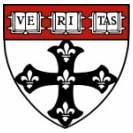

PROGRAM ON THE GLOBAL DEMOGRAPHY OF AGING AT HARVARD UNIVERSITY

Working Paper Series

Life Expectancy and Life-Cycle Wages: Evidence from the Cardiovascular Revolution in U.S. States

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April 2018

PGDA Working Paper No. 156

http://www.hsph.harvard.edu/pgda/working/

Research reported in this publication was supported by the National Institute on Aging of the National Institutes of Health under Award Number P30AG024409. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Life Expectancy and Life-Cycle Wages: Evidence from the Cardiovascular Revolution in U.S. States*

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April 20, 2018

Abstract

This paper exploits quasi-experimental variation in mortality from cardiovascular diseases across U.S. states to establish a positive causal link between adult life expectancy and average wages per worker. A cohort-specific analysis reveals that wage gains accrue to prime-age workers between 25 and 54 as well as old-age workers above 65. This pattern is consistent with a shift in life-cycle earnings toward a profile that increases more steeply for young ages and that flattens out more slowly at advanced ages. Health improvements, higher educational attainment, and changes in individual behavior constitute potential channels for this shift.

JEL-classification: I15, J11, J24, O40

Keywords: Mortality from Cardiovascular Diseases; Health Improvements; Life-Cycle Earnings; Labor Supply; Educational Attainment

*The author thanks participants of the RES Annual Conference 2018, the International Conference on "Demographics, Immigration, and the Labor Market" in Nuremberg, seminar audiences at the LMU Munich, as well as David Bloom, Lukas Buchheim, David Canning, Oliver Falck, and Uwe Sunde for helpful suggestions. Moreover, the author thanks the Michigan Center on the Demography of Aging and the Harvard Center for Population and Development Studies for providing access to the data of the Health and Retirement Study. Finally, the author gratefully acknowledges funding through the International Doctoral Program "Evidence-Based Economics" of the Elite Network of Bavaria.
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1 Introduction

Medical advancement in the twentieth century has spurred a substantial increase in longevity in the United States. As a consequence, the number of older but also healthier workers increased substantially. This development raises several questions. Do improved health conditions as measured by adult life expectancy lead to more productive workers? Moreover, do health shocks affect the population homogeneously? And, finally, what are potential channels for a causal link?

In order to answer these questions, I exploit variation in the unexpected sharp decline in mortality rates from cardiovascular diseases among U.S. states beginning in the 1960s. This decline, also referred to as cardiovascular revolution (e.g., Foege, 1987), is used as an instrument for adult life expectancy in a balanced ten-year panel from 1940 to 2000 for the 48 contiguous U.S. states. The identification strategy exploits initial differences in mortality from cardiovascular diseases across U.S. states in 1960, when there existed little treatment possibilities for these diseases. Between 1960 and 1970, a number of path-breaking innovations in the treatment of cardiovascular diseases were introduced and behavioral risk factors identified. The availability of these treatments as well as follow-up inventions and public education about risks helped reduce mortality from cardiovascular diseases by roughly 50 percent between 1970 and 2000 (CDC, 1999b; National Heart, Lung, and Blood Institute, 2012a). The decline in cardiovascular mortality entailed a substantial increase in adult life expectancy, which varied across states, depending on the initial prevalence of cardiovascular diseases. Therefore, this quasi-experimental source of variation allows the estimation of a differences-in-differences model where all states are treated though with varying treatment intensities. State-year observations for 1940–1960 constitute the pre-treatment and for 1970–2000 the post-treatment period.

The paper contributes to the literature in several ways. First, the empirical results establish a positive causal link between adult health, as measured by adult life expectancy, and age-specific wages per worker. The decline of cardiovascular mortality in the U.S. from 1968 onward led to an increase of life expectancy at 50 of approximately 3.16 years, or roughly two thirds of the increase between 1960 and 2000. According to the baseline estimation, this rise in life expectancy caused an increase of average gross wages for the group of the 45 to 54 year olds of roughly 9,762$, or 31 percent of initial wages in 1960. This wage hike corresponded to 47 percent of the wage change observed in the same time window. Furthermore, the results reveal that wage gains accrued to workers in the prime-age group between 25 and 54 as well as old-age workers above 65. Compared to earlier generations, the life-cycle earnings profile of an average worker, thus, increases more steeply at younger ages, while it flattens out more slowly at higher ages. Figure 1 illustrates this shift for wages of U.S. whites born between 1916 and 1955 grouped in ten-year cohorts. Overall, this pattern is consistent with a workforce that over time becomes healthier at any given age, and at higher ages in particular.

