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Health Dynamics Shape Life-Cycle Incomes

Abstract

This paper empirically investigates the long-run effects of major health improvements on income growth in the United States. To isolate exogenous changes in health, the econometric model uses quasi-experimental variation in cardiovascular disease mortality across states over time. The results show that there is a causal link between health and income per person, and they provide novel evidence that health dynamics shape life-cycle incomes. Compared to previous generations, life-cycle income profiles slope more strongly at the beginning and at the end of work life, such that age becomes a more prominent determinant of income dynamics. The channels for this transformation include better health, higher educational attainment, and changing labor supply.

JEL-Codes: I150, J110, J240, J310, O400.

Keywords: age, mortality, life expectancy, productivity, education, labor supply.

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1 Introduction

Improving the health of the population is an important objective of social policy. While economists agree that rising aggregate income improves health, they still debate over whether improving health reversely spurs aggregate income. Such a causal link is conceivable if health affects incomes by raising worker productivity, educational attainment, or labor supply (for example, Ben-Porath, 1967). While some studies provide evidence for a causal link between health and income (Weil, 2007; Aghion et al., 2011; Cervellati and Sunde, 2011; Bloom et al., 2014), other studies contest this link (Acemoglu and Johnson, 2007, 2014; Hansen, 2014; Hansen and Lønstrup, 2015). This debate focuses on the health effects for the entire population and, thereby, neglects age. Given that the workforce ages and as the susceptibility to diseases changes with age, this focus overlooks how health improvements shape incomes over the life cycle, how measurement of health at various ages affects outcomes, and which economic factors impact incomes at different ages.

This paper investigates the long-run effects of health dynamics—that is, major improvements in health over time—on income growth in the United States. In particular, I examine the causal link between health and income for the entire workforce, and I will then test for heterogeneity in the effects of health dynamics on incomes over the life cycle. I exploit large improvements in health, proxied by life expectancy, which arrive through medical advance in treating cardiovascular diseases and public health campaigns starting in the 1960s. The subsequent sharp decline in cardiovascular disease mortality accounts for 70 percent of the increase in life expectancy between 1960 and 2000 (Cutler et al., 2006, p. 104). Thanks to its salient impact on public health, epidemiologists refer to this decline as the cardiovascular revolution (Foege, 1987).

The cardiovascular revolution has provided me with a quasi-experimental variation to isolate exogenous changes in health across states over time. How strongly medical advance in treating cardiovascular diseases impacts a state's life expectancy depends on the initial prevalence of these diseases within the state. The empirical model combines the differences in cardiovascular disease mortality before the cardiovascular revolution with the unexpected medical advance in treating these diseases between 1960 and 1970 to predict the mortality decline for each state. The model then uses these predictions as instruments for changes in life expectancy. Conceptually, this approach follows the empirical strategy by Acemoglu and Johnson (2007) and Bleakley (2007).

My results show that health improvements boost aggregate incomes: an increase in life expectancy by 1 percent raises income per person by 1.7 percent. In total, the health dynamics of the cardiovascular revolution account for an increase in income of 13 percent compared to 1960, and thus explain one-quarter of the income growth between 1960 and 2000. These health effects differ qualitatively and quantitatively across age cohorts. Life expectancy positively affects income per person at prime working ages between 25 and 54, whereas it exerts no or even has a negative effect on income per person under age 24 and over age 55. It is important for the estimation results at which age life expectancy is measured because the cardiovascular revolution predominantly improves adult health. In particular, the results overstate the health effects on income for proxies that understate the age-specific changes in life expectancy and they understate the health effects on income for proxies that overstate the age-specific changes in life expectancy. The cardiovascular revolution directly shapes life-cycle incomes by improving individual health, and it indirectly shapes them by raising educational attainment and by changing labor supply.

This paper contributes to the literature in several ways. It most closely relates to work by Hansen and Strulik (2017), who are the first to use variation from the cardiovascular revolution to show a causal link between health and college enrollment in the United States. In contrast to their work, I analyze how health dynamics shape life-cycle incomes and which factors explain this process. My results indicate that educational attainment is one channel through which health dynamics raise incomes, and thus complement their results.[1]

The results in this paper show that health improvements spur income per person in the long run and thus provide a further piece of evidence for a causal link running from health to aggregate income. My evidence for positive effects of adult health on incomes, education, and labor supply complements evidence by Bleakley (2007) and Bhalotra and Venkataramani (2015) for positive effects of childhood health on education, income, and labor supply following medical advances and health interventions in the United States during the first half of the twentieth century—and, more generally, evidence from the microeconomic literature that stresses the importance of childhood health for economic outcomes at adult age (see Currie, 2009, and Almond et al., 2018, for a review). In addition, my results concur with the empirical finding of Lorentzen et al. (2008) that high mortality impairs economic development.

Moreover, this paper demonstrates that the age at which health conditions are measured matters for the estimation results. Many studies use a summary measure of health (for example, life expectancy at birth) to investigate its effects on income. However, if this measure overstates the changes in health of the workforce, then the estimation results understate the health effects on income. Consider, for example, the third stage of the epidemiological transition, which is characterized by a sharp decline of infectious disease mortality. During this transition, life expectancy at birth primarily increases due to reductions in infant and child mortality (WHO, 2008). Therefore, using the change in life expectancy at birth to measure improvements in adult health produces estimates that understate the effects on income. Hence, this finding suggests more pronounced health effects on aggregate income for published work (for example, Acemoglu and Johnson, 2007, 2014; Cervellati and Sunde, 2011; Bloom et al., 2014; Hansen, 2014).

Furthermore, this paper provides novel evidence for transformations of life-cycle incomes for the entire workforce over time. Following the cardiovascular revolution, life-cycle income profiles slope more strongly at both the beginning and at the end of work life. Compared to previous generations, age thus becomes a more prominent determinant of income dynamics. However, the average age of productivity peaks does not shift. This evidence extends previous research that shows shifts of productivity peaks toward higher ages if measured by scientific breakthroughs of researchers (Jones and Weinberg, 2011), and toward younger ages if measured by skills in information and communication technologies (Falck et al., 2016). Together, this evidence suggests that shifts in productivity peaks do not confine potential adverse effects of an aging workforce. Hence, this paper sheds new light on results of research on the effects of demographic change for economic development (Feyrer, 2007; Sánchez-Romero, 2013; Kotschy and Sunde, 2018).

[1] In ancillary results, Hansen and Strulik (2017) find no effect of health on income per person aged 30–65 years. Their results are surprising because the salient increase in college education that they document does not manifest in higher incomes, despite the growing college wage premium (Card and Lemieux, 2001). The difference in results arises because the health effects of the cardiovascular revolution significantly differ across age cohorts, as I document in this paper, and also because I omit the war decade for which state differences in military drafting affect labor supply and incomes (Acemoglu et al., 2004).

Finally, this paper shows that health improvements shape life-cycle incomes by raising educational attainment and by changing labor supply over the life cycle without substantially altering lifetime working hours. Therefore, increasing longevity does not require higher lifetime labor supply to boost educational attainment. These findings confirm the theoretical predictions and simulation results of Cervellati and Sunde (2013) and Strulik and Werner (2016), which are based upon Ben-Porath models that incorporate health. These results are also consistent with work by d'Albis et al. (2012), which demonstrates that health improvements during working ages lead to earlier retirement if mortality reductions raise expected lifetime wealth sufficiently.

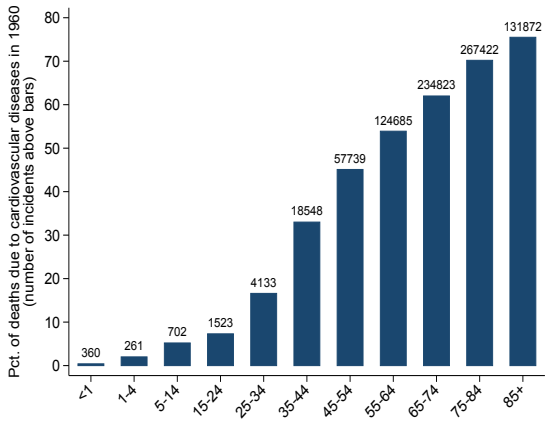
The remainder of this paper is structured as follows. Section 2 presents the background of the health improvements that were made during the cardiovascular revolution. Section 3 introduces the empirical framework and the data. Section 4 presents the estimation results. Finally, Section 5 concludes this paper.

2 Background: The Cardiovascular Revolution

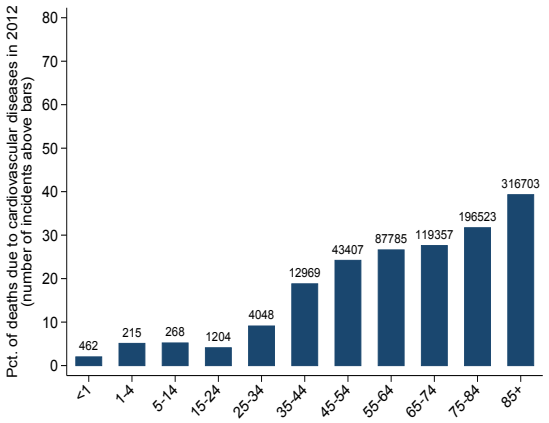
During the second half of the twentieth century, the United States experienced substantial improvements in health that came from medical advances in the treatment of cardiovascular diseases, starting in the 1960s. These improvements led to a pronounced decline in cardiovascular disease mortality, which epidemiologists classify together with mortality reductions from other degenerative diseases as the fourth stage of the epidemiological transition (Olshansky and Ault, 1986; Omran, 1998). Because the aging tissues of the cardiovascular system lose some of their flexibility, the incidence of cardiovascular diseases increases with age (Kirkwood, 2001). Correspondingly, the share of deaths attributable to cardiovascular diseases also increases with age, as illustrated in Panels (a) and (b) of Figure 1.

The cardiovascular revolution considerably reduces mortality rates from a broad spectrum of cardiovascular diseases. For example, in 2000 coronary heart disease still accounted for approximately 12 percent of total deaths in the United States (National Center for Health Statistics, 2017a), and in 2004 was still the most common cause of death in high-income countries (WHO, 2008). Panels (c) and (d) of Figure 1 showcase the size of the mortality decline: between the peak levels in 1968 and the year 2000, mortality from coronary heart disease fell by roughly two-thirds for males and females. As Panels (a) and (b) indicate, the number of incidents drops for all age cohorts, except for infants and individuals over age 85, although the median age of the white population increases from 30.8 to 37.7 years at the same time (Hobbs and Stoops, 2002). This decline is especially pronounced for individuals aged 35–84 years, and thus predominantly boosts adult health and increases longevity. Panels (e) and (f) illustrate this increase in health, measured by life expectancy at age 30, at both the federal and at the state level. During this period, the incidents more than doubled for the age cohort over 85, whereas the overall share of deaths due to cardiovascular diseases halved from almost 80 to below 40 percent. One reason for this is that novel pharmaceuticals and treatments delayed the critical point at which cardiovascular diseases become lethal. Therefore, incidents occur either at a higher age or death originates from other sources, such as cancer.

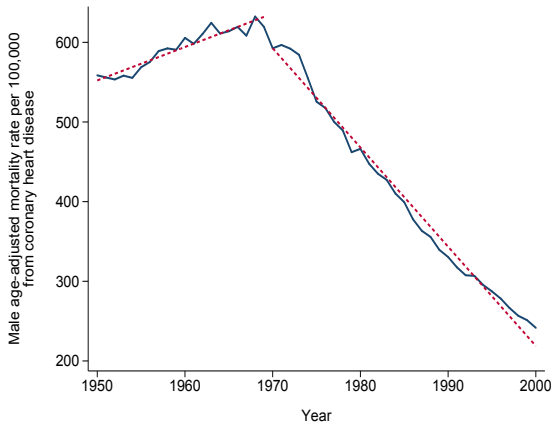
Reductions in cardiovascular disease mortality arrive through two channels. First, seminal medical innovations between 1960 and 1970 enabled physicians to prevent certain diseases or to



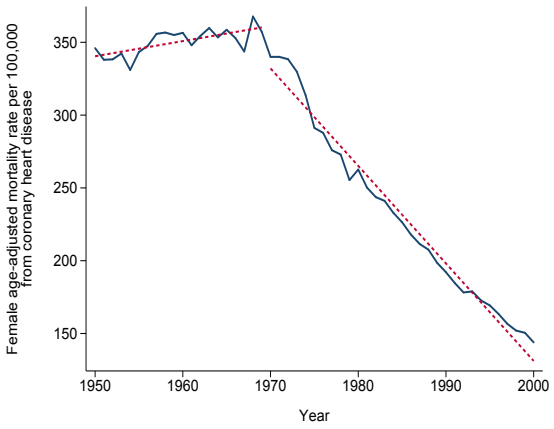
(a) Deaths (%) Due to Cardiovascular Diseases: 1960



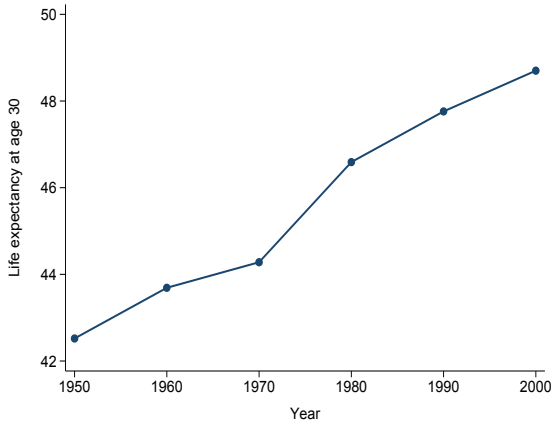
(b) Deaths (%) Due to Cardiovascular Diseases: 2012



