, which varies by age s, representing changes over the lifecycle in the direct and opportunities costs of participating in market work. There is also a term in front of this component of the household utility, which is $e^{g_y t(1-\sigma)}$. This term is included so that the disutility of labor is scaled by the underlying economic growth, given by g_y. This scaling in necessary because household consumption and savings grow at the rate g_y, while labor supply is stationary. Thus, without this scaling, in the long run, the disutility of labor would shrink relative to the contributions from consumption and the bequest motive.

The disutility of labor function is an elliptical function, developed by Evans and Phillips (2017). It has the nice property of asymptotes at 0 and $\tilde{l}$, which in turn helps the numerical optimization stay within the bounds on labor supply. The third and final term in the utility function is warm-glow bequest motive. This term has a utility weight of χ^b_j, which varies by household type (i.e., skill level or productivity) in order to match the skewed distribution of bequests. Note that this term is discounted by the mortality rate, ρ_s, since a household only leaves a bequest if they do not survive to the next period. The function for the warm glow bequest motive is also a CRRA function with the same coefficient as the function for consumption. The functional form for the consumption and bequest motive both share the same shape. This helps ensure the model can be stationarized when there is underlying

technological and population growth that increase the size of c and b over time.

The household maximizes the discounted expected value of the lifetime of utility flows:

$$\max_{\{(c_{j,s,t}),(n_{j,s,t}),(b_{j,s+1,t+1})\}_{s=E+1}^{E+S}} \sum_{s=1}^{S} \beta_j^{s-1} \left[\Pi_{u=E+1}^{E+s}(1-\rho_{u,t+u-E})\right] u(c_{j,s,t+s-1}, n_{j,s,t+s-1}, b_{j,s+1,t+s}), \tag{2}$$

Where β_j is the discount factor for households of type j. Utility is maximized subject to the household's budget constraint, where the period budget constraint is given as:

$$\begin{aligned} p_t c_{j,s,t} + b_{j,s+1,t+1} =& (1+r_{p,t})b_{j,s,t} + w_t e_{j,s,t} n_{j,s,t} + bq_{j,s,t} + tr_{j,s,t} + -T_{j,s,t} \\ & \forall j,t \quad \text{and} \quad s \geq E+1 \quad \text{where} \quad b_{j,E+1,t} = 0 \quad \forall j,t \end{aligned} \tag{3}$$

The budget constraint has the following prices. The price p_t represents the tax inclusive price of the composite consumption good in period t, $r_{p,t}$ is the rate of return on the household's financial portfolio, and w_t is the wage rate per effective unit of labor. The parameter $e_{j,s,t}$ represents the labor productivity of households of type j of age s in time t and can be thought of as the number of effective units of labor supplied per unit of time working. Reading from left to right, the budget constraint says that in each period spending on consumption ($p_t c_{j,s,t}$) plus new asset purchases ($b_{j,s+1,t+1}$) must equal gross asset income ($[1+r_{p,t}]b_{j,s,t}$) plus labor income ($w_t e_{j,s,t} n_{j,s,t}$) plus bequests ($bq_{j,s,t}$) and government transfers ($tr_{j,s,t}$), less total income and wealth taxes paid ($T_{j,s,t}$).

One can use the budget constraint to solve for $c_{j,s,t}$ as a function of saving ($b_{j,s+1,t+1}$) and labor supply ($n_{j,s,t}$), reducing the household's per-period problem to the choice of savings and labor supply. The necessary conditions for the optimal choices of savings and labor supply in each period are given by the following equations:

$$\begin{aligned} \frac{(c_{j,s,t})^{-\sigma}}{p_t} =& \chi_j^b \rho_{s,t} (b_{j,s+1,t+1})^{-\sigma} + \beta_j \big(1-\rho_{s,t}\big) \left(\frac{1+r_{p,t+1}\big[1-\tau_{s+1,t+1}^{mtry}\big]}{p_{t+1}}\right) (c_{j,s+1,t+1})^{-\sigma} \\ & \forall j,t, \quad \text{and} \quad E+1 \leq s \leq E+S-1 \end{aligned} \tag{4}$$

$$\frac{w_t e_{j,s,t}}{p_t}\left(1-\tau^{mtrx}_{s,t}\right)\left(c_{j,s,t}\right)^{-\sigma} = e^{g_y(1-\sigma)}\chi^n_s\left(\frac{b}{\tilde{l}}\right)\left(\frac{n_{j,s,t}}{\tilde{l}}\right)^{\upsilon-1}\left[1-\left(\frac{n_{j,s,t}}{\tilde{l}}\right)^{\upsilon}\right]^{\frac{1-\upsilon}{\upsilon}} \quad (5)$$
$$\forall j,t, \quad \text{and} \quad E+1 \leq s \leq E+S$$

In the two first order conditions above, there are just two new terms, $\tau^{mtry}_{s+1,t+1}$ and $\tau^{mtrx}_{s,t}$. These represent the marginal tax rates on capital income and labor income, respectively. Equation 4 tells us that the household will choose savings to equate the marginal utility per dollar spent today to the expected marginal value of a larger bequest plus the discounted, expected marginal utility of consumption in the next period, where expectations are over the probability of survival, $(1-\rho_s)$. Equation 5 notes that at the optimal choice of labor supply, the household will equate the marginal utility of consumption per hour worked to the marginal disutility of labor supply.