Another contribution is the focus on the role of measurement of health conditions in the context of age-specific outcomes. In many studies, life expectancy, a one-dimensional summary measure of the survival experience of the population, serves as a proxy for the average health status of the population of interest, for example, the total workforce. In such a case, one implicitly assigns all individuals the same health status (or change thereof). This assumption may produce severe systematic measurement error, if the chosen proxy does not closely reflect

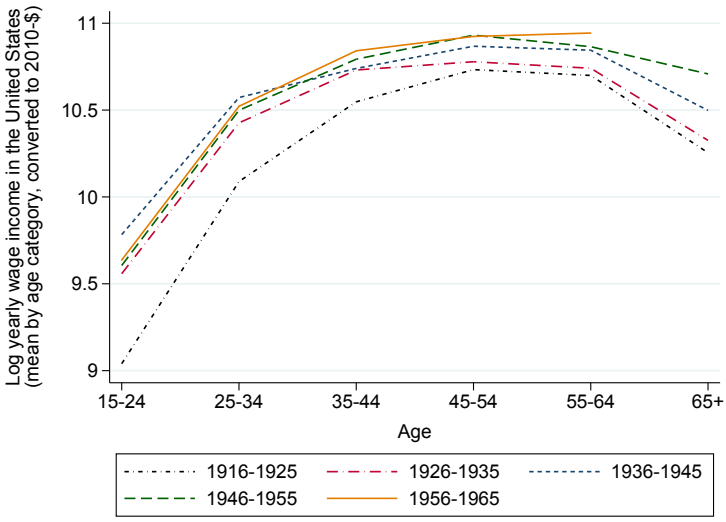


Figure 1: Life-Cycle Wage Profiles by Birth Cohort

Data source: IPUMS (Ruggles et al., 2015).

the health conditions of the population of interest. For example, consider the third stage of the epidemiological transition during which life expectancy at birth substantially increased thanks to reduced infant mortality following the invention of vaccines and antibiotics (WHO, 2008). As important as this health shock was, it may grossly overstate the health improvement for the median American who is around age 30 at this time.[1] This type of mismeasurement introduces a systematic correlation between the proxy of the health shock and the error term, thus leading to biased estimates. In particular, mismeasurement leads to downward-biased estimates if the change in average population health is overstated. Therefore, age-specific heterogeneity in the effect of health shocks and mismeasurement might be a reason for null results of life expectancy at birth on GDP per capita found by Acemoglu and Johnson (2007, 2014), Hansen (2014) and Bloom, Canning, and Fink (2014). Even though these papers use the mortality rate from infectious diseases as instrumental variable for life expectancy at birth, they cannot mend the measurement problem since the first stage again overstates the health improvement for the median person. Hence, the published estimates can be considered a lower bound for the causal effect of health improvements on economic growth.

Lastly, this paper can make progress in analyzing potential channels through which adult health affects average wages by using individual data on health outcomes and economic variables, as well as by combining data on longevity from vital statistics with census data on wages, educational attainment, and labor supply. In particular, U.S. states provide a favorable setting, because the institutional environment for the labor market is homogeneous in contrast to cross-country studies. In addition, there is no binding statutory retirement age in the U.S., which offers a clearer picture of productivity and labor supply for old-age workers above 65 compared to other developed countries.[2] The timing of wage hikes suggests that potential channels are health

[1] The median age of the U.S. population was 29.0 in 1940 and 30.2 in 1950 (Hobbs and Stoops, 2002).

[2] Nonetheless, certain age thresholds may still affect the timing of retirement. In particular, Americans become eligible for Medicare at age 65; full Social Security benefits can be claimed around age 66 depending on birth cohort; and there exist no further monetary incentives for delaying retirement beyond age 70.

improvements, in particular in the short-run, and higher educational attainment and changes in individual behavior toward a more healthy lifestyle in the long-run. In contrast, adjustments in labor supply cannot explain the wage increase since labor force participation rates as well as usual working hours and weeks either declined or remained unchanged during the treatment period. Moreover, heterogeneity in age group estimates preclude the possibility of unilateral indirect wage effects through out-selection or increased bargaining power. Thus, thanks to higher adult life expectancy, workers earn more, invest more in educational attainment, but work slightly less. This evidence confirms theoretical predictions and results from simulation exercises by Cervellati and Sunde (2013) and Strulik and Werner (2016). They show that individuals may invest more in schooling and, at the same time, reduce lifetime working hours, if leisure time while at work and consumption over the life-cycle increase.[3] Therefore, higher lifetime labor supply is not a necessary condition for increased educational attainment, as was claimed by Hazan (2009).