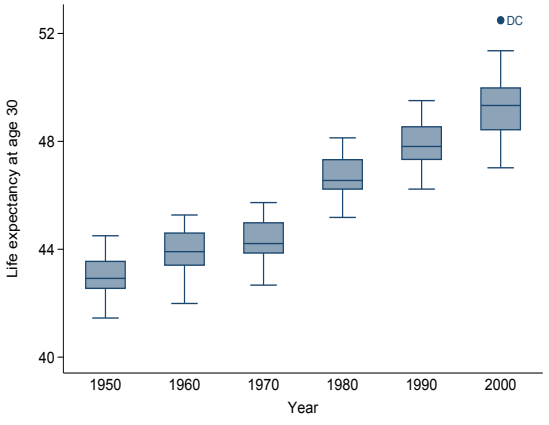
(c) Age-Adjusted Cardiovascular Mortality: Males



(d) Age-Adjusted Cardiovascular Mortality: Females



(e) Life Expectancy at Age 30 in the United States



(f) Life Expectancy at Age 30 Across States

Figure 1: The Cardiovascular Revolution Boosts Adult Health

Data sources: (a) National Center for Health Statistics (1963), (b) National Heart, Lung, and Blood Institute (2012b), (c) and (d) National Heart, Lung, and Blood Institute (2012a), (e) and (f) National Center for Health Statistics (2017b).

treat their symptoms. The most remarkable among these inventions were the artificial cardiac pacemaker, which is first implanted in 1958; the application of chest compression to restore blood circulation of a person in cardiac arrest, which began in 1960; the beta blocker, which was invented in 1962 and is used to lower blood pressure and to treat cardiac arrhythmia; the portable defibrillator, which was invented in 1959 and applied in the United States from 1966 onward; and, the first adult human heart transplantation, which was conducted in 1968. Subsequent innovations include the first thrombolytic therapies in 1986 to treat myocardial infarction, stroke, and pulmonary embolisms; the invention of cholesterol lowering statins, which were first marketed in 1987; and, the implantation of intravascular stents to address acute closure of arteries and blood vessels, which began in 1988. An increasing number of specialists and care centers for cardiovascular diseases have complemented these advances in medical technology (CDC, 1999b).

Second, rising awareness of risk factors and changes in behavior have also led to mortality decline. For example, the research results by Keys et al. (1963), Keys (1980), and Dawber (1980) established high blood cholesterol, high blood pressure, physical inactivity, smoking, obesity, and unbalanced diet as major risk factors for cardiovascular diseases.[2] The Federal Government initiated national programs to educate specialists and the general public about the risks of high blood pressure in 1972; of high blood cholesterol in 1985; and of the importance of cardiovascular health in 1989 (CDC, 1999a). These programs have increased the share of patients with high blood pressure whose condition is treated and under control (CDC, 1999b). Moreover, the report to the Surgeon General in 1964 highlighted the adverse effects of smoking on health (U.S. Department of Health, Education, and Welfare, 1964). To reduce the demand for cigarettes, the Federal Government banned broadcasting advertisement in 1971 and increased the cigarette taxes in the 1980s (CDC, 1999a). Generally, the preventive measures against smoking are successful.[3]

The unexpected surge of medical breakthroughs during the 1960s and the extensive preventive efforts thereafter separate the sample into a pre-treatment period that lasted until 1960 (before the cardiovascular revolution) and a post-treatment period from 1970 onward (the cardiovascular revolution). The next section explains how the empirical strategy uses variation from the cardiovascular revolution to identify the effect of health on economic outcomes.

3 Empirical Framework and Data

3.1 Empirical Framework

This section introduces the empirical framework to study the effect of health improvements on economic outcomes. The outcome equation is

$$y_{s,t} = \alpha x_{s,t} + \sum_{\tau \in \mathcal{T}} w_s' d_t^{\tau} \beta_\tau + \gamma_s + \delta_t + \varepsilon_{s,t}, \tag{1}$$

where $y_{s,t}$ denotes the outcome of interest (for example, log income per person) for state s and

[2] According to Ezzati and Riboli (2012, p. 1485), high blood pressure and high blood cholesterol alone account for one-half of the global incidence of coronary heart disease. Too high body weight and smoking are responsible for another 20 and 13 percent, respectively.

[3] Figure A2 in the Appendix shows changes in smoking behavior. The population share of smokers started to decline in the middle of the 1960s and per capita cigarette consumption began to fall during the 1970s.

time period t; $x_{s,t}$ represents health, proxied by log life expectancy; w_s is a vector of controls, which are measured in 1960 and interacted with a full set of time dummies d_t^τ, $\tau \in \mathcal{T}$, using the year 1960 as reference category; γ_s and δ_t denote state-fixed and time-fixed effects; and $\varepsilon_{s,t}$ is the error term. The analysis focuses on the parameter α, which captures the importance of health dynamics for economic outcomes.

Because of omitted variables and reverse causality, log life expectancy might be endogenous. To isolate exogenous changes in health over time, the empirical model exploits variation in the prevalence of cardiovascular diseases across states in 1960 (before the cardiovascular revolution), and combines them with the timing of medical advances between 1960 and 1970 to predict the mortality decline for each state. The model uses these predictions as instrument for changes in log life expectancy. The corresponding first-stage model is given by

$$x_{s,t} = \sum_{\tau \in \mathcal{T}} \eta_\tau z_s d_t^\tau + \sum_{\tau \in \mathcal{T}} w_s' d_t^\tau \theta_\tau + \kappa_s + \lambda_t + \xi_{s,t}, \tag{2}$$

where cardiovascular disease mortality in 1960, z_s, is interacted with a full set of time dummies d_t^τ using the year 1960 as reference category; κ_s and λ_t denote state-fixed and time-fixed effects; and $\xi_{s,t}$ is the error term.

Conceptually, the first stage uses variation in the predicted decline of cardiovascular disease mortality, which derives from the initial cardiovascular disease mortality across states. The first stage compares these differences in the predicted mortality decline between the pre-treatment and post-treatment period to differences in the increase of (log) life expectancy between the pre-treatment and post-treatment period. Hence, the first stage corresponds to a differences-in-differences model, in which all states are treated with the cardiovascular revolution but with different treatment intensities. Therefore, the first stage has a natural interpretation in this context: a decline in cardiovascular disease mortality initiates an increase in life expectancy, which affects economic outcomes.

Several conditions must be fulfilled if the predicted mortality decline is to be a valid instrument for health. First, initial cardiovascular disease mortality must be (quasi-)randomly assigned conditional on covariates. This assumption requires that the instrument is independent of potential outcomes and potential treatment assignments given the complete set of covariates. Notably, there are distinct geographic differences in the initial prevalence of cardiovascular diseases across states, as shown in Figure 2. These differences—which are mainly rooted in social, cultural, and environmental factors (CDC, 1999b)—determine how beneficial the cardiovascular revolution is for each state: the higher the initial mortality, the larger the potential health improvement through the cardiovascular revolution.[4] To capture potential non-random variation in initial cardiovascular disease mortality, the baseline model adds controls to take up state-level selection in the adoption of health technologies that results from initial disparities in income or educational attainment. In particular, these controls account for the possibility of causal links between income and health or between education and health, which may both affect the initial health gradient across states and its subsequent trajectory.[5] Following the suggestions of Aghion

[4]The geographic distribution of life expectancy at age 30 is a mirror image of the distribution of cardiovascular disease mortality, as shown in Figure A1 in the Appendix.

[5]See, for example, Ettner (1996), Frijters et al. (2005), Lindahl (2005), and Chetty et al. (2016) for evidence

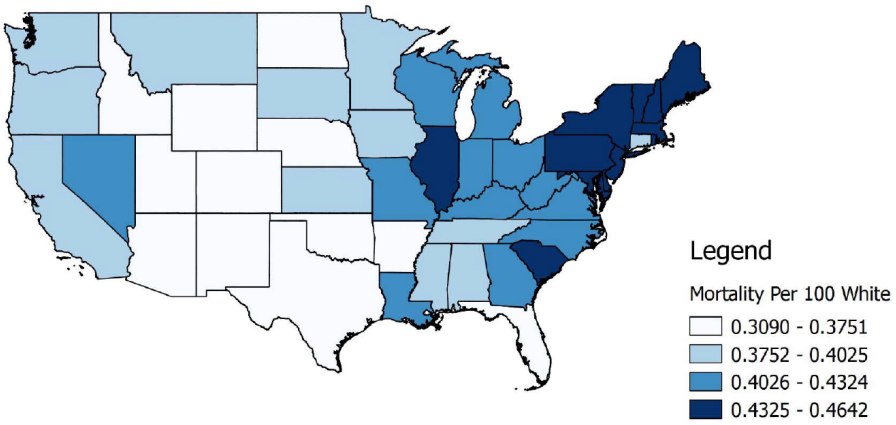


Figure 2: Geographic Dispersion of Cardiovascular Mortality in 1960

Data source: Grove and Hetzel (1968).

et al. (2011) and Bloom et al. (2014), the model further controls for the health gradient before the cardiovascular revolution, which may itself affect subsequent health dynamics. Specifically, the model follows the approach by Hansen and Strulik (2017) and adds non-cardiovascular disease mortality as a measure of the initial health gradient. Importantly, the empirical model does not include current values of the controls because they are themselves affected by the cardiovascular revolution and thus constitute bad controls. Finally, state-fixed and time-fixed effects eliminate systematic variation, due to factors that do not vary concomitantly over states and time.

Second, cardiovascular disease mortality must only affect outcomes through the channel of health in the first stage. This exclusion restriction is untestable. It would be violated if changes in cardiovascular disease mortality were to affect the outcome of interest through a channel other than health as measured by life expectancy. However, as the instrument is specific to the channel of health on the aggregate level, this assumption should plausibly be fulfilled in the context under consideration. Moreover, the empirical model accounts for initial disparities in non-cardiovascular disease mortality to prevent the instrument from taking up beneficial effects attributable to medical advance in the treatment of other diseases taking place at the same time.

Third, the identification strategy requires that pre-treatment trends in health and economic outcomes are parallel given the set of covariates. The empirical model allows me to test this assumption through interactions between initial cardiovascular disease mortality with the full set of time-fixed effects in the first-stage and in the reduced-form model.

Fourth, the predicted mortality instrument must sufficiently correlate with changes in life expectancy. The first-stage F-test assesses this assumption.

Given these assumptions, I use two-stage least squares (2SLS) to estimate two variants of the empirical model. The first variant follows the tradition of the macroeconomic literature and computes parameters using long differences based on the observations at the beginning and at the end of the treatment period to derive the long-run effects of health dynamics on economic outcomes (see, for example, Acemoglu and Johnson, 2007). The second variant uses all available observations in a panel to obtain additional insights regarding the timing of the health effects.

on a causal link between income and health, and Grossman and Kaestner (1997) and Lleras-Muney (2005) for evidence on a causal link between education and health.

3.2 Data

The dataset is a balanced panel of the 48 contiguous US states ranging over the period 1950–2000 in 10-year intervals; hence, the estimation sample comprises 288 observations in total. I exclude Alaska and Hawaii because of missing data for early periods, and the District of Columbia because of its special nature of a metropolitan region. Given that data on life expectancy are in early periods only available for whites, I restrict the sample to the white population. Table A1 in the Appendix reports descriptive statistics.[6]

Data on gross wage income, gross total income, labor supply, and educational attainment stem from individual data in decennial US censuses (IPUMS) by Ruggles et al. (2015). I use the urban Consumer Price Index by the Bureau of Labor Statistics to adjust wage income and total income for inflation. While wage income comprises wages, salaries, commissions, cash bonuses, tips, and other money income received from an employer, total income comprises all possible sources of income including also social benefits and different forms of capital incomes. Labor supply covers individual labor force participation, usual hours worked per week, and usual weeks worked per year. Working weeks are not available as a continuous measure in 1960 and 1970, whereas the series on usual working hours only starts in 1980. However, the intervalled hours and weeks are available throughout all time periods. Consequently, I construct a continuous measure for weeks and hours from bivariate regressions of the continuous on the intervalled measure. For the cohort-specific analysis, I use age-specific regressions to construct hours and weeks. I proxy initial educational attainment by the share of college graduates in the population based on the the number of individuals who attend at least four years of college in their life. I collapse the data to the state level using person-sample weights to ensure representativity. Due to the collapsing, incomes, education, and labor supply are grouped on the state level. For this reason, I weight all regressions by group size; that is, the initial population of a specific age cohort or the total population.[7] Population counts are from Hobbs and Stoops (2002).

The data on life expectancy are drawn from the US life tables and vital statistics provided by the National Center for Health Statistics (2017b). Notably, life expectancy in 1960 differs considerably across states. For example, life expectancy at age 30 varies by 2.94 years between North Dakota (the state with the highest value) and Nevada (the state with the lowest value). Moreover, life expectancy is high in the western central states, whereas it is comparatively low in New England and most Atlantic states. As the median age of the population increases from 30.8 to 37.7 years over the observation period, it is not clear at which age life expectancy should be measured to optimally capture the health dynamics over all age cohorts. Therefore, I proceed as follows: I construct a measure of adult life expectancy based on the arithmetic mean of life expectancy measured at ages 20 through 60 and show that the results are robust to different measures of life expectancy. I further discuss the role of measurement aspects in Section 4.2.