From the equations describing the household problem, we have terms reflecting mortality and productivity, both of which will be affected by the simulations of aging improvements. Mortality rates show up in the household's problem in the first order conditions for savings. In particular, as the mortality rate goes down, the expected value of future consumption increases, resulting in higher savings. But a decline in mortality rates also decreases the expected value of the warm glow bequest, leading to a decrease in savings. Thus, from the household's problem, there is an ambiguous effect of changes in mortality rates on savings and therefore in aggregate supply of capital.

In terms of productivity, we consider the life cycle profiles of effective labor given by $e_{j,s,t}$ as well as the utility weight on the disutility of labor supply, χ^n_s. These show up in the household's first order condition for the supply of labor, Equation 5. An increase in $e_{j,s,t}$ will have two countervailing effects on labor supply. First, there is a substitution effect. Namely, if $e_{j,s,t}$ increases, the household gets more income for each hour worked and will substitute away from leisure and towards market work. On the other hand, as $e_{j,s,t}$ increases, household income goes up and the household will choose to consume more leisure. Thus, changes in labor supply from a change in effective labor per hour worked have an ambiguous effect on labor supply and will depend on the relative size of the substitution versus income effects.

In addition, a change to the utility weight, χ_s^n affects labor supply in a clear direction. Lowering the disutility of labor supply through a lower value of χ_s^n will significantly increase labor supply.

2.2.2 Mortality and fertility's effects on the economy

Moving beyond individual household choices, we consider the effects of mortality on the aggregate economy. This is also where changes in fertility will manifest. To see this, we need to discuss, briefly, how demographics are handled in the macroeconomic model and the market clearing conditions that must be satisfied in general equilibrium.

Let Ω_t represent the population distribution, the number of agents of each age s, at time t. This Ω_t matrix has elements $\omega_{s,t}$ and these $\omega_{s,t}$ evolve according to the law of motion:

$$\begin{aligned} \omega_{1,t+1} &= (1-\rho_{0,t})\sum_{s=1}^{E+S} f_{s,t}\omega_{s,t} + i_{1,t}\omega_{1,t} \quad \forall t \\ \omega_{s+1,t+1} &= (1-\rho_{s,t})\omega_{s,t} + i_{s+1,t+1}\omega_{s+1,t} \quad \forall t \quad \text{and} \quad 1 \leq s \leq E+S-1 \end{aligned} \tag{6}$$

The terms in these equations not described above are $f_{s,t}$, the fertility rates of agents of age s at time t, and $i_{s,t}$, the rate of immigration of agents of age s at time t. Fertility rates are given as the fraction of agents of age s that have a child. Immigration rates are defined as net immigration of agents aged s as a fraction of the population aged s. The first equation in 6 says the number of one-year-olds is equal to the number of newborns ($f_{s,t}\omega_{s,t}$) who survive infancy plus the number of net immigrants of age one ($i_{1,t}\omega_{1,t}$). The second equation says the population of those older than one evolves as a function of the survival rates of those in the country plus net immigration. From these two equations we see that improvements in reproductive aging which result in increased fertility rates in turn increase the size and growth rate of the population. By contrast, decreases in mortality will increase the size of the population, but not its long-run growth rate.

In terms of the macroeconomic effects of mortality and fertility, it is changes in the size of the population that will impact economic aggregates most. The market clearing conditions perhaps show this most clearly. The market clearing condition for the labor market says that total labor demand from producers, L_t must be equal to the total supply of effective

labor units from households:

$$L_t = \sum_{s=E+1}^{E+S} \sum_{j=1}^{J} \omega_{s,t} \lambda_j e_{j,s,t} n_{j,s,t} \quad \forall t \tag{7}$$

From this, we can see that if the population increases (through lower mortality or increased fertility), then aggregate labor supply will increase. In equilibrium, wages will move so that this supply is met by producer demand for labor. And with higher labor demanded, more output can be produced given the aggregate production function $Y = F(K, L)$. Note also from this market clearing condition that, holding constant labor supply, an increase in productivity ($e_{j,s,t}$) will increase the aggregate effective labor supply and increase output.