The paper's main result of a positive association between adult health and average wages per worker also holds for long-differences models and specifications that either use a shorter pre- or post-treatment window, or both. Furthermore, the econometric model accounts for initial state-level differences in income, education, and the rural-urban gradient, as well as state-fixed effects and differential time trends across census regions. Moreover, robustness tests show that sub-state level heterogeneity in the prevalence of cardiovascular diseases or interstate migratory patterns are unlikely to produce a spurious correlation between adult life expectancy and average wages on the state level. Finally, the analysis reveals heterogeneity in the beneficial effects of health improvements on average wages between rural and metropolitan areas as well as different occupational groups.

This paper relates most closely to work by Hansen and Strulik (2017), who investigate the link between adult health and college enrollment of 18 to 24 year old Americans by also exploiting variation from the cardiovascular revolution across U.S. states. Instead, this study examines specifically how adult health affects average wages of different age groups by using cohort-specific variation over time. In particular, this approach uncovers age-specific heterogeneity with respect to the causal effect of health gains on wages, which, otherwise, could not be detected: Gains in adult health exert a positive effect on wages for workers aged 25–54 but not for workers aged 55–64. This finding is also consistent with a side result of Hansen and Strulik (2017), who find no causal effect of adult life expectancy on wages pooling variation for workers aged 30–65. Furthermore, both papers complement each other: According to the results presented in this study, education constitutes one potential channel through which health improvements increase worker wages in the long-run; however, education cannot explain hikes in wages immediately following the treatment.[4] Based on micro data, this study additionally finds that health innovations have marginalized negative effects of cardiovascular diseases on individual income over relatively short time. This result indicates significant positive health effects on average wages per worker. Other closely related work is from Bleakley (2007) and Bhalotra and Venkataramani (2015), who exploit

[3] This finding is also consistent with work by d'Albis, Lau, and Sánchez-Romero (2012), who demonstrate that gains in life expectancy may lead to earlier retirement given that mortality reductions occur at sufficiently young age to provide substantial increases in individual's expected lifetime human wealth.

[4] As an internal consistency check, I re-estimate the effect of adult life expectancy on college enrollment using the baseline specification of this paper. The resulting parameter estimates are quantitatively similar to those of Hansen and Strulik (2017).

similar empirical strategies to identify positive long-run effects of health improvements during childhood on adult education, income, and labor supply. In contrast to their work, this study focuses on a health shock that predominantly affects adults and that, due to the nature of cardiovascular diseases, unfolds heterogeneous effects across age groups.

Furthermore, this study relates to a large macro literature that has investigated the effect of aggregate health measures on economic outcomes. In particular, reductions in mortality and gains in longevity foster per capita income growth (Bloom, Canning, and Sevilla, 2004; Lorentzen, McMillan, and Wacziarg, 2008; Cervellati and Sunde, 2011; Strittmatter and Sunde, 2013; Hyclak, Skeels, and Taylor, 2016); are conducive to investment in educational attainment (Tamura, 2006; Jayachandran and Lleras-Muney, 2009; Hansen and Strulik, 2017); spur old-age savings (Bloom, Canning, and Graham, 2003; De Nardi, French, and Jones, 2009); and reduce fertility (Hansen, Jensen, and Lønstrup, 2014; Ager, Hansen, and Jensen, 2018). In addition, these channels potentially interrelate closely (Zhang and Zhang, 2005). This paper provides a cohort-based analysis of the effect of adult health on average wages, which is novel to this literature. By focusing on different age groups, the empirical analysis uncovers that health improvements benefit prime-age workers between 25 and 54 as well as old-age workers above 65. Therefore, the cohort analysis allows to track shifts in the life-cycle earnings profile of the average worker that follow from the standard theories of human capital by Mincer (1958) and Ben-Porath (1967). Related work reports mixed results on the change of life-cycle earnings profiles for specific occupational groups. For example, Jones and Weinberg (2011) find that creativity peaks of researchers as measured by scientific breakthroughs have shifted to higher ages. In contrast, evidence by Falck, Heimisch, and Wiederhold (2016) indicates that introduction of information and communication technologies might shift productivity peaks to younger ages. The evidence in this paper implies a steeper life-cycle profile at younger ages, which flattens out more slowly at higher ages. An optimistic interpretation of this result suggests that health gains for prime-age and old-age workers might boost aggregate productivity for aging societies and, thus, confine potentially adverse effects of demographic change. Therefore, this paper also connects to a strand of the literature that investigates the role of demographic change for past and future development, for example, Feyrer (2007), Sánchez-Romero (2013), Cuaresma, Lutz, and Sanderson (2014), and Kotschy and Sunde (2018).