[6]Data on wage income and life expectancy are available as early as 1940. The sample starts in 1950 because differential mobilization rates across states during World War II temporarily increase female labor supply. This temporary increase affects both male and female earnings during the war decade but not thereafter (Acemoglu et al., 2004, p. 505).

[7]Because the population equation of interest is the effect of improved health on economic outcomes, weighting the regression equation by the group size yields estimation results that are closer to the micro data than unweighted averages (see, for example, Angrist and Pischke, 2009). Nevertheless, I also report unweighted estimates in the robustness section.

Age-adjusted mortality rates are obtained from Grove and Hetzel (1968) and expressed in deaths per 100 persons.[8] Between 1960 and 2000, average cardiovascular disease mortality declines by roughly 50 percent from 0.4 to 0.2 deaths per 100 persons. Importantly, life expectancy and mortality rates provide a conservative view on health improvements because they cannot fully capture the morbidity reductions following the cardiovascular revolution. In the absence of better health measures across states and time, they represent the best option available.

In Section 4.4, I use data from the Health and Retirement Study (HRS, 2017) to examine whether health gains directly contribute to income growth. This dataset provides representative, individual longitudinal data on income and health status for more than 20,000 people over age 50 in the United States. Data on income comprise wages, salaries, bonuses, overtime pay, commissions, tips, second jobs, military reserve earnings, professional practice, or trade income, and refer to the previous year. I adjust incomes for inflation.

4 Results

4.1 Health Dynamics Boost Incomes in the Long Run

The empirical analysis begins by examining the effect of health improvements on income per person aged 15–64 years. Given that health is endogenous to economic factors for reasons of omitted variables and reverse causality, the analysis concentrates on the instrumental variables model.[9] Table 1 presents results for a long-differences model, which estimates the long-run impact of changes in log life expectancy on log income per person. This specification uses two observations per state, one for the pre-treatment period before the cardiovascular revolution in 1960 and one for the final post-treatment period in 2000. The model instruments log life expectancy by cardiovascular disease mortality interacted with a post-1960 dummy, which indicates treatment through the cardiovascular revolution. The table reports results for the second stage in Panel (a), results for the reduced form in Panel (b), and results for the first stage in Panel (c). In Panels (a) and (b), the dependent variable is log wage income per person in the first three columns and log total income per person in the last three columns. In Panel (c), the dependent variable is log life expectancy. All estimates are weighted by the population in 1960.

Columns (1) and (4) show estimates for a parsimonious model without controls except state-fixed and time-fixed effects. The second stage and the reduced form reveal a negative but insignificant correlation between health and incomes. Low income growth associates with high life expectancy and a large predicted decline in cardiovascular disease mortality. In contrast, the first stage shows a positive and significant correlation between a large predicted decline in cardiovascular disease mortality and high life expectancy.

The parsimonious specifications do not rule out that initial disparities in education or income affect the initial health gradient across states, the subsequent adoption of health technologies, and the growth of income per person. This omission results in downward-biased estimates if,

[8] Age-adjustment allows me to compare the changes in mortality rates between states even if they have different age structures. Due to the adjustment, the mortality rates cannot be interpreted as crude rates, unless a state has the same age structure as the standard population. Therefore, the absolute values of cardiovascular disease mortality are not of importance but their relative changes over time are.

[9] Table A2 in the Appendix shows results for ordinary least squares, which produces biased estimates.

Table 1: Effect of Adult Health on Income Per Person: Long Differences 1960–2000

	Log wage income per person 15–64			Log total income per person 15–64		
	(1)	(2)	(3)	(4)	(5)	(6)
	(a) Second stage (2SLS)					
Log life expectancy	-1.23	1.94**	1.65**	-0.44	2.07**	1.75**
	(1.00)	(0.88)	(0.81)	(0.88)	(0.89)	(0.82)
First-stage F–statistic	17.2	22.4	20.1	17.2	24.4	22.1
	(b) Reduced form					
Mortality × Post 1960	-0.52	0.74**	0.62**	-0.19	0.79***	0.66**
	(0.39)	(0.29)	(0.28)	(0.37)	(0.28)	(0.27)
	(c) First stage					
Mortality × Post 1960	0.43***	0.38***	0.38***	0.43***	0.38***	0.38***
	(0.10)	(0.08)	(0.08)	(0.10)	(0.08)	(0.08)
Controls in 1960 × Post 1960:						
Initial share college	—	✓	✓	—	✓	✓
Initial income	—	✓	✓	—	✓	✓
Initial mortality	—	—	✓	—	—	✓
States	48	48	48	48	48	48
Observations	96	96	96	96	96	96

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. In Panels (a) and (b), the dependent variable is log wage income per person aged 15–64 (Columns 1 to 3) and log total income per person aged 15–64 (Columns 4 to 6). In Panel (c), the dependent variable is log life expectancy. Estimates are weighted by the population in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

for example, education or income in 1960 reduce the scope for subsequent improvements in health, education, and income through a reverse causal link with health. To address this concern, the specifications in Columns (2) and (5) additionally control for the initial share of college graduates and initial income, both measured in 1960 and interacted with the post-1960 treatment dummy. The resulting second-stage and reduced-form estimates are positive and significant at the 5-percent level. The first-stage estimates slightly reduce in magnitude but remain positive and significant at the 1-percent level. Large health improvements through the cardiovascular revolution thus correlate with large income growth.

Columns (3) and (6) present full specifications that further control for the health gradient before the cardiovascular revolution by adding an interaction between initial non-cardiovascular disease mortality and the post-1960 treatment dummy. Including this variable accounts for any effect of the initial health gradient on the subsequent health dynamics. Moreover, it prevents the mortality instrument from taking up beneficial effects of medical innovations that operate through health channels other than the cardiovascular revolution. Conditional on the set of covariates, the instrument exploits quasi-natural variation, such that the estimates of the full specifications have a quantitative interpretation.

The health dynamics of the cardiovascular revolution boost aggregate income. The results show a quantitatively similar effect of health on wage income per person and total income per person. An increase in life expectancy by one percent leads to an income increase of 1.7 percent for the average worker. The first-stage estimates take a value of 0.38. Between 1960 and 2000, the cardiovascular revolution halves cardiovascular disease mortality from 0.4 to 0.2 per 100 persons and thus raises adult life expectancy by 2.68 years.[10] This estimate is reasonably close to the increase in life expectancy at birth of 3.27 years that Cutler et al. (2006, p. 104) attribute to medical advances in treating cardiovascular diseases between 1960 and 2000. Combining first-stage and second-stage estimates, the rise in life expectancy between 1960 and 2000 predicts an increase in income per person of 13 percent compared to the year 1960. This increase explains 24 percent of overall wage growth and 27 percent of total income growth over this time period.[11] Importantly, this number comprises not only direct income gains through better health but also indirect income gains (for example, through higher education). All estimates are significant at the 5-percent level. Values of the first-stage F-statistic around 20 indicate a strong instrument considering the conventional threshold of 10.

Table 2 reports the results for panel specifications, which use all time periods from 1950 to 2000. The model instruments life expectancy by initial cardiovascular disease mortality interacted with a full set of year dummies, in which the year 1960 is the reference category and is thus omitted. This model reveals insights regarding the timing of the health effects in the first stage and in the reduced form. For example, the interactions between initial cardiovascular disease mortality and the dummy for the year 2000 provide estimates for the difference between the years 1960 and 2000, so that the corresponding estimates are identical to those of the long-difference specifications in Table 1. Analogously, the interactions between initial cardiovascular disease mortality and the dummy for the year 1950 provide estimates for the difference between the years 1950 and 1960, which enable me to test the parallel trends assumption in the pre-treatment period. Overall, the panel structure allows me to examine the short-run and long-run effects of health improvements on economic outcomes. The table reports results for the second stage in Panel (a), results for the reduced form in Panel (b), and results for the first stage in Panel (c). In Panels (a) and (b), the dependent variable is log wage income per person in the first three columns and log total income per person in the last three columns. In Panel (c), the dependent variable is log life expectancy. All estimates are weighted by the population in 1960.

The results qualitatively confirm those of the long-difference model and show a positive link between health and aggregate income. Quantitatively, the second-stage estimates are slightly smaller and less precise, such that the estimates are marginally insignificant in the full specifications in Columns (3) and (6). However, the reduced-form estimates indicate that this loss in precision mainly arrives through the timing of the health effects. For the years 1970 and 1980, the effect of the predicted mortality decline on incomes is small and only positive and

[10] To arrive at this number, compute $\Delta_x = \widehat{\eta} \cdot \Delta_z \cdot \mu_x^{1960} = 0.38 \cdot 0.20 \cdot 35.26 \approx 2.68$, where μ_x^{1960} denotes the sample mean of life expectancy evaluated in 1960.

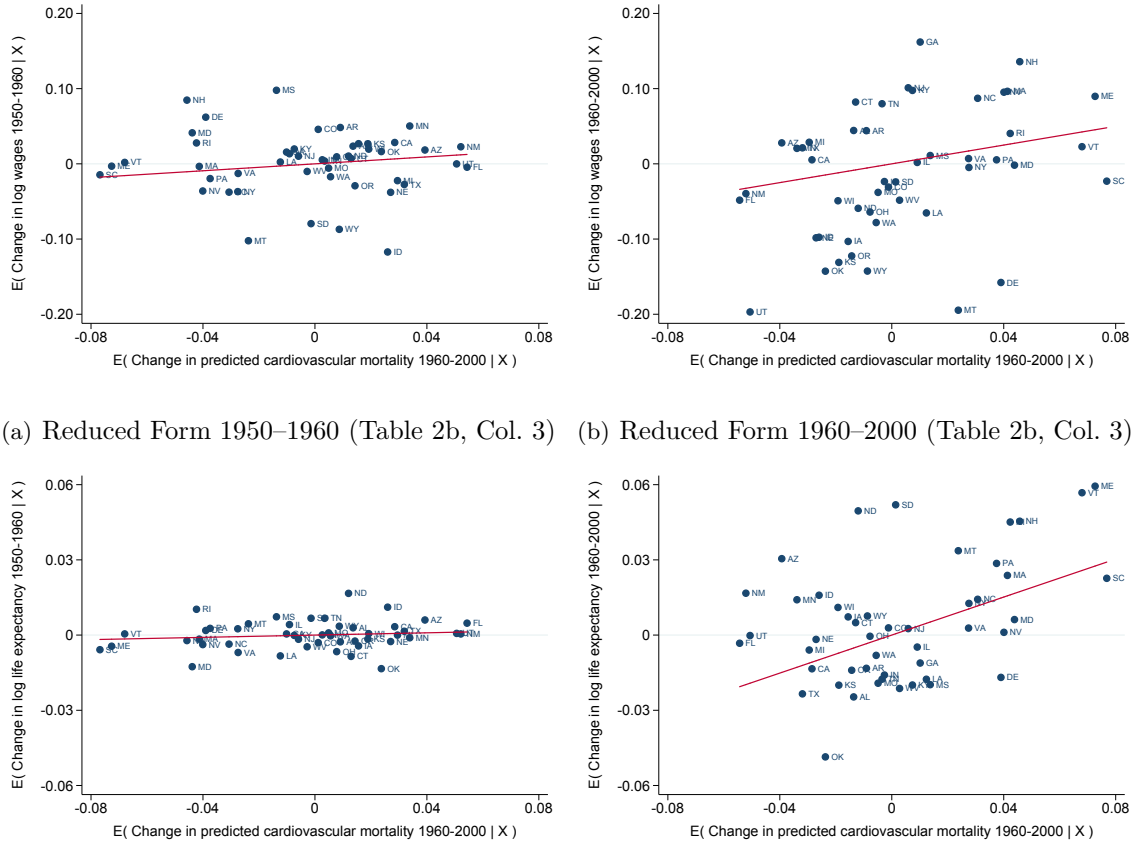
[11] To arrive at the numbers for wage income, compute $\Delta_y = \widehat{\alpha} \cdot \Delta_x / \mu_x^{1960} \cdot \mu_y \approx 1.65 \cdot 2.68 / 35.26 \cdot 26454.36 \approx 3317.67$ with $\Delta_y / \mu_y^{1960} \approx \frac{3317.67}{26454.36} \approx 0.13$ and $\Delta_y / (\mu_y^{2000} - \mu_y^{1960}) \approx \frac{3317.67}{13547.93} \approx 0.24$, where μ_y^{1960} and μ_y^{2000} denote the sample means evaluated in 1960 and 2000. Analogously, compute $\Delta_y = \widehat{\alpha} \cdot \Delta_x / \mu_x^{1960} \cdot \mu_y \approx 1.65 \cdot 2.68 / 35.26 \cdot 28114.20 \approx 3525.84$ with $\Delta_y / \mu_y^{1960} \approx \frac{3525.84}{28114.20} \approx 0.13$ and $\Delta_y / (\mu_y^{2000} - \mu_y^{1960}) \approx \frac{3525.84}{13284.45} \approx 0.27$ to arrive at the numbers for total income. Similar numbers obtain for the reduced-form estimates. Note, the absolute wage gains are difficult to interpret as incomes are inflation-adjusted and refer to the base year 2010.