Similarly, for asset markets, the total demand for debt and private capital must be equal to the supply of savings from the household:

$$D_t + K_t = \sum_{s=E+2}^{E+S+1} \sum_{j=1}^{J} \Big(\omega_{s-1,t-1} \lambda_j b_{j,s,t} \Big) + \text{Net Foreign Investment}_t \quad \forall t \tag{8}$$

As well, lower mortality and higher fertility lead to a larger population and thus a larger supply of savings. This increase in the supply of savings will lower interest rates until the capital demanded (K_t) and government bonds (D_t) equal the supply of savings. More capital demanded by producers will increase output.

2.3 Mapping the Effects of Improved Aging to an Economic Model

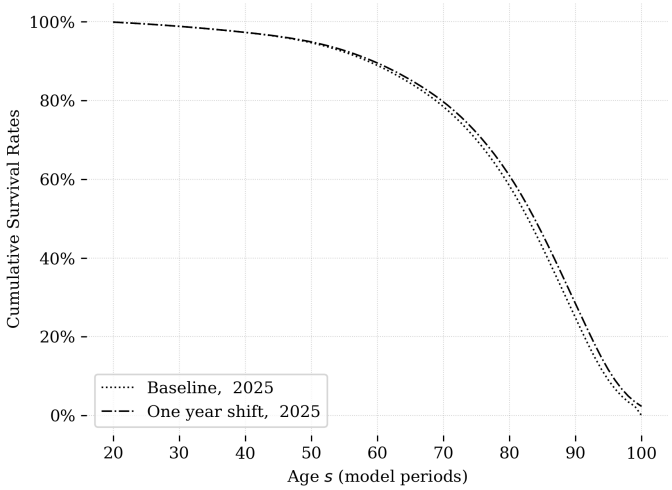
In this section, we specify the quantitative values by which the three channels (productivity, mortality, and fertility) are affected in our simulations in Section 3. We also discuss how to operationalize these effects in our model.

When making a change to any one of these levers, we need to make assumptions about the average effect size and the pattern through which that average effect is obtained. That is, we need to make assumptions about how treatment effects vary with age. Average effect sizes of aging treatments cannot be fully benchmarked through existing medical trials. Given the market failures described in the introduction, clinical trials have so far lacked the mechanisms to measure age-specific effects. Though significant results exist in animal models, aging trials

are only beginning in humans, and will take years to conclude. Because of these difficulties, we proxy for age-specific effects by making changes relative to observed age patterns (e.g., how mortality rates increase by age). Specifically, when simulating a change in a lever, we will shift the observed lifecycle profile forward (or backward) to match a specific average treatment effect. Our implicit assumption, in other words, is that chronological 41-year-olds, if they enjoyed the same biological profile of 40-year-olds, would have mortality rates, fertility rates, and labor productivity levels of current 40-year-olds. This approach is helpful because while we lack age-specific treatment effects from controlled trials, we have good measurement of mortality, fertility, and labor productivity of existing adults. In addition, shifting age profiles in this way maps nicely to the general idea of improving aging, which is to shift biological age relative to chronological age.

Figure 1 shows a one-year shift in current US mortality rates for those aged 40 and older, as observed through the survival functions before and after this shift. This shift method helps us approximate average treatment effects. By shifting the curve forward, so that older patients have the mortality/productivity/fertility of patients one year younger, we can infer US adults' behavioral choices based on their biological profile.

Figure 1: Current US Survival Rates, Before and after a shift in biological age, 2025



Source: 2025 US survival rates by age forecasts taken from United Nations Population Data Portal using the `Population-Data` Python package at https://github.com/EAPD-DRB/Population-Data.

Figure 2 shows the analogous one-year shifts in worker productivity profiles, where solid lines represent profiles of hourly earnings for workers in various percentile groups of the lifetime income distribution, as estimated from a large panel of US workers.[9] This figure in particular, where different groups of workers have very different productivity profiles, highlights the advantages of shifting the empirically estimated profiles, instead of applying a constant percentage change.