The remainder of this paper is structured as follows. Section 2 presents background information on health improvements during the cardiovascular revolution. Section 3 introduces the data as well as the empirical framework and discusses key identifying assumptions. Section 4 presents the estimation results and examines potential channels through which adult health may affect average wages. Finally, Section 5 concludes.

2 Background: The Cardiovascular Revolution

Over the course of the twentieth century, the United States experienced substantial improvements in public health leading to a marked increase in life expectancy. In particular, these improvements came down to two separate waves of medical breakthroughs: the epidemiological transition and the cardiovascular revolution. Figure 2 depicts the decline in mortality rates for infectious and cardiovascular diseases in the U.S. which resulted from these events.

The invention of antibiotics and vaccines in the first half of the twentieth century initiated a sharp reduction in mortality from communicable infectious diseases, which was especially pronounced for infants and children. The inventions during this period, which was termed the third stage of the epidemiological transition (Omran, 1971), caused an exceptional increase in life expectancy at birth, however, a significant but in comparison modest gain for higher ages.

The pointed increase in old-age life expectancy had to wait until the second wave of medical innovations around 1960, which was labeled cardiovascular revolution and identified as fourth stage of the epidemiological transition (Olshansky and Ault, 1986; Omran, 1998). The unexpected invention of new treatment possibilities for the non-communicable cardiovascular diseases boosted life expectancy predominantly through a decrease or delay in old-age mortality. Cardiovascular diseases become more likely as the tissues of the cardiovascular system age and lose some of their flexibility (Kirkwood, 2001). Therefore, mortality rates from cardiovascular diseases increase steadily with age, as exemplified by Figure 3.

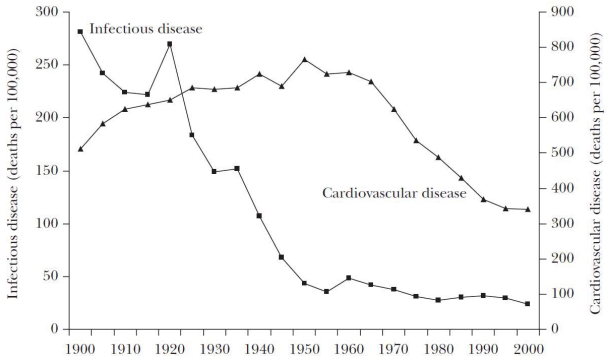
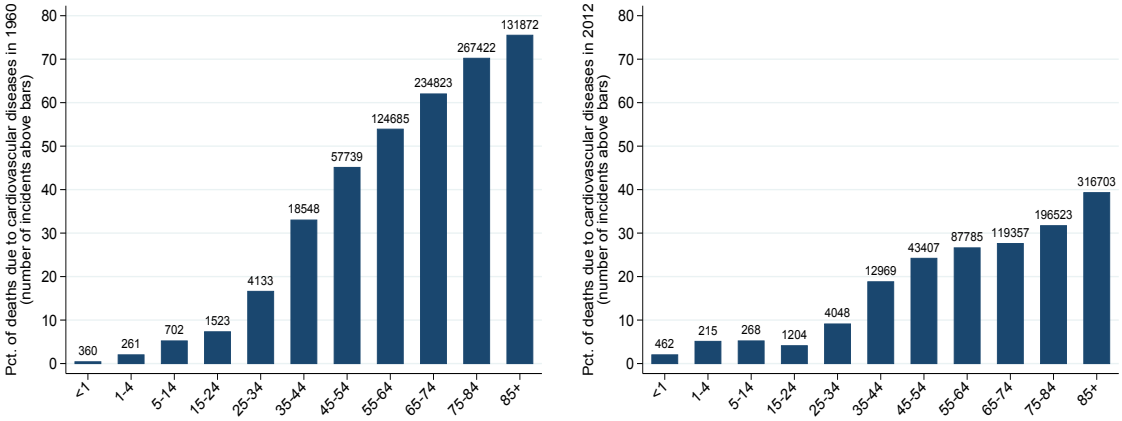