Table 2: Effect of Adult Health on Income Per Person: Panel 1950–2000

	Log wage income per person 15–64			Log total income per person 15–64		
	(1)	(2)	(3)	(4)	(5)	(6)
	(a) Second stage (2SLS)					
Log life expectancy	-1.51	1.49*	1.23	-0.68	1.74*	1.47
	(1.02)	(0.89)	(0.85)	(0.93)	(0.97)	(0.92)
First-stage F–statistic	7.5	6.8	6.4	7.5	7.4	6.9
	(b) Reduced form					
Mortality × 1950	0.13	0.23	0.23	0.08	0.16	0.14
	(0.18)	(0.19)	(0.19)	(0.20)	(0.21)	(0.21)
Mortality × 1970	-0.05	0.36*	0.33*	0.08	0.41**	0.36**
	(0.17)	(0.18)	(0.18)	(0.17)	(0.16)	(0.15)
Mortality × 1980	-0.77***	-0.18	-0.20	-0.67**	-0.21	-0.25
	(0.28)	(0.21)	(0.21)	(0.31)	(0.22)	(0.22)
Mortality × 1990	-0.38	0.75***	0.67***	-0.09	0.82***	0.72***
	(0.35)	(0.24)	(0.24)	(0.34)	(0.24)	(0.23)
Mortality × 2000	-0.52	0.74**	0.62**	-0.19	0.79***	0.66**
	(0.39)	(0.29)	(0.28)	(0.37)	(0.28)	(0.27)
	(c) First stage					
Mortality × 1950	-0.02	0.02	0.02	-0.02	0.01	0.01
	(0.03)	(0.03)	(0.03)	(0.03)	(0.02)	(0.02)
Mortality × 1970	0.15***	0.14***	0.13***	0.15***	0.14***	0.13***
	(0.04)	(0.03)	(0.04)	(0.04)	(0.03)	(0.03)
Mortality × 1980	0.23***	0.24***	0.24***	0.23***	0.23***	0.23***
	(0.06)	(0.05)	(0.05)	(0.06)	(0.05)	(0.05)
Mortality × 1990	0.33***	0.33***	0.32***	0.33***	0.32***	0.31***
	(0.06)	(0.07)	(0.07)	(0.06)	(0.07)	(0.07)
Mortality × 2000	0.43***	0.38***	0.38***	0.43***	0.38***	0.38***
	(0.10)	(0.08)	(0.08)	(0.10)	(0.08)	(0.08)
Controls in 1960 × year dummies:						
Initial share college	—	✓	✓	—	✓	✓
Initial income	—	✓	✓	—	✓	✓
Initial mortality	—	—	✓	—	—	✓
States	48	48	48	48	48	48
Observations	288	288	288	288	288	288

Note: This table reports estimates for ten-year panel data over the time period 1950–2000. All regressions include state-fixed and time-fixed effects. In Panels (a) and (b), the dependent variable is log wage income per person aged 15–64 (Columns 1 to 3) and log total income per person aged 15–64 (Columns 4 to 6). In Panel (c), the dependent variable is log life expectancy. Estimates are weighted by the population in 1960. Control variables are measured in 1960 and interacted with year dummies; the year 1960 serves as reference category and is omitted. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

significant for the year 1970. In contrast, the effect is considerably larger, positive, and significant at the 5-percent level for the years 1990 and 2000. Hence, the cardiovascular revolution only unfolds a positive and significant effect on economic outcomes in the long run, whereas the short-run effects are at best modest. Consequently, I will concentrate on the long-run effects for the remainder of the analysis.



(a) Reduced Form 1950–1960 (Table 2b, Col. 3) (b) Reduced Form 1960–2000 (Table 2b, Col. 3)

(c) First Stage 1950–1960 (Table 2c, Col. 3) (d) First Stage 1960–2000 (Table 2c, Col. 3)

Figure 3: Partial Correlations Before and After the Beginning of the Cardiovascular Revolution

The first-stage estimates are positive and significant at the 1-percent level for the post-treatment years 1960–2000 throughout all specifications. A concern might be that the first-stage correlations between the instruments and the endogenous variable are weak because the first-stage F-statistic takes values below 10. To address this concern, I use the heteroskedasticity-robust limited information maximum likelihood (LIML) estimator, which is robust to weak identification in the first stage. The results are very similar to those of the long-difference specifications and they confirm that there is a positive link between health and aggregate incomes.[12]

The panel model further allows me to test whether or not the pre-treatment trends in health and economic outcomes were parallel across states given the set of covariates. This is an indirect test for the identifying assumption that all states would have undergone a similar medical and economic development in the absence of the cardiovascular revolution. This test requires the estimates for the interaction between initial cardiovascular disease mortality and the dummy for the year 1950 to be close to zero and insignificant.

Figure 3 displays pre-treatment trends for the period 1950–1960 and contrasts them with the post-treatment trends for the period 1960–2000; the estimates are obtained from the full specification shown in Column 3 of Table 2, where wage income is the dependent variable. Panels (a) and (c) demonstrate that the predicted mortality instrument has no effect on changes in life

[12]See Table A3 in the Appendix.

expectancy or log wage income per person for the years 1950 to 1960. Hence, the pre-treatment trends are parallel. In contrast, Panels (b) and (d) show a positive effect of the predicted mortality decline on subsequent improvements in life expectancy and log wages after the beginning of the cardiovascular revolution. Single outliers do not drive these results, as the figure reveals.[13]

4.2 The Role of Age: Health Dynamics Shape Life-Cycle Incomes

The results so far provide evidence for a causal link between health improvements and average income of the workforce. Before turning to the health effects for each age cohort, I consider the measurement of health changes in light of the demographic structure of the population—a point that is largely unnoticed by the literature. The convention is to use a summary measure, such as life expectancy, to capture the effects of health changes. By construction, this measure encompasses the expected remaining lifetime at a given age and for all following ages, and thus provides a gross approximation of expected health over the remaining life cycle. Hence, this measure implicitly assigns the same health to everyone in the population of interest. This assumption is restrictive and masks heterogeneous health effects across age cohorts. Furthermore, the proxy need not adequately capture the health improvements for the cohort of interest, and the instrumentation strategy cannot solve this problem.

This deficit becomes clear in light of the cohort structure of the workforce. On average, young workers have good health and can expect to live many more years. In contrast, aging has already eroded some of the older workers' health, such that their expected remaining life years are considerably smaller than those of the young workers. A proxy that predominantly captures the health changes of young workers thus overstates the health changes of older workers, and vice versa. Suppose, for example, that one is interested in estimating the effect of health improvements on income growth of the total workforce. To capture health improvements, one uses the (log-)change in life expectancy at age 40 as a proxy. By assumption, one assigns every worker the expected health improvement of a forty-year-old. This proxy may overstate or understate the actual average health improvement, depending on how the health of the various age cohorts changes.

One way to think about this problem is systematic measurement error.[14] Specifically, one does not use the measure $x_{s,t}$ but

$$p_{s,t} = x_{s,t} + \nu_{s,t}, \tag{3}$$

where $p_{s,t}$ is the observed proxy in the sample and $\nu_{s,t}$ is a measurement error. Whether this proxy correctly captures the average health improvement of the workforce depends on its demographic structure. If the average worker is older than 40, then the proxy assigns a too small health improvement to the workforce, such that proxy $p_{s,t}$ and measurement error $\nu_{s,t}$ correlate negatively. Plugging the expression from (3) into the regression model yields

$$y_{s,t} = \alpha p_{s,t} + \sum_{\tau \in \mathcal{T}} w_s' d_t^\tau \beta_\tau + \gamma_s + \delta_t + \varepsilon_{s,t} - \alpha \nu_{s,t}. \tag{4}$$

[13] Figure A3 confirms these findings for total income as dependent variable.

[14] Note, this approach does not require the health proxy to be mismeasured. It is enough that the incorrect choice of the health proxy introduces a systematic deviation from the true health status.

Define $e_{s,t} = \varepsilon_{s,t} - \alpha\nu_{s,t}$ as the composite error and suppose the proxy $p_{s,t}$ and the idiosyncratic error $\varepsilon_{s,t}$ do not correlate. Given the negative correlation between $p_{s,t}$ and $\nu_{s,t}$, the proxy $p_{s,t}$ and the composite error $e_{s,t}$ correlate positively for $\alpha > 0$ (upward bias) and negatively for $\alpha < 0$ (downward bias). Accordingly, the estimate $\widehat{\alpha}$ is biased away from zero and is too large in absolute terms. If, conversely, the proxy assigns a too large health improvement, then $p_{s,t}$ and $\nu_{s,t}$ correlate positively, such that $p_{s,t}$ and $e_{s,t}$ correlate negatively for $\alpha > 0$ (downward bias) and positively for $\alpha < 0$ (upward bias). Consequently, the estimate $\widehat{\alpha}$ is biased toward zero and too small in absolute terms. In sum, systematic mismeasurement of the health proxy (or incorrect choice of the same) biases the estimate of interest away from zero if the proxy overstates the true health improvements, and it biases the estimate toward zero if the proxy understates the true health improvements. The more that proxy and true health diverge, the more severe this bias is. This problem can be solved by using correct, age-specific measures of health.

Table 3 presents the effects of changes in log life expectancy on log wage income by age cohorts in Panel (a) and the effects of changes in log life expectancy on log total incomes by age cohorts in Panel (b). Columns (1) to (5) report results for age cohorts in 10-year brackets from 15 up to 64, Column (6) reports estimates for the workforce aged 15–64, and Column (7) reports estimates for workers over age 65. The age-specific estimates are weighted by the initial population of their specific age cohort, the estimates for the workforce aged 15–64 are weighted by the entire population in 1960.[15]

Notably, health unfolds qualitatively and quantitatively different effects on income per person across age cohorts. Life expectancy positively affects average wages and incomes of prime-age workers aged 25–54 years, whereas it exerts no or even a negative effect on average incomes for workers under age 24 and over age 55. The health effects are largest for the age cohort 25–34 and decrease from this age onward. Overall, the estimates are slightly more pronounced for total income per person in Panel (b). High values of the F-statistics document a strong first-stage correlation between life expectancy and the instrument throughout all specifications. The estimates are statistically significant at conventional levels for workers aged 15–44 and the overall workforce in Panel (a) and for all age cohorts except the cohort aged 55–64 in Panel (b). Moreover, the positive estimates for prime-age workers significantly differ from the negative estimates for the workers under age 24 and over age 55. Hence, the results reveal meaningful heterogeneity with respect to the health effects across age cohorts. Following the cardiovascular revolution, life-cycle income profiles slope more strongly at the beginning and the end of the standard work life. Compared to previous generations, age thus becomes a more prominent determinant of income dynamics. Nevertheless, the age at which life-cycle productivity peaks remains roughly stable. In 1960, this age lies between 40 and 50. While the large health estimates for young working ages suggest a shift of productivity peaks toward younger ages, the health improvements are larger for older working ages, such that both effects balance each other. Hence, the health improvements of the cardiovascular revolution do not compensate potential adverse effects of an aging workforce by shifting the age of productivity peaks.

Table 4 replicates the analysis from Table 3(a) but uses the same proxy of health for all age cohorts instead of age-specific measures. Using an inaccurate proxy for health improvements

[15] Table A5 in the Appendix reports the corresponding reduced-form and first-stage estimates. Table A6 in the Appendix presents results for ordinary least squares.

Table 3: Effect of Adult Health on Income Per Person by Age Cohort

	Long-difference specifications: 1960–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Log wage income per person in age cohort						
Log life expectancy	-3.29**	4.59***	2.49***	0.96	-0.21	1.65**	-0.36
(of specific age cohort)	(1.51)	(1.25)	(0.82)	(0.63)	(0.45)	(0.81)	(0.62)
First-stage F–statistic	17.3	17.5	18.2	18.6	22.2	20.1	18.5
	(b) Log total income per person in age cohort						
Log life expectancy	-3.65***	4.28***	2.67***	1.09*	0.09	1.75**	-0.95**
(of specific age cohort)	(1.38)	(1.16)	(0.86)	(0.60)	(0.46)	(0.82)	(0.45)
First-stage F–statistic	17.9	19.0	19.8	28.6	24.8	22.1	20.2
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage income per person in a specific age cohort in Panel (a) and log total income per person in a specific age cohort in Panel (b). Estimates are weighted by the initial population of the corresponding age cohort in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

leads to biased estimates if the proxy does not closely reflect the health changes of the population of interest. In its lower part, Table 4 reports changes in life expectancy between the years 1960 and 2000 for every age cohort and the total workforce in percent. These health improvements amount to 9.8 percent if measured at birth and average 13.3 percent for the workforce aged 15–64. Their size increases with age: life expectancy at age 65 increases almost three times as much as life expectancy at age 20. In Panel (a), life expectancy at age 40 proxies health in all specifications. This proxy choice produces estimates that are too large in magnitude for all age cohorts whose health improvements are overstated (workers aged 15–34 and the total workforce) and estimates that are too small in magnitude for age cohorts whose health improvements are understated (all workers over age 45). The differences are sizable and amount up to 75 percent of the estimates for the adequate proxies in Table 3(a).

Panel (b) repeats this exercise for life expectancy at birth, which is the most common health proxy in cross-country studies. Changes in life expectancy at birth understate the health improvements of the cardiovascular revolution for all age cohorts over 25. Thus, the proxy produces estimates that are too large in magnitude—especially for older workers. Compared to the results for the adequate proxies, the estimates double on average and even increase by factor of four for workers over age 55. The same patterns emerge for the standard errors. Moreover, the considerable drop in the first-stage F-statistics indicates that the cardiovascular revolution predominantly affects life expectancy at adult age.