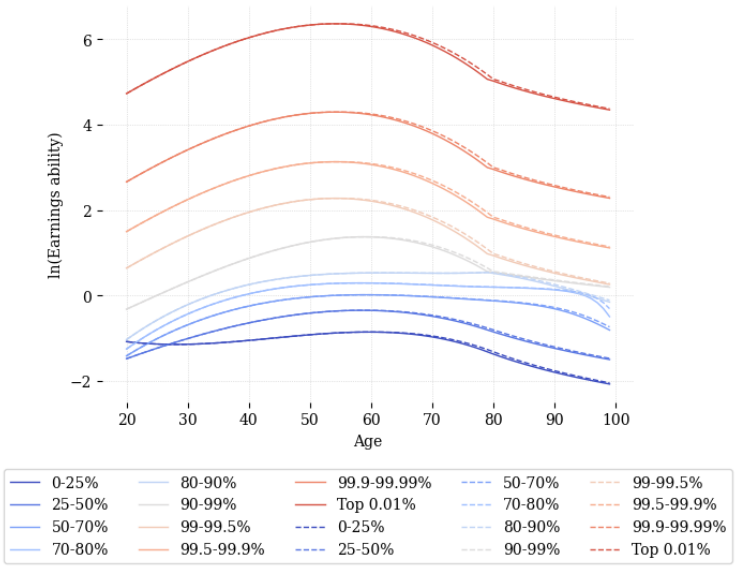
Finally, Figure 3 relates fertility rates for 2025 as forecast by the World Population Prospects project at the United Nations and a one-year shift in fertility rates for US adults aged 40 and older.

3 Effects of Aging Interventions on the Macroeconomy

From the preceding sections, one pattern is clear. While interventions that increase population growth have positive effects on the size and growth of the economy, the role of mortality

[9]See DeBacker et al. (2017b) for details on the estimation of these earnings profiles.

Figure 2: Labor productivity, Before (solid) and after (dashed) a shift in biological age

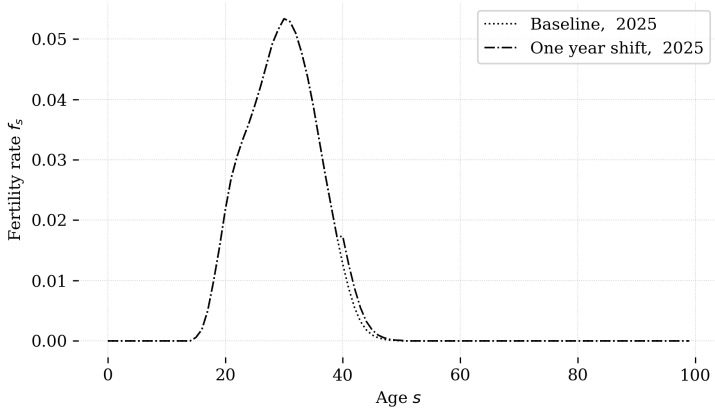


Source: Lifetime earnings profiles estimated from longitudinal proprietary US IRS data in DeBacker et al. (2017b).

and productivity in influencing individuals' decisions regarding labor supply and savings is ambiguous. This highlights the needs for a quantitative model, which can help provide evidence of the direction and size of effects of changes in mortality, fertility, and productivity when model parameters at baseline are set to capture observed characteristics of the economy.

In this section, we use a quantitative macroeconomic model to conduct two sets of such simulations. In the first, we simulate the economic effects of aging treatments that are likely to result from a near-term large-scale trial of an existing drug with the potential to directly target biological aging (Barzilai et al., 2016b). In the second, we simulate the impact of a one-year change in biological age. We note that this one-year shift is different from extending human life expectancy by one year. A one-year increase in life expectancy has been engineered many times before—for instance, by the introduction of refrigeration and the ability to safely store food. Improvements in the biology of aging, even marginal, may be more difficult to engineer. However, they may also have less diminishing results. While hypothetical, our two simulations give us a sense of the scale of the macroeconomic effects of reversing biological aging, first marginally, with existing therapeutics, then by one

Figure 3: Fertility rates, Before and after a shift in biological age



Source: United Nations, World Population Prospects

Source: 2025 US fertility rates by age forecasts taken from United Nations Population Data Portal using the `Population-Data` Python package at https://github.com/EAPD-DRB/Population-Data.

full year.

3.1 Economic Effects of Potential Near-term Improvements in Aging

As of January 2025, the most well-established trial for measuring the effects of an existing therapeutic on biological aging is the Targeting Aging with Metformin (TAME) Trial. As the reader may suspect, metformin is not an ideal drug for targeting the biology of aging, since it a) has negative side effects for young adults, and b) has only marginal (and so far unproven) effects on the biology of human aging (it has proven effects on the biology of other animal's aging). This means the TAME trial also falls within the category of late-stage treatments, with therapeutic interventions beginning at age 65. It is likely that in the category of existing therapeutics, GLP-1 agonists or rapamycin would have superior effects on mortality, but they may or may not have a less favorable safety profile. We chose to consider the effects of metformin given its relatively well-studied safety profile in older adults. The TAME trial is also a helpful benchmark in that it allows us to infer plausible

effects of near-term therapeutics on biological aging. Scientifically, it is a useful trial in that it will be designed to collect and improve biomarkers of aging (i.e., the ability to measure age-related health decline more precisely in future trials).