Both naive models suggest health effects on wages that differ substantially from the estimates

Table 4: Results by Age Cohort for Naive Measurement of Health Dynamics

	Long-difference specifications: 1960–2000						
	Dependent variable is log wage income per person in age cohort						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Life expectancy measured at age 40						
Log life expectancy at 40	-2.02**	3.59***	2.49***	1.10*	-0.34	1.49**	-0.63
	(0.88)	(0.99)	(0.82)	(0.67)	(0.76)	(0.74)	(1.06)
First-stage F–statistic	17.4	17.7	18.2	18.7	18.9	17.7	18.9
	(b) Life expectancy measured at birth						
Log life expectancy at birth	-4.92**	8.78***	6.07***	2.66*	-0.82	3.64**	-1.51
	(2.29)	(2.55)	(2.02)	(1.60)	(1.84)	(1.78)	(2.56)
First-stage F–statistic	12.5	12.8	13.4	13.8	14.0	12.8	14.0
Changes in life expectancy	9.7%	11.5%	14.3%	18.4%	22.6%	13.3%	25.7%
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage income per person in a specific age cohort. Adult health is proxied by log life expectancy at age 40 in Panel (a) and by log life expectancy at birth in Panel (b). Estimates are weighted by the initial population of the corresponding age cohort in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Changes in life expectancy refer to changes between 1960 and 2000 expressed as percent of life expectancy in 1960. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

for adequate proxies of health.[16] Because the correlation between life expectancy and mortality depends on age, the instrumentation strategy cannot eliminate this problem. If measured at too high age, changes in life expectancy do not contain health improvements resulting from a mortality reduction at younger ages. In contrast, if life expectancy is measured at too young age, then the instrument incorrectly assigns individuals health changes of older ages. Hence, the first stage also systematically overstates or understates the average health improvement if one uses an incorrect age-specific proxy.

This subtle point is of significant importance for published work. For example, the growth literature investigates the effects of health improvements on long-run changes in output per capita. These studies usually proxy health by life expectancy at birth, which is sensitive to health improvements of infants and young children. If this proxy overstates the health improvements of the workforce, then the estimation results understate the effect of health improvements on income growth. How severe this bias is depends on how well the changes in life expectancy at birth capture the health changes of the workforce. Consider, for example, the medical advances of the third stage of the epidemiological transition, which primarily benefit infants and young children. In this case, the improvements in life expectancy at birth considerably exceed the improvements in life expectancy at higher ages, such that the bias is most pronounced. This finding suggests

[16]Similar results obtain for total income per person as Table A7 in the Appendix shows.

more pronounced health effects on income growth for published work (for example, Acemoglu and Johnson, 2007, 2014; Cervellati and Sunde, 2011; Bloom et al., 2014; Hansen, 2014). The results also indicate that this bias is still substantial for the cardiovascular revolution, for which increases in life expectancy at birth more closely reflect health improvements at older ages. Therefore, age plays a significant role for the effects of health dynamics on economic outcomes and it deserves more attention in future research.

4.3 Robustness and Further Results

This section discusses the robustness of results with respect to different measures of life expectancy and income, alternative specifications, and inter-state migration. It also discusses heterogeneity in health effects across genders, occupation types, and education types. The corresponding tables are presented in the Appendix.

Alternative Measurement of Life Expectancy. The measurement of adult life expectancy is a potential concern. The proxy used in the baseline analysis combines variation from life expectancy measured at ages 20 through 60 to capture the health improvements of the workforce. As Table A8 shows, the same qualitative results are obtained for every single component of the composite indicator and for life expectancy measured at birth. In particular, all of the estimates provide evidence for a positive causal link between health and income per person. If life expectancy is measured close to the median age of the population—which increased from 31 to 38—, then the model delivers results that are quantitatively similar to the baseline.

The baseline specification uses the log of life expectancy. Table A9 reports similar quantitative results for specifications in which life expectancy enters linearly. In particular, the estimates predict almost identical effects as the baseline estimates for the health dynamics of the cardiovascular revolution on income per person. Moreover, the evidence confirms that these health dynamics shape life-cycle incomes.

Evidence for Real GDP per Worker. The baseline analysis investigates the health effects on wage and total income per person based on aggregated census data. An alternative is to use aggregate data on output. The results in Table A10 present evidence for real GDP per worker obtained from Turner et al. (2006). The results show a positive and significant effect of health on GDP per worker, irrespective of the age at which life expectancy is measured. The estimates are somewhat larger than those obtained for income per person and thus point at potential spillover effects on the macroeconomic level. Hence, the results confirm a positive causal link between health and income also for aggregate output data.

Unweighted Estimates. The baseline specification weights the estimates by the initial population to obtain results that reflect the micro structure of the data. Table A11 presents unweighted estimates to preclude the concern that weighting drives the results qualitatively. The estimates show the same qualitative patterns as the baseline model. In particular, the results confirm a positive health effect on income per person for the entire workforce and meaningful heterogeneity of the health effects across age cohorts. Quantitatively, the estimates are somewhat larger

compared to the baseline results. Therefore, the weighting provides a more conservative view on the health effects of the cardiovascular revolution.

Linear State-Time Trends. Table A12 reports results for panel specifications that control for linear state-time trends to account for the possibility of state-specific growth trends. The results show similar qualitative patterns as those in the baseline model. The parameters are quantitatively similar to the baseline results, although they are less precise due to the increased number of parameters to be estimated. Hence, state-specific growth trends do not drive the baseline results.

Migration. Workforce migration is a potential concern. About 1.5 percent of the US population moves between states per year and one-third of the citizens do not live in the state in which they were born (Molloy et al., 2011). If high productivity workers primarily migrate into states where life expectancy is high, then the estimates are biased upward. To address this problem, I restrict the sample to individuals who live in the state in which they were born.[17] Table A13 reports results for this non-migrant sample. The estimates are quantitatively similar to the baseline results, even though the standard errors are larger and the first stage is somewhat weaker. Hence, workforce migration does not drive the qualitative results. Moreover, this evidence tends to the concern whether health should be measured by state of birth or state of residence: the results are robust for the non-migrant sample, for whom the state of birth and state of residence coincide.

Heterogeneity Across Genders. Men and women differ in their risk of cardiovascular disease. Estrogen protects women against atherosclerotic diseases; but this protection diminishes as the estrogen level falls due to the menopause (Mendelsohn and Karas, 1999). Hence, the cardiovascular revolution should benefit men more than women under age 50, whereas there should be no substantial discrepancy over age 50. The evidence confirms these predictions for both, health and economic outcomes. Table A14 shows first-stage results using gender-health variation within states. The specification compares differences in predicted mortality and life expectancy between men and women within the same state before and after the cardiovascular revolution. Gender differences in predicted mortality affect life expectancy significantly between ages 15 and 54, whereas there is no significant association over age 55. Because the predicted mortality decline is larger for men than for women, male life expectancy increases more than female life expectancy. Table A15 reports second-stage results for a male and a female sample. The estimates demonstrate a positive effect of health on income per person of the workforce, which is larger for men than for women. Moreover, the results show heterogeneity across genders. For the age cohorts 25–54, the health effects differ considerably between men and women. Incomes of males increase almost twice as much as incomes of females. These differences vanish over age 55.

Heterogeneity Across Occupation Types. Blue-collar workers usually execute physically demanding tasks, whereas white-collar jobs perform cognitive tasks. With increasing age, blue-collar workers are thus more vulnerable to negative income shocks due to declining health than white-collar workers. Accordingly, medical advances should benefit blue-collar workers more than

[17] Note, however, that the effect of migrants cannot be deducted from measured life expectancy.

white-collar workers. To test this hypothesis, I classify workers as blue-collar or as white-collar based on the occupation coding guidelines for the 1970 census (U.S. Bureau of the Census, 1972, pp. 152–154). White-collar workers belong to the group of professional, technical and kindred workers, managers and administrators except farm, sales workers, or clerical and kindred workers. Blue-collar workers belong to the group of craftsmen and kindred workers, operatives except transport, transport equipment operatives, and laborers except farm. The remaining workers belong to farm or service occupations and are exempt from the analysis.

Table A16 presents results for health effects on average wages of white-collar and blue-collar workers. Health exerts a positive and significant effect on wages of blue-collar workers aged 25–54. The health effects considerably exceed the baseline estimates for all cohorts between age 15 and 64. Results for the white-collar workers sample contrast this picture. Their health effects are considerably smaller compared to the baseline estimates and are close to zero for most of the age cohorts. The only notable exception is the age cohort 25–34, which is consistent with higher educational attainment of young white-collar workers. Altogether, this evidence supports the hypothesis that health dynamics especially benefit blue-collar workers who are more susceptible to negative income shocks due to declining health.

Heterogeneity Across Education Types. Whether health dynamics should have heterogeneous effects on wage incomes across education types is a priori unclear. On the one hand, wages of college-educated workers grow faster with additional work experience compared to wages of workers without college education (see, for example, Ashenfelter and Rouse, 1999). On the other hand, wage growth starts at a later point in life because students must invest time on acquiring college education. Furthermore, increased longevity raises enrollment rates in the population. Selection into college at the margin thus slows down wage growth of college-educated workers. Finally, educational attainment is a dimension of occupational choice. If non-college jobs are physically demanding, then health dynamics especially benefit non-college workers.

To test whether health dynamics affects wages differently across education types, I create a sample for workers with at least some college education and a sample for workers with a high-school degree or less. Table A17 reports the health effects on wages of college-educated and non-college-educated workers. For the college sample, there are only significant health effects for the age cohort 15–34. The remaining estimates are negative and close to zero. In contrast, the non-college sample reveals a positive link between health and prime-age workers similar to the baseline sample. At the same time, the estimates are more negatively pronounced for workers under 24 and over 65. Non-college-educated workers in these age cohorts are more likely to be selected in 2000 than in 1960 because educational attainment increases over time. Overall, the health effects are more pronounced for non-college-educated workers; however, this result does not preclude wage growth through a growing share of college education in the workforce.

4.4 Channels

What are the channels through which the health dynamics of the cardiovascular revolution raise income per person? This section discusses three potential channels: labor supply, educational attainment, and better health.

Table 5: Effect of Adult Health on Labor Supply by Age Cohort

	Long-difference specifications: 1960–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Labor force participation (estimates divided by 100)						
Log life expectancy	-0.50	0.66	0.29	0.03	-0.06	0.05	-0.28
(of specific age cohort)	(0.66)	(0.58)	(0.33)	(0.21)	(0.23)	(0.33)	(0.18)
	(b) Usual hours worked per week (estimates divided by 100)						
Log life expectancy	-0.34***	0.37***	0.17**	0.18**	0.06	0.27*	-0.16***
(of specific age cohort)	(0.09)	(0.13)	(0.08)	(0.08)	(0.06)	(0.16)	(0.06)
	(c) Usual weeks worked per year (estimates divided by 100)						
Log life expectancy	-1.14***	0.08	0.01	-0.07	-0.08	-0.18**	-0.06
(of specific age cohort)	(0.33)	(0.09)	(0.05)	(0.06)	(0.06)	(0.09)	(0.09)
First-stage F–statistic	17.3	17.5	18.2	18.6	22.2	20.1	18.5
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is the labor force participation rate (from 0 to 100 percentage points) in Panel (a), usual hours worked per week in Panel (b), and usual weeks worked per years in Panel (c). Estimates are weighted by the initial population of the corresponding age cohort in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Labor Supply. Changes in labor supply may shape incomes in different ways. At the extensive margin, healthy workers continue to work at advanced ages. If predominantly productive workers stay in the workforce, then this raises average incomes. At the intensive margin, healthy workers work longer hours per week or more weeks per year and thus earn higher incomes. Table 5 reports semi-elasticities for labor force participation in Panel (a), usual hours worked per week in Panel (b), and usual weeks worked per year in Panel (c). Because the dependent variable enters the empirical models linearly, the estimates must be divided by 100 to obtain the marginal effects.

Health dynamics do not affect labor force participation. The estimates in Panel (a) are close to zero and insignificant across all age cohorts. Therefore, rising incomes do not result from changes in labor supply at the extensive margin. Panel (b), by contrast, reveals significant health effects on usual hours worked per week, which differ across age cohorts. These health effects are positive for prime-age workers aged 25–54, whereas they are negative for workers under age 24 and over age 65. For the workforce aged 15–64, a 1-percent increase in life expectancy raises usual time worked per week by 15 minutes. The cardiovascular revolution thus accounts for an increase of two hours usually worked per week. At the same time, usual work weeks themselves do not change considerably. As Panel (c) shows, life expectancy significantly affects usual work weeks only for workers aged 15–24. Therefore, the changes in labor supply at the intensive margin are qualitatively consistent with the income changes documented in the baseline analysis.

Table 6: Effect of Adult Health on College Graduation by Age Cohort

	Long-difference specifications: 1960–2000						
	Dependent variable: college graduation rates in age cohort (estimates divided by 100)						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log life expectancy	1.42***	2.52***	1.41***	0.61*	0.15	1.31***	-0.05
(of specific age cohort)	(0.34)	(0.64)	(0.43)	(0.34)	(0.19)	(0.37)	(0.10)
First-stage F–statistic	17.3	17.5	18.2	18.6	22.2	20.1	18.5
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is the college graduation rate of a specific age cohort (from 0 to 100 percentage points). Estimates are weighted by the initial population of the corresponding age cohort in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Individuals work more during prime working ages, and they work less under age 24 and over age 65. However, these changes are quantitatively too small to account for the entire income change: for the workforce aged 15–64, income per person increases by 13 percent, whereas health dynamics raise labor supply only by 2 percent. Lifetime working hours, which also include labor supply over age 65, even marginally decline.[18] Hence, labor supply is one channel—but is not the only channel—through which the cardiovascular revolution affects incomes.