TAME is a series of several, six-year clinical trials with over 3,000 participants. Among the outcomes the trial will study are the effects of the drug metformin on mortality and cognitive and physical function for *non-diabetic* patients. Trial subjects were selected from a pool of candidates 65-79 years of age who showed some age-related impairment. Two trial endpoints with implications for our macroeconomic levers of mortality and labor productivity are the following: a 20% reduction in outcomes including all cause mortality and a 30% reduction in the decline of certain biomarkers of aging (e.g. walking speed, bodily pain, cognitive function).

To map these trial endpoints into our model levers, we make the following assumptions. First, since the TAME trial endpoint groups mortality decline with other outcomes, we are unable to separate changes in mortality from the other outcomes. We therefore turn to Campbell et al. (2017), who provide a meta-study of the effects of metformin. They find a 7% reduction in the mortality hazard rates as the central estimate over several metformin studies. Given the similar treatment in the TAME trial and the fact that this finding is within the bounds of what is expected from the TAME trial, we assume a decline in all-cause mortality of 7%, equating to a 6.1% reduction in mortality rates as a result of treatment. We assume only 11% of the population would be treated, following the sampling of the TAME trial, resulting in a 0.67% reduction in mortality rates across the population (6.1% $\times$ 11% = 0.67%).

Second, we connect biomarkers of aging (e.g., cognitive function) to productivity through the research of Lindqvist and Vestman (2011) who find that functions similar to these biomarkers account for about 30% of labor productivity. In US data we find that average hourly earnings decline by about 66% from age 65 to 80. We thus take the expected effect of the TAME treatment on productivity to be a change of 30% $\times$ 66% $\times$ 30% = 5.94% higher labor productivity. Given the assumption that only 11% of the population would be treated (excluding diabetics, for which metformin was formally approved), we arrive at an average effect across the population of a 0.65% increase in productivity. We apply these pop-

ulation average effects to all agents aged 40 and older in the model. Our model start period is 2025. We conservatively assume it takes 10 years for these treatments to be approved (by 2035), and another 10 years (2045) for the average effects to fully phase in.

The TAME trial endpoints suggest modest declines in mortality, leading to small increases in the size of the population. Together with increases in productivity, these two levers increase GDP, as we show in Figure 4.[10] Though these effects are relatively small, the cumulative effects are not trivial. Over the the first 20 years this treatment has full market penetration in our simulations (2045-2065), the average annual increase in GDP is $68 billion in constant, 2025 dollars. Table 1 summarizes the net present value of the increases in GDP over the next 100 years. The increase is computed as the difference between the current forecast and the counterfactual under four different discount factors. We use constant discount rates of 2%, 4% and 6%, as well as the time-varying endogenous interest rate from the model equilibrium, which arises in our counter-factual simulation. These NPVs vary between $430 billion and $3 trillion. Even the lower end of the range of NPVs is several orders of magnitude higher than the $45-70 million the TAME trial is expected to cost.[11] This highlights the potential net benefits of addressing the market failure in research on improvements in biological aging. For comparison, Budish et al. (2015) estimate that delays in the commercialization of cancer drugs cost the US about $2.2 trillion in NPV at a 5% discount rate. In 2025$, this number is about $3.5 trillion, similar to the impacts the treatments studied in the TAME trial may have.

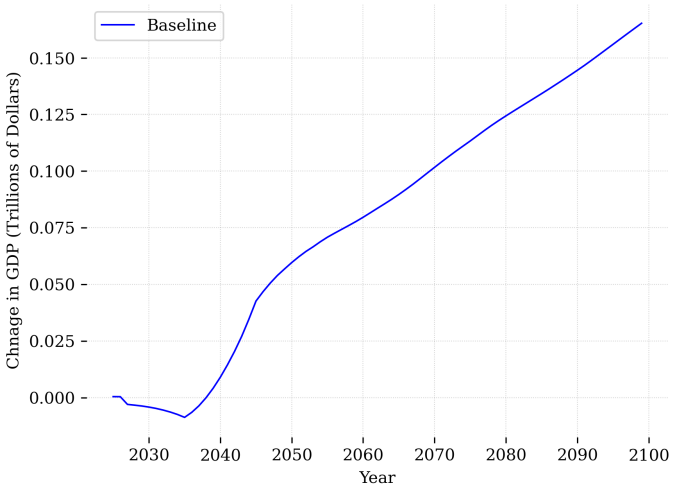
3.2 Economic Effects of a One-Year Shift in Biological Age

We now consider a one-year shift in biological age. In this hypothetical example, we shift the life cycle profiles of mortality, productivity, and fertility back one year for those aged 40 and older. In other words, a person with a calendar age of s will have the mortality risk

[10] This figure plots a baseline forecast that is constructed to match the Congressional Budget Office's Long Term Budget Outlook: 2024 to 2054 (CBO, 2024), extrapolated to 2100. The TAME trial results are based on percentage changes from the baseline forecast implied by the model's transition path equilibrium under the changes to mortality and productivity described above.

[11] See https://www.npr.org/sections/health-shots/2024/04/22/1245872510/a-cheap-drug-may-slow-down-aging-a-study-will-determine-if-it-works.

Figure 4: Change in GDP, TAME Trail vs. Baseline Forecast



The baseline forecast is the Congressional Budget Office's Long Term Budget Outlook: 2024 to 2054 (CBO, 2024), which we extrapolated to 2100. The TAME trial results are based on percentage changes from the baseline forecast implied by the model's transition path equilibrium under the changes to mortality and productivity described in the text.

of a person of age $s-1$, and likewise for labor productivity, and fertility as represented in Figures 1 through 3.

Shifting biological age by one year represents a more significant impact on age-related outcomes than that of the endpoints of the TAME trial. Figure 5 plots changes in US GDP under the counterfactual scenario, which shows a significant increase in GDP over the next several decades. This is driven, in part, by increased participation due to the lower disutility of labor, but more significantly by the increased population resulting from higher survival

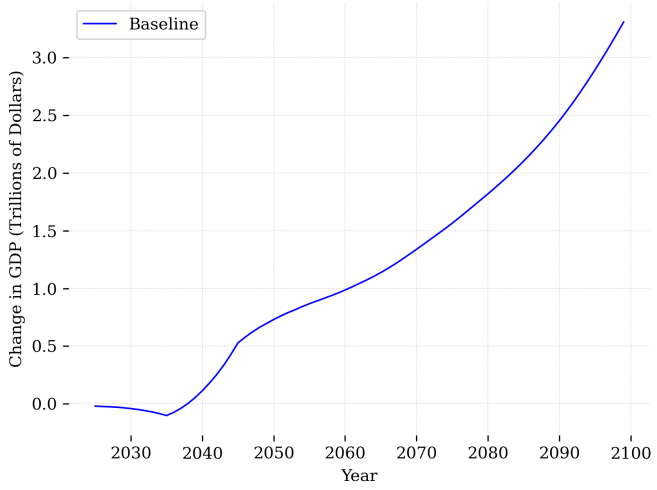
Table 1: TAME Trial, NPV of Cumulative Change in GDP (Trillions of 2025$)

Discount rate	NPV, Δ GDP
2%	3.06
4%	1.05
6%	0.43
Model r	0.54

and fertility rates. Increases in fertility result in a permanently higher level of population growth. This is apparent through the increasing slope of the two series in the out years.

The increases in population growth and productivity per capita result in higher GDP. During the first 20 years after the treatment has fully penetrated the market (2045-2065), the average annual increase in GDP is about $840 billion in constant 2025 dollars. Over the 100 years from 2025 to 2125, real GDP increases by $6 trillion to $54 trillion, depending on the discount factor used. These effects on GDP are summarized in Table 2. The magnitude of these effects can be compared to the findings of Murphy and Topel (2006), who estimate that curing all cancers would be worth about $50 trillion to U.S households, or about $80 trillion in 2025$. The large difference between the NPV of the change in GDP at an interest rate of 2% versus 6% is due to the benefits increasing the further out in the forecast one is. Population changes, especially due to increases in fertility, can take a generation to begin to significantly affect the labor force.

Figure 5: Changes in GDP, One-year Shift in Biological Age vs. Baseline Forecast



The baseline forecast is the Congressional Budget Office's Long Term Budget Outlook: 2024 to 2054 (CBO, 2024), which we extrapolated to 2100. The one-year shift in biological age results are based on percentage changes from the baseline forecast implied by the model's transition path equilibrium under the changes to mortality and productivity described in the text.

Table 2: One-year Shift in Biological Age, NPV of Cumulative Change in GDP (Trillions of 2025$)

Discount Rate	NPV, Δ GDP
2%	53.79
4%	16.23
6%	6.04
Model r	7.64

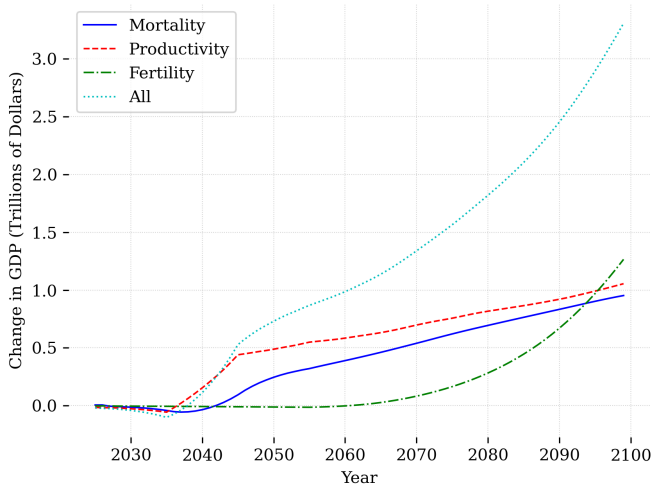
3.3 Decomposing the Roles of Mortality, Productivity, and Fertility on the Macroeconomy