Educational Attainment. Higher educational attainment is another channel through which health dynamics impact incomes. Between 1960 and 2000, the share of whites who enrolled into college at least once in their lifetime more than doubled from 18 to 46 percent, and the share of graduates with at least four years of college education roughly tripled from 7 to 23 percent (Ruggles et al., 2015). According to Hansen and Strulik (2017, p. 438), the cardiovascular revolution accounts for slightly more than one-half of the observed increase in higher education over this time period. To compare the effects of health on education with the age-specific effects on income per person, I estimate the effect of health on college graduation rates across age cohorts. Table 6 reports the corresponding semi-elasticities. Because the dependent variable enters the models linearly, the estimates must be divided by 100 to obtain the marginal effects.

Adult health positively affects college graduation rates of the workforce aged 15–64. A 1-percent increase in life expectancy raises the college graduation rate by 1.31 percentage points. Hence, the health dynamics between 1960 and 2000 account for an increase in college graduation

[18] To derive the change in labor supply of the workforce, compute $\Delta_y = \Delta_x/\mu_x^{1960}(\widehat{\alpha}^{(b)}\mu_y^{(c),1960} + \widehat{\alpha}^{(c)}\mu_y^{(b),1960}) \approx 2.68/35.26 \cdot (0.27 \cdot 42.10 - 0.16 \cdot 40.29) \approx 37.40$ with $\Delta_y/(\mu_y^{(b)} \cdot \mu_y^{(c)}) \approx \frac{37.40}{1696.21} \approx 0.02$, where $\mu_y^{(b),1960}$ and $\mu_y^{(c),1960}$ denote the sample means of usual hours worked per week and usual weeks worked per year, both evaluated in 1960. Analogously, compute $\Delta_y \approx 2.68/35.26 \cdot (0.13 \cdot 42.10 - 0.19 \cdot 40.29) \approx -16.59$ with $\Delta_y/(\mu_y^{(b)} \cdot \mu_y^{(c)}) \approx -\frac{16.59}{1696.21} \approx -0.01$ to derive the change in lifetime working hours. The estimates 0.13 and 0.19 obtain from unreported regressions including all age cohorts.

rates of 10 percentage points, or roughly 60 percent of the overall increase.[19] Furthermore, the estimates differ considerably across age cohorts: there are positive and significant health effects for the age cohorts 15–54 but not for the older age cohorts. This heterogeneity is consistent with education investments over the life cycle. Given that individuals usually complete their education between age 20 and 30, the cardiovascular revolution does not raise educational attainment of the age cohorts that complete their education before its beginning. Overall, increasing educational attainment accords with the income changes in the baseline results. Incomes and educational attainment both increase for prime-age workers aged 25–54, they both stagnate for the age cohort 55–64, and they both slightly decline for workers over age 65. Only for the cohort 15–24, incomes and educational attainment move in different directions. However, this discrepancy is consistent with productive individuals selecting to go to college instead of working.

The results for labor supply and education are consistent with predictions of prototype Ben-Porath models that incorporate health (for example, Cervellati and Sunde, 2013; Strulik and Werner, 2016). Increasing health raises benefits of education relative to its costs, thereby unfolding a positive income effect on lifetime wealth. Individuals need not to increase their labor supply to accommodate for higher longevity if this income effect is large enough to afford both, higher lifetime consumption and leisure time while at work. The empirical evidence shows that increasing longevity does not require higher lifetime labor supply to boost educational attainment, which confirms the theoretical predictions and simulation results of Cervellati and Sunde (2013) and Strulik and Werner (2016).

Better Health. Health dynamics affect incomes also directly by boosting worker productivity and by reducing adverse effects of poor health (Currie and Madrian, 1999; Schultz, 2002). To test whether the health dynamics of the cardiovascular revolution directly raise incomes, I use longitudinal data from the Health and Retirement Study. The dataset contains up to 12 observations from biannual interviews between 1992 and 2014 for 22,000 respondents. Table 7 reports the effects of individual health on incomes. Estimates are obtained by regressing log income $y_{i,s,t}$ of individual i in state s at interview wave t on a binary indicator for health status $h_{i,t}$; its interaction $h_{i,t}b_i^\tau$ with a dummy that takes a value of one if individual i was born before a cutoff year τ; a quartic age trend $a_{i,t}^k$, $k \in \{1,2,3,4\}$; and a set of fixed effects. Specifically,

$$y_{i,s,t} = \vartheta h_{i,t} + \rho h_{i,t} b_i^\tau + \sum_{k=1}^{4} \phi_k a_{i,t}^k + \iota_i + \chi_t + \psi_{s,t} + \omega_{r,t} + \epsilon_{i,t} \tag{5}$$

where ι_i, χ_t, $\psi_{s,t}$, and $\omega_{r,t}$ denote individual-fixed, wave-fixed, state-fixed, and census-region-wave effects; and $\epsilon_{i,t}$ constitutes an idiosyncratic error term. Importantly, the individual-fixed effect eliminates time-invariant heterogeneity in pivotal dimensions such as ability, educational attainment, and occupational choice. State-fixed effects control for state-specific heterogeneity. Finally, wave and region-wave-fixed effects account for differential wage trends over time.

The parameter ϑ describes the direct effect of having ever been diagnosed with an adverse health status such as 'having heart problems'; in this case, $h_{i,t}$ takes a value of one. The estimates

[19] To obtain these numbers, compute $\Delta_y = \widehat{\alpha} \cdot \Delta_x / \mu_x^{1960} \cdot 100 \approx 1.31 \cdot 2.68/35.26 \cdot 100 \approx 9.96$ with $\Delta_y/(\mu_y^{2000} - \mu_y^{1960}) \approx \frac{9.96}{16.15} \approx 0.62$, where μ_y^{1960} and μ_y^{2000} denote the sample means evaluated in 1960 and 2000.

Table 7: Effect of Individual Health on Income

	Dependent variable: respondents' log incomes					
	(1)	(2)	(3)	(4)	(5)	(6)
Heart Disease	-0.06**	-0.06**	-0.06**	-0.07**	-0.01	-0.06
	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.06)
× born before 1910		-0.89***				
		(0.07)				
× born before 1920			-0.22			
			(0.23)			
× born before 1930				0.09		
				(0.10)		
× born before 1940					-0.10**	
					(0.05)	
× born before 1950						-0.00
						(0.06)
Individuals	22214	22214	22214	22214	22214	22214
Born before cutoff year	—	71	1062	5947	36112	63154
Observations with heart disease	10023	10023	10023	10023	10023	10023
Total observations	84041	84041	84041	84041	84041	84041

Note: All regressions include individual-fixed, state-fixed, wave-fixed, and region-wave-fixed effects, and a quartic age trend. Heart disease is a binary indicator that takes value one if respondents reports to ever have heart problems diagnosed, and zero else. Heart disease is interacted with a dummy indicator that takes value one if the individual is born before a certain threshold level, and zero else. Standard errors are clustered at the individual level. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

correspond to roughly 0.06 throughout all specifications except Column (5). Accordingly, workers who have been diagnosed with heart problems earn on average 6 percent lower incomes than workers without heart problems. However, adverse health affects incomes even more negatively because the analysis cannot capture particularly severe cases of heart problems, which end lethally or in disability. Hence, the estimates constitute an upper bound.

The parameter ρ captures heterogeneity with respect to the health effect for different birth cohorts. For example, individuals born before 1910 were already aged over 50 when the cardiovascular revolution began. Hence, it came too late for them to considerably affect their work life. Column (2) reports a significant negative health effect for this group compared to younger cohorts. Due to the small number of only 71 observations before 1910, the resulting coefficient may, however, be plagued by small sample properties and should thus be seen as suggestive. Columns (3) and (4) illustrate that once the cutoff year shifts to younger cohorts, which are at least partly affected by the cardiovascular revolution, the interaction term becomes insignificant. Finally, the results in Columns (5) and (6) indicate that suffering from heart problems poses no negative effect on individual incomes for birth cohorts that are young enough to fully harness the health improvements of the cardiovascular revolution. In particular, the direct adverse effect of suffering from heart problems becomes insignificant once the interaction term splits the sample into individuals who benefit only partly or not at all from the treatment and individuals of the reference group who fully avail of it. Overall, this evidence suggests that health innovations marginalize adverse effects of cardiovascular diseases on productivity and income over time.[20]

[20]Similar findings apply if the health status is measured by 'high blood pressure' as Table A18 in the Appendix

5 Concluding Remarks

Economists still debate over the question of whether or not improvements in population health spur income growth. However, because this debate concentrates on the average health effect for the entire population, it overlooks how these effects vary over the life cycle, how the age at which health is measured affects estimation outcomes, and which economic factors explain income dynamics at different ages.

This paper empirically investigates the long-run effects of rising life expectancy on income growth for the US workforce and tests for heterogeneity in the effects of health dynamics on incomes over the life cycle. To isolate exogenous changes in health, the empirical strategy exploits differences in the decline of cardiovascular disease mortality across states over time. Starting in the 1960s, medical advance in treating cardiovascular diseases and public health campaigns considerably reduce cardiovascular disease mortality in all states. The extent of this decline depends on the initial prevalence of these diseases within each state. The empirical model combines the differences in cardiovascular disease mortality in 1960 with the unexpected medical advance in treating these diseases between 1960 and 1970 to predict the mortality decline for each state, and uses them as instruments for changes in life expectancy.

The results show that health improvements spur aggregate incomes between 1960 and 2000. In total, the cardiovascular revolution accounts for an increase in income per person of 13 percent compared to 1960 and thus explains one-quarter of the income growth between 1960 and 2000. These health effects differ qualitatively and quantitatively across age cohorts. While life expectancy positively affects income per person aged 25–54, it exerts no or even has a negative effect on income per person under age 24 and over age 55. Consequently, life-cycle income profiles slope more strongly at the beginning and at the end of work life. Compared to previous generations, age thus becomes a more prominent determinant of income dynamics. Because health improvements also differ across age cohorts, the age at which life expectancy is measured can affect the estimation results. In particular, the results overstate the health effects on income for proxies that understate the age-specific changes in life expectancy, and vice versa. Future research should thus pay more attention to the age structure of the population.

The evidence suggests that the cardiovascular revolution has shaped life-cycle incomes directly by improving individual health, and indirectly by raising educational attainment and by changing labor supply. Public health campaigns may also change behavior toward a healthier lifestyle by raising awareness of individual risk factors of cardiovascular diseases. These changes would be consistent with the effects of medical advance on health and income dynamics that I document in this paper. I leave this channel for future research.

shows. For 'stroke' as proxy of the health status, the results show qualitatively similar though statistically insignificant effects. However, this finding is not surprising because the sample contains considerably fewer observations for stroke and selection out of the labor market is particularly strong for this group.

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Appendix

Additional Figures

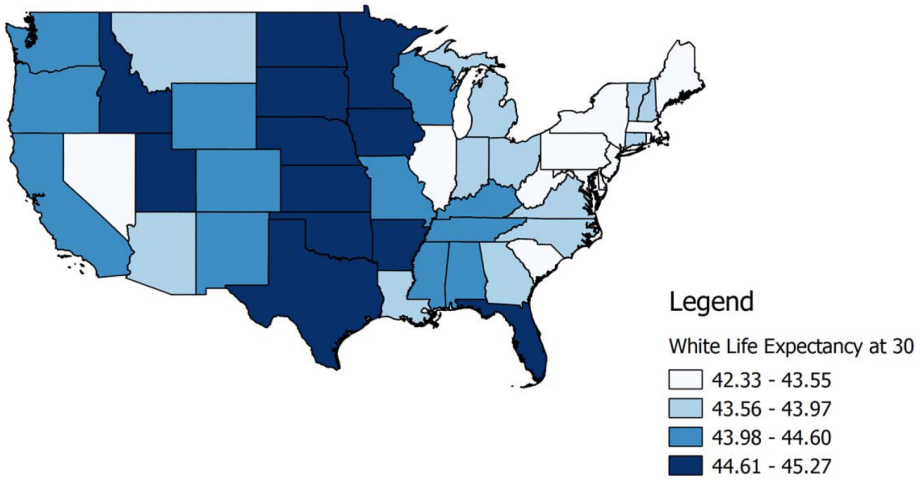
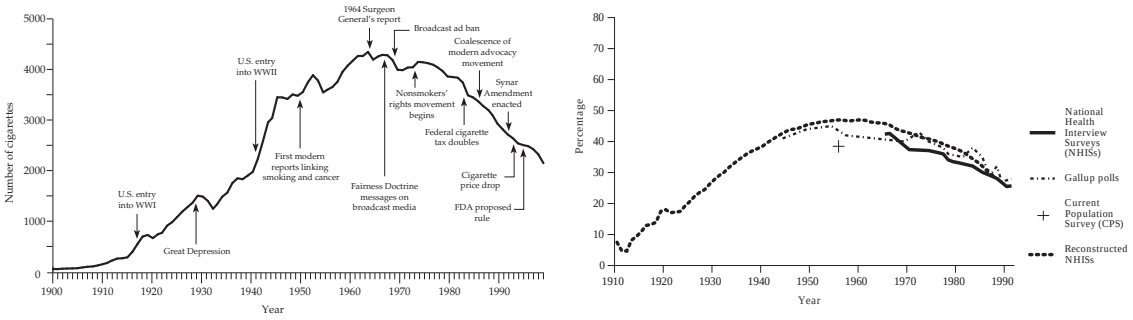


Figure A1: Geographic Dispersion of Life Expectancy at 30 in 1960

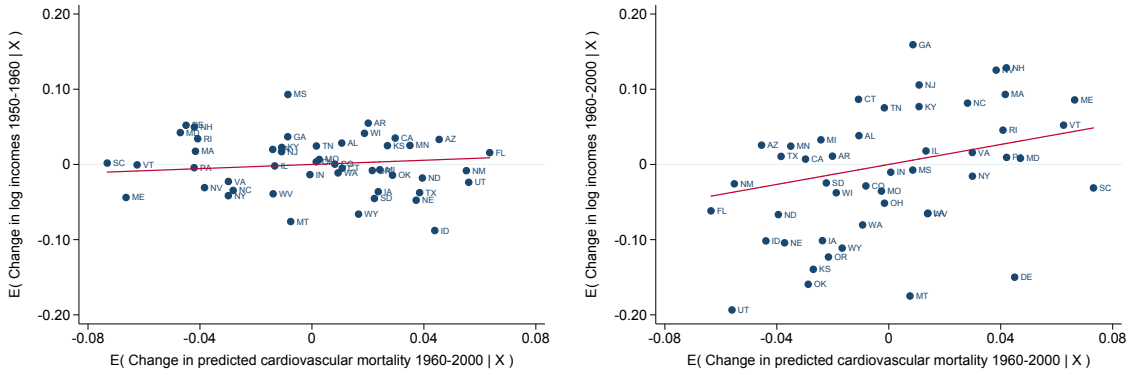
Data source: Grove and Hetzel (1968).