In order to better understand what is driving the overall results of the one-year shift in biological age, we now decompose the total effect by moving each age-related parameter separately. That is, we simulate three counterfactual scenarios: mortality rates shift by one year, labor productivity shifts by one year, and fertility shifts by one year.

Figure 6 relates the change in GDP from each of these simulations. This figure highlights one of the key differences between these levers, the timing with which they impact the economy. Recall we assume it takes 20 years, or until 2045, before any of these effects are fully phased in. Following this assumption, the effects of the shift in productivity are almost immediate. On the other hand, changes in mortality take slightly longer to fully manifest. Fertility's effects take even longer. The latter are hardly noticeable in until roughly 2075, after which point they continue to grow larger given the compounding population growth rate. This helps explain why the NPV of benefits are so much larger in every scenario with a lower discount rate. Fertility changes have very large effects in the long run, but take almost 50 years to start showing any significant impacts.

Table 3 summarizes the NPV of changes in GDP in the four simulations. We see that the NPV of the effects of fertility changes decrease most quickly as the discount factor rises. The effects of productivity, on the other hand, decrease least quickly when the discount factor rises.

Figure 6: Change in GDP, One-year Shift in Biological Age vs. Baseline Forecast



The baseline forecast is the Congressional Budget Office's Long Term Budget Outlook: 2024 to 2054 (CBO, 2024), which we extrapolated to 2100. The decomposition of one-year shift in biological age results are based on percentage changes from the baseline forecast implied by the model's transition path equilibrium under the changes to mortality and productivity described in the text.

Table 3 shows that the one-year shift in productivity has the largest impact on GDP, under all discount rates.

For another interpretation of the relative impacts of each age-related parameter on GDP, we compute the elasticity of the NPV of changes in GDP with respect to each parameter. To begin this calculation, we compute the population average percentage change in each age-related parameter using the 2025 population distribution.[12] Considering adults aged 40

[12]Recall, the age distribution changes over time and is different when we change morality and fertility rates.

Table 3: Decomposition of One-year Shift in Biological Age, NPV of Cumulative Change in GDP (Trillions of Constant 2025$)

Discount Rate	Mortality	Productivity	Fertility	All
2%	15.86	21.56	15.68	53.79
4%	5.02	7.84	3.16	16.23
6%	1.86	3.43	0.67	6.04
Model r	2.39	4.30	0.91	7.64

and older, we find that a one-year shift in mortality reduces mortality rates by 7.19%, while a one-year shift in productivity increases average labor productivity by 2.07%. Coincidentally, a one-year shift in fertility is also a 2.07% increase in fertility rates. Dividing the percentage change in the NPV of GDP over the next 100 years by these percentage changes in parameters, we find our elasticity of the change in GDP with respect to each parameter. These elasticities are summarized in Table 4. On a percentage change basis, we see that under a 2% discount rate, changes in labor productivity yield the largest impacts on GDP over a 100-year time series when considering a 1-year shift in biological age. With a discount rate of 2%, a one-percentage point increase in productivity increases the NPV of GDP over the next 100 years by 0.42%. Fertility rates have the next largest elasticity: a 1% increase in fertility results in a 0.32% increase in GDP over the next 100 years.

Table 4: Elasticity of GDP with respect to Model Parameters

Discount rate	Mortality rates	Labor productivity	Fertility rates
2%	0.09	0.45	0.32
4%	0.06	0.35	0.14
6%	0.04	0.25	0.05
Model r	0.04	0.27	0.06

4 Discussion

Our simulations show that the economic returns to treatments targeting aging that are on the horizon (e.g., those being studied in the TAME trial) are significant and far exceed any reasonable costs of development. In addition, we find that reducing biological age by one year could have very large effects on economic output. We note, again, that this one-year shift is different from extending the human life expectancy by one year. We chose to focus our analysis on the macroeconomic aggregates GDP. While we appreciate humans value their health and leisure, we have narrowed our focus in this way to avoid the uncertainties and additional assumptions necessary to value leisure time and healthspan. Instead, we consider

the quantifiable effects of improved health on the economy. Assigning direct value to health and leisure would result in dramatic increases in the potential benefits of targeting biological aging.