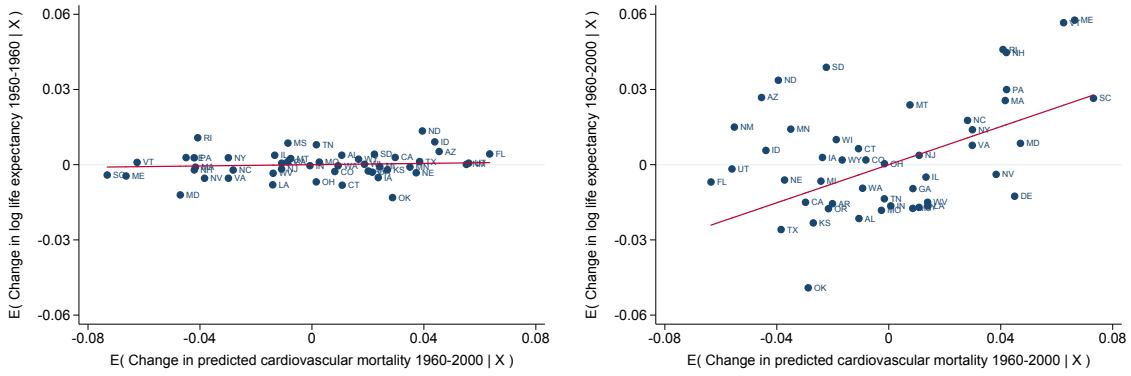
(a) Cigarette Consumption Per Capita (b) Share of Smokers in Population

Figure A2: Smoking in the United States

Sources: US Department of Health and Human Services (1998, p. 123; 2000, p. 33).



(a) Reduced Form 1950–1960 (Table 2b, Col. 6) (b) Reduced Form 1960–2000 (Table 2b, Col. 6)



(c) First Stage 1950–1960 (Table 2c, Col. 6) (d) First Stage 1960–2000 (Table 2c, Col. 6)

Figure A3: Partial Correlations Before and After the Beginning of the Cardiovascular Revolution

Additional Tables

Table A1: Descriptive Statistics

Age cohort	15–64	15–24	25–34	35–44	45–54	55–64	65+
			Adult health and mortality				
Log life expectancy in 1960	3.56	3.98	3.79	3.55	3.25	2.90	2.69
	(0.02)	(0.01)	(0.02)	(0.02)	(0.03)	(0.03)	(0.03)
Log life expectancy in 2000	3.69	4.07	3.89	3.68	3.42	3.10	2.91
	(0.02)	(0.02)	(0.02)	(0.02)	(0.03)	(0.03)	(0.04)
Cardiovascular disease mortality in 1960	0.40	—	—	—	—	—	—
	(0.04)	—	—	—	—	—	—
Non-cardiovascular disease mortality in 1960	0.37	—	—	—	—	—	—
	(0.03)	—	—	—	—	—	—
			Economic outcomes				
Log wage income per person in 1960	10.17	9.39	10.26	10.39	10.35	10.28	9.86
	(0.14)	(0.13)	(0.11)	(0.13)	(0.14)	(0.16)	(0.19)
Log wage income per person in 2000	10.58	9.49	10.49	10.76	10.85	10.76	10.21
	(0.16)	(0.10)	(0.14)	(0.16)	(0.15)	(0.15)	(0.20)
Log total income per person in 1960	10.24	9.44	10.31	10.45	10.41	10.26	9.61
	(0.13)	(0.12)	(0.11)	(0.12)	(0.14)	(0.15)	(0.16)
Log total income per person in 2000	10.62	9.48	10.50	10.78	10.88	10.79	10.38
	(0.15)	(0.09)	(0.14)	(0.16)	(0.15)	(0.15)	(0.11)
Log output per worker in 1960	10.22	—	—	—	—	—	—
	(0.14)	—	—	—	—	—	—
Log output per worker in 2000	10.91	—	—	—	—	—	—
	(0.13)	—	—	—	—	—	—
			Labor supply				
Labor force participation in 1960	61.60	48.99	63.93	68.08	69.29	57.94	19.81
	(2.88)	(3.92)	(2.43)	(2.46)	(3.03)	(4.48)	(2.87)
Labor force participation in 2000	76.65	67.64	82.87	83.33	82.00	59.96	13.65
	(3.54)	(3.82)	(3.17)	(2.98)	(3.73)	(5.34)	(1.70)
Usual work hours per week in 1960	40.29	35.67	42.25	42.46	42.47	41.62	36.30
	(0.85)	(1.06)	(0.91)	(1.12)	(1.29)	(1.13)	(1.03)
Usual work hours per week in 2000	32.25	32.01	41.49	41.91	42.08	39.91	30.34
	(1.39)	(1.12)	(0.72)	(0.71)	(0.68)	(0.67)	(1.31)
Usual work weeks per year in 1960	42.10	32.02	44.07	45.28	45.34	44.35	37.51
	(1.04)	(1.72)	(0.89)	(0.88)	(0.94)	(1.09)	(1.52)
Usual work weeks per year in 2000	44.97	35.49	46.36	47.41	47.72	45.47	38.24
	(0.69)	(1.17)	(0.81)	(0.62)	(0.53)	(0.76)	(1.31)
			Education and population				
Share college graduation in 1960	7.26	2.46	11.06	9.06	7.65	6.05	4.09
	(1.45)	(0.65)	(2.26)	(1.92)	(1.52)	(1.24)	(1.10)
Share college graduation in 2000	23.41	5.73	29.35	26.95	30.30	23.47	15.68
	(4.66)	(1.81)	(5.85)	(5.22)	(5.31)	(5.05)	(3.48)
Log population in 1960	14.54	—	—	—	—	—	—
	(1.00)	—	—	—	—	—	—
Log population in 2000	14.90	—	—	—	—	—	—
	(0.93)	—	—	—	—	—	—

Note: Descriptive statistics for the 48 contiguous US states in 1960 and 2000. Number are means for the corresponding variable in the working-age population or a specific age cohort. All numbers refer to the white population. Standard deviations are in parentheses.

Table A2: OLS: Adult Health and Income Per Person

	Long-difference specifications: 1960–2000					
	Log wage income per person 15–64			Log total income per person 15–64		
	(1)	(2)	(3)	(4)	(5)	(6)
Log life expectancy	0.20	0.71	0.55	0.36	1.03**	0.84*
	(0.58)	(0.47)	(0.47)	(0.54)	(0.45)	(0.43)
Controls in 1960 × Post 1960:						
Initial share college	—	✓	✓	—	✓	✓
Initial income	—	✓	✓	—	✓	✓
Initial mortality	—	—	✓	—	—	✓
States	48	48	48	48	48	48
Observations	96	96	96	96	96	96

Note: This table reports ordinary least squares estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage income per person aged 15–64 (Columns 1 to 3) and log total income per person aged 15–64 (Columns 4 to 6). Estimates are weighted by the population in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A3: Effect of Adult Health on Income Per Person: Panel 1950–2000

	Log wage income per person 15–64			Log total income per person 15–64		
	(1)	(2)	(3)	(4)	(5)	(6)
	Second stage (LIML)					
Log life expectancy	-1.61	1.79	1.50	-0.76	2.03*	1.72
	(1.08)	(1.10)	(1.04)	(1.01)	(1.19)	(1.13)
First-stage F–statistic	7.5	6.8	6.4	7.5	7.4	6.9
Controls in 1960 × year dummies:						
Initial share college	—	✓	✓	—	✓	✓
Initial income	—	✓	✓	—	✓	✓
Initial mortality	—	—	✓	—	—	✓
States	48	48	48	48	48	48
Observations	288	288	288	288	288	288

Note: This table reports second-stage results for the heteroskedasticity-robust limited information maximum likelihood estimator (LIML). The specifications use ten-year panel data over the time period 1950–2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage income per person aged 15–64 (Columns 1 to 3) and log total income per person aged 15–64 (Columns 4 to 6). Estimates are weighted by the population in 1960. Control variables are measured in 1960 and interacted with year dummies. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A4: Effect of Adult Health on Income Per Person by Age Cohort

	Panel specifications: 1950–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Log wage income per person in age cohort						
Log life expectancy	-1.99	4.58***	2.22***	0.05	-0.70**	1.23**	-0.90*
(of specific age cohort)	(1.36)	(0.87)	(0.58)	(0.49)	(0.34)	(0.56)	(0.47)
First-stage F–statistic	9.5	10.9	12.9	13.0	17.4	12.8	15.8
	(b) Log total income per person in age cohort						
Log life expectancy	-2.27*	4.43***	2.49***	0.58	-0.44	1.47**	-1.80***
(of specific age cohort)	(1.33)	(0.80)	(0.62)	(0.49)	(0.34)	(0.58)	(0.43)
First-stage F–statistic	9.4	11.5	13.6	16.4	18.8	13.6	16.7
States	48	48	48	48	48	48	48
Observations	288	288	288	288	288	288	288
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for ten-year panel data over the time period 1950–2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage income per person in a specific age cohort in Panel (a) and log total income per person in a specific age cohort in Panel (b). Estimates are weighted by the initial population of the corresponding age cohort in 1960. Control variables are measured in 1960 and interacted with year dummies; the year 1960 serves as reference category and is omitted. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A5: Reduced Form and First Stage by Age Cohort

	Long-difference specifications: 1960–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Reduced form: log wage income per person in age cohort						
Mortality × Post 1960	-0.84**	1.48***	1.05***	0.47	-0.15	0.62**	-0.28
	(0.39)	(0.27)	(0.30)	(0.29)	(0.36)	(0.28)	(0.50)
	(b) Reduced form: log total income per person in age cohort						
Mortality × Post 1960	-0.89***	1.37***	1.11***	0.59*	0.06	0.66**	-0.72
	(0.32)	(0.24)	(0.28)	(0.29)	(0.34)	(0.27)	(0.47)
	(c) First stage: log wage income per person in age cohort						
Mortality × Post 1960	0.25***	0.32***	0.42***	0.49***	0.72***	0.38***	0.77***
	(0.06)	(0.08)	(0.10)	(0.11)	(0.15)	(0.08)	(0.18)
	(d) First stage: log total income per person in age cohort						
Mortality × Post 1960	0.24***	0.32***	0.42***	0.54***	0.71***	0.38***	0.75***
	(0.06)	(0.07)	(0.09)	(0.10)	(0.14)	(0.08)	(0.17)
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage per person in a specific age cohort in Panel (a) and log total income per person in a specific age cohort in Panel (b). Panel (c) shows first-stage estimates for the model with log average wages and Panel (d) for the model with log average total incomes. Estimates are weighted by the initial population of the corresponding age cohort in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A6: OLS: Adult Health and Income Per Person by Age Cohort

	Long-difference specifications: 1960–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Log wage income per person in age cohort						
Log life expectancy (of specific age cohort)	-1.05 (0.75)	1.78*** (0.55)	1.23** (0.47)	-0.18 (0.33)	-0.29 (0.34)	0.55 (0.47)	-0.09 (0.43)
	(b) Log total income per person in age cohort						
Log life expectancy (of specific age cohort)	-0.80 (0.82)	2.20*** (0.46)	1.52*** (0.45)	0.04 (0.34)	-0.16 (0.33)	0.84* (0.46)	-1.29*** (0.24)
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports ordinary least squares estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage income per person aged 15–64 in Panel (a) and log total income per person aged 15–64 in Panel (b). Estimates are weighted by the initial population of the corresponding age cohort in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A7: Results by Age Cohort for Naive Measurement of Health Dynamics