In our discussion on productivity, we focus on aggregate production (i.e., total GDP) because we want to be sure to value the additional life-years gained from increases in longevity and from new births. Similar to the theoretical value of a statistical life (VSL) literature and quality adjusted life year (QALY) literature, our approach includes a life cycle microeconomic model of individual behavior that incorporates both optimization over consumption, savings, labor supply, and leisure. But we also include the effects of many general equilibrium life cycle households in our macroeconomic model to capture the aggregate effect of our simulated changes across a long forecast time series. Thus, instead of asking what happens to total output, we might ask how the circumstances of any given household changes due to better targeting of aging. GDP per capita provides a reasonable measure of the well-being of each household. Under the TAME trial simulation, GDP per capita becomes about 0.04 percent higher in the long run (a difference of less than $300 per capita). With a one-year reduction in biological age for those aged over forty, GDP per capita would be about 0.37 percentage points higher in the long run (a difference of about a few thousand dollars per capita). When we look at changes to mortality or fertility only, while there are positive aggregate impacts, the effects on annual per capita income are (slightly) negative.[13] Productivity gains, as expected, have the largest effects on GDP per capita, since they directly impact the amount of output (and therefore income) each worker can produce in each year of life after age 40. Indeed, productivity gains are the promise of research on aging biology, where the goal is not to merely extend life expectancy, but healthy lifespan.

As we decompose the relative contributions of each lever in Section 3.3, we can make some inferences about policy priorities when incentivizing aging research. In particular, the elasticities of GDP with respect to the parameters suggest that over a medium-term horizon, investments in preventing the cognitive and physical decline that come with aging would produce the largest returns out of perhaps all R&D investments. Over the longer run,

[13]Note that with decreases in mortality rates there is an increase in expected lifetime income, even if average income in some year of life are lower.

Table 5: Percentage Changes in GDP per Capita Relative to Baseline

Year	TAME trial	One Year, mortality	One Year, productivity	One Year, fertility	One Year, All
2025	0.00	0.02	-0.05	-0.01	-0.07
2050	0.06	-0.35	1.11	-0.03	0.75
2075	0.04	-0.65	1.20	-0.17	0.39
2100	0.03	-0.76	1.18	-0.06	0.37

investments in treating age-related fertility decline have the largest relative payoff, if one considers a one-year shift. However, there are likely to be diminishing returns to fertility from slowing reproductive aging. Of course, the only way to know which research area offers the highest quantifiable return on investment using our model would be to know for certain how much funding its success would demand.

5 Conclusion

We provide simulations showing the economic impacts of improvements in biological aging. We consider both the anticipated effects of an existing trial, as well as a hypothetical scenario of delaying aging by one year. Our findings corroborate the estimates and conclusions of Goldman et al. (2013), Murphy and Topel (2006), as well as Scott et al. (2021), that investments in improving aging have exceptionally high returns, even relative to other excellent investments (e.g., cancer treatments). For instance, if all cancers could be prevented or cured, this would add only about three years to life expectancy.[14] This is because the median age for a cancer diagnosis is 66, and at that age, patients are likely to be diagnosed soon after with a different condition of aging, like heart disease, osteoporosis, type II diabetes, cognitive impairment, or a broken rib.

In our simulation results, we focus on the effects of improved aging on GDP and find that delaying aging by one year would increase GDP by about $50 trillion in NPV over the next 100 years. Over the two decades after these effects fully manifest, GDP would be about $840 billion higher per year. Using the expected results of the TAME trial, we find increases

[14] See Goldman et al. (2013).

on the order of $3 trillion, or $68 billion per year in the near term.

As we decompose the contributions of delayed aging on mortality, productivity, and fertility, we find that productivity has the largest effects in the near term and long run. Under different discount factors, the relative strengths of each of these three levers manifest differently. As noted in Section 4, our approach has some limitations. Using the net present value of the change in GDP as our measure of value excludes the direct individual utility value of leisure and healthspan. This approach means that our value estimates may be best considered as lower bounds for the true value of these possible medical breakthroughs.

Similar to the medical value estimate research in Murphy and Topel (2006) and Budish et al. (2015), we estimate the value of health effects to the United States. An extension to this research would be to quantify the global impacts of improvements in biological aging. This would involve country-specific calibration of the macroeconomic model and would highlight heterogeneity in the impacts of improved aging across countries with low and high incomes and different demographic trajectories.

As Becker et al. (2005) show, longevity is not merely a convenience, but is a major determinant in different countries' economic health. Nordhaus (2005) notes, "the economic value of increases in longevity in the last hundred years is about as large as the value of measured growth [in every other part of the economy]." Yet market failures in medical markets have thus far made it difficult to bring to bear significant resources to target aging and longevity. Our study quantifies potential benefits to the US economy that could be available through new technologies that improve biological aging.

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