	Long-difference specifications: 1960–2000						
	Dependent variable is log total income per person						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Life expectancy measured at age 40						
Log life expectancy at 50	-2.18***	3.33***	2.67***	1.39*	0.15	1.62**	-1.68**
	(0.77)	(0.91)	(0.86)	(0.76)	(0.77)	(0.77)	(0.84)
First-stage F–statistic	19.0	19.3	19.8	20.3	20.2	19.1	19.9
	(b) Life expectancy measured at birth						
Log life expectancy at birth	-5.46**	8.39***	6.70***	3.46*	0.36	4.05**	-4.13**
	(2.12)	(2.45)	(2.23)	(1.91)	(1.91)	(1.94)	(2.07)
First-stage F–statistic	13.1	13.4	13.9	14.4	14.4	13.3	14.1
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log total income per person in a specific age cohort. Adult health is proxied by log life expectancy at 40 in Panel (a) and by log life expectancy at birth in Panel (b). Estimates are weighted by the initial population of the corresponding age cohort in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A8: Robustness: Different Measures of Adult Health

	Long-difference specifications: 1960–2000						
	Life expectancy measured at						
	birth	20	30	40	50	60	mean
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Log wage income per person aged 15–64						
Log life expectancy	3.64**	2.46**	1.91**	1.49**	1.29*	0.91**	1.65**
(of specific age cohort)	(1.78)	(1.17)	(0.93)	(0.74)	(0.68)	(0.46)	(0.81)
First-stage F–statistic	12.8	17.4	17.5	17.7	18.4	19.5	20.1
	(b) Log total income per person aged 15–64						
Log life expectancy	4.05**	2.73**	2.07**	1.62**	1.25**	0.98**	1.75**
(of specific age cohort)	(1.94)	(1.25)	(0.97)	(0.77)	(0.59)	(0.48)	(0.82)
First-stage F–statistic	13.3	17.9	18.8	19.1	27.6	22.1	22.1
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage income per person aged 15–64 in Panel (a) and log total income per person aged 15–64 in Panel (b). Estimates are weighted by the population in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A9: Robustness: Linear Specification of Life Expectancy

	Long-difference specifications: 1960–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Log wage income per person in age cohort						
Life expectancy	-0.06**	0.11***	0.08***	0.04	-0.01	0.05**	-0.03
(of specific age cohort)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.05)
First-stage F–statistic	13.7	13.5	13.5	10.9	14.2	14.6	10.8
	(b) Log total income per person in age cohort						
Life expectancy	-0.07**	0.10***	0.08***	0.05*	0.01	0.05**	-0.07**
(of specific age cohort)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)	(0.03)
First-stage F–statistic	14.1	14.6	14.5	18.4	15.4	16.1	11.4
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage income per person in a specific age cohort in Panel (a) and log total income per person in a specific age cohort in Panel (b). Life expectancy enters all specification linearly. Estimates are weighted by the initial population of the corresponding age cohort in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A10: Robustness: Effect of Adult Health on Real GDP Per Worker

	Long-difference specifications: 1960–2000						
	Life expectancy measured at						
	birth	20	30	40	50	60	mean
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log life expectancy	13.25***	8.95***	6.95***	5.45***	4.71***	3.33***	6.00***
(of specific age cohort)	(3.92)	(2.68)	(1.90)	(1.47)	(1.19)	(0.78)	(1.54)
First-stage F–statistic	12.8	17.4	17.5	17.7	18.4	19.5	20.1
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log real GDP per worker. Estimates are weighted by the population in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A11: Robustness: Unweighted Estimates

	Long-difference specifications: 1960–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Log wage income per person in age cohort						
Log life expectancy	-0.16	5.63***	3.30***	1.54**	0.76	3.15***	-0.87
(of specific age cohort)	(1.40)	(1.70)	(1.06)	(0.78)	(0.63)	(1.14)	(1.06)
First-stage F–statistic	10.6	9.9	10.7	10.6	9.8	10.2	8.4
	(b) Log total income per person in age cohort						
Log life expectancy	-0.80	6.09***	4.17***	2.17**	1.49**	3.93***	0.18
(of specific age cohort)	(1.62)	(2.02)	(1.38)	(0.87)	(0.69)	(1.39)	(0.74)
First-stage F–statistic	7.9	8.1	9.1	10.2	8.7	8.6	7.6
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage income per person in a specific age cohort in Panel (a) and log total income per person in a specific age cohort in Panel (b). Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A12: Robustness: Controlling for Linear State-Time Trends

	Panel specifications: 1950–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Log wage income per person in age cohort						
Log life expectancy	-4.68	3.93	3.39	1.26	1.32*	1.69	0.15
(of specific age cohort)	(2.90)	(3.18)	(2.83)	(1.11)	(0.75)	(1.57)	(1.24)
First-stage F–statistic	6.1	4.8	4.7	3.4	7.2	5.6	7.3
	(b) Log total income per person in age cohort						
Log life expectancy	-6.45**	2.70	4.10	2.73	2.03**	1.62	2.98*
(of specific age cohort)	(2.85)	(3.36)	(3.39)	(1.70)	(0.86)	(1.77)	(1.66)
First-stage F–statistic	5.5	4.8	4.5	3.2	8.3	6.2	8.7
States	48	48	48	48	48	48	48
Observations	288	288	288	288	288	288	288
State-time trends	✓	✓	✓	✓	✓	✓	✓
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for ten-year panel data over the time period 1950–2000. All regressions include state-fixed, time-fixed effects, and state-linear time trends. The dependent variable is log wage income per person in a specific age cohort in Panel (a) and log total income per person in a specific age cohort in Panel (b). Estimates are weighted by the initial population of the corresponding age cohort in 1960. Control variables are measured in 1960 and interacted with year dummies. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A13: Robustness: No Interstate Migration

	Long-difference specifications: 1960–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Log wage income per person in age cohort						
Log life expectancy	-7.30	5.12**	1.74	0.79	-1.17*	0.51	-1.37
(of specific age cohort)	(4.73)	(2.60)	(1.26)	(1.02)	(0.68)	(1.39)	(1.00)
First-stage F–statistic	2.3	4.1	5.6	11.4	10.8	5.8	11.3
	(b) Log total income per person in age cohort						
Log life expectancy	-7.88*	4.15**	2.05*	0.81	-0.66	0.94	-1.61**
(of specific age cohort)	(4.55)	(1.83)	(1.15)	(0.71)	(0.66)	(1.22)	(0.67)
First-stage F–statistic	3.2	6.0	7.7	19.5	14.8	8.7	15.1
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage income per person in a specific age cohort in Panel (a) and log total income per person in a specific age cohort in Panel (b). Average wages and incomes include only workers who live in the same state they were born in. Estimates are weighted by the initial population of the corresponding age cohort in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A14: Heterogeneity: Gender-Health Differences

	Long-difference specifications: 1960–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Mortality × 1960	0.19***	0.26***	0.28***	0.25**	0.11	0.22***	0.00
	(0.06)	(0.07)	(0.09)	(0.11)	(0.17)	(0.08)	(0.21)
States	48	48	48	48	48	48	48
Observations	192	192	192	192	192	192	192
State-time-fixed effects	✓	✓	✓	✓	✓	✓	✓
State-gender-fixed effects	✓	✓	✓	✓	✓	✓	✓
Gender-time-fixed effects	✓	✓	✓	✓	✓	✓	✓
Controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports first-stage estimates based on gender-health differences for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed, state-time-fixed, state-gender-fixed, and gender-time-fixed effects. The dependent variable is log life expectancy of a specific age cohort. Estimates are weighted by the initial population of the corresponding age cohort in 1960. All specifications control for initial non-cardiovascular disease mortality measured in 1960 and interacted with year dummies. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A15: Heterogeneity: Health Effects by Gender

	Long-difference specifications: 1960–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Log wage income per male person in age cohort						
Log life expectancy	-3.44	5.82***	3.13***	1.62*	0.10	2.61**	-0.30
(of specific age cohort)	(2.11)	(1.82)	(1.12)	(0.97)	(0.66)	(1.21)	(0.86)
First-stage F–statistic	9.0	12.5	13.7	10.4	11.3	12.2	10.0
	(b) Log wage income per female person in age cohort						
Log life expectancy	-3.04***	3.47***	1.91***	0.61	-0.28	1.09*	-0.37
(of specific age cohort)	(1.09)	(0.86)	(0.62)	(0.44)	(0.34)	(0.60)	(0.49)
First-stage F–statistic	20.5	20.7	21.5	23.0	24.9	22.9	20.9
	(c) Log total income per male person in age cohort						
Log life expectancy	-3.96**	5.26***	3.22***	1.56**	0.40	2.56**	-0.78
(of specific age cohort)	(1.95)	(1.65)	(1.09)	(0.78)	(0.63)	(1.13)	(0.69)
First-stage F–statistic	9.4	13.2	14.8	18.0	13.4	13.6	12.3
	(d) Log total income per female person in age cohort						
Log life expectancy	-3.25***	3.38***	2.13***	0.80	-0.04	1.23*	-0.95***
(of specific age cohort)	(0.97)	(0.86)	(0.69)	(0.49)	(0.36)	(0.65)	(0.33)
First-stage F–statistic	21.0	21.2	22.1	27.4	24.9	23.5	20.5
States	48	48	48	48	48	48	48
Observations	96	96	968	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log wage income per person in a specific age cohort in Panels (a) and (b) and log total income per person in a specific age cohort in Panels (c) and (d). Panels (a) and (c) report estimates for males, whereas Panels (b) and (d) report estimates for females. Estimates are weighted by the initial population of the corresponding age-gender group in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A16: Heterogeneity: Health Effects by Occupation Type

	Long-difference specification: 1960–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Log wage income of blue-collar workers						
Log life expectancy	0.16	6.43***	3.95***	2.42***	0.16	3.43***	-1.03
(of specific age cohort)	(1.44)	(1.72)	(1.16)	(0.93)	(0.47)	(1.10)	(0.81)
First-stage F–statistic	18.6	19.1	20.4	23.3	25.3	24.0	21.3
	(b) Log wage income of white-collar workers						
Log life expectancy	-0.31	3.54***	1.35*	0.09	-0.46	1.32*	-0.05
(of specific age cohort)	(1.71)	(1.08)	(0.75)	(0.57)	(0.47)	(0.76)	(0.57)
First-stage F–statistic	17.2	17.6	18.0	17.3	22.7	20.8	17.6
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log average wage income of blue-collar workers in a specific age cohort in Panel (a) and log average wage income of white-collar workers in a specific age cohort in Panel (b). Estimates are weighted by the initial population of the corresponding age-occupation group in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A17: Heterogeneity: Health Effects by Education Type

	Long-difference specification: 1960–2000						
Age cohort	15–24	25–34	35–44	45–54	55–64	15–64	65+
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Log wage income of college-educated workers						
Log life expectancy	3.55*	2.47**	-0.03	-0.83	-0.78	-0.04	-0.41
(of specific age cohort)	(1.93)	(1.12)	(0.91)	(0.63)	(0.54)	(0.89)	(0.79)
First-stage F–statistic	18.9	18.6	19.3	20.0	21.9	21.9	16.0
	(b) Log wage income of non-college-educated workers						
Log life expectancy	-6.71***	3.86***	1.98***	1.29**	-0.34	1.61**	-1.62**
(of specific age cohort)	(2.30)	(1.07)	(0.61)	(0.55)	(0.34)	(0.64)	(0.73)
First-stage F–statistic	16.9	17.2	17.8	18.6	21.8	20.4	18.2
States	48	48	48	48	48	48	48
Observations	96	96	96	96	96	96	96
Full controls	✓	✓	✓	✓	✓	✓	✓

Note: This table reports estimates for long-difference specifications with two observations, one in 1960 and one in 2000. All regressions include state-fixed and time-fixed effects. The dependent variable is log average wage income of college-educated workers in a specific age cohort in Panel (a) and log average wage income of non-college-educated workers in a specific age cohort in Panel (b). Estimates are weighted by the initial population of the corresponding age-education group in 1960. Control variables are measured in 1960 and interacted with the post-1960 treatment dummy. The full set of controls comprises the initial share of college graduates, initial log income, and initial non-cardiovascular disease mortality. Clustered standard errors are reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A18: Effect of Individual Health on Income

	Dependent variable: respondents' log incomes					
	(1)	(2)	(3)	(4)	(5)	(6)
High blood pressure	-0.05**	-0.05**	-0.05**	-0.05**	-0.01	-0.02
	(0.02)	(0.02)	(0.02)	(0.02)	(0.02)	(0.03)
× born before 1910		-1.39***				
		(0.22)				
× born before 1920			-0.02			
			(0.29)			
× born before 1930				-0.01		
				(0.10)		
× born before 1940					-0.08*	
					(0.04)	
× born before 1950						-0.03
						(0.04)
Individuals	22213	22213	22213	22213	22213	22213
Born before cutoff year	—	72	1061	5942	36103	63142
Observations with high blood pressure	34171	34171	34171	34171	34171	34171
Total observations	84016	84016	84016	84016	84016	84016

Note: All regressions include individual-fixed, state-fixed, wave-fixed, region-wave-fixed effects, and a quartic age trend. High blood pressure is a binary indicator that takes value one if respondents report to have ever had high blood pressure been diagnosed, and zero else. High blood pressure is interacted with a dummy indicator that takes value one if the individual is born before a certain threshold level, and zero else. Standard errors are clustered at the individual level. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.