Figure 2: Mortality Rates from Infectious and Cardiovascular Diseases

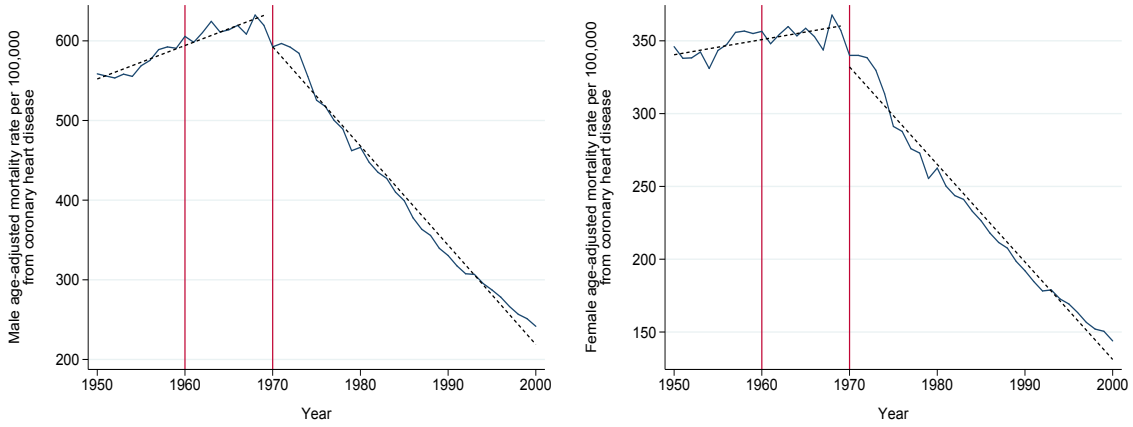
Source: Cutler, Deaton, and Lleras-Muney (2006).



(a) 1960 (b) 2012

Figure 3: Percent of Deaths that are Attributable to Cardiovascular Diseases

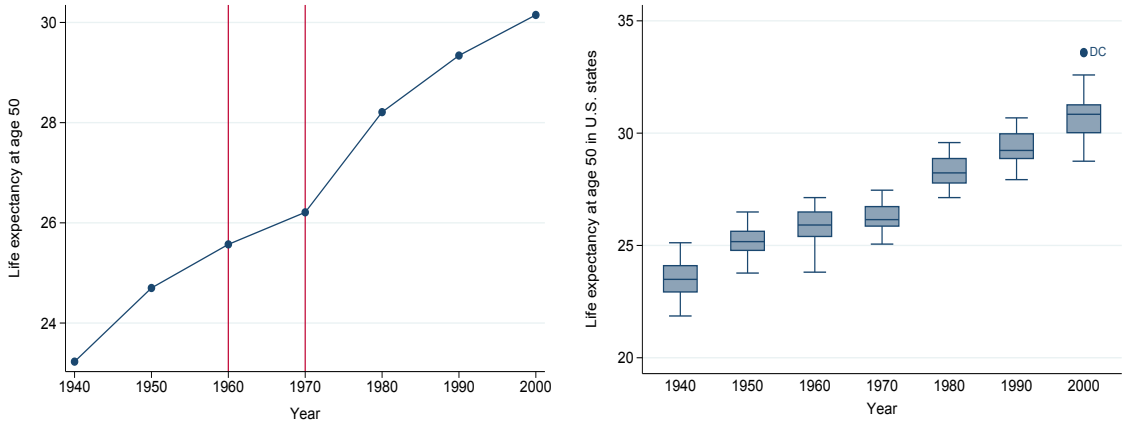
Data sources: National Center for Health Statistics (1963) and National Heart, Lung, and Blood Institute (2012b).



(a) Males (b) Females

Figure 4: Age-Adjusted Mortality from Cardiovascular Diseases

Data source: National Heart, Lung, and Blood Institute (2012a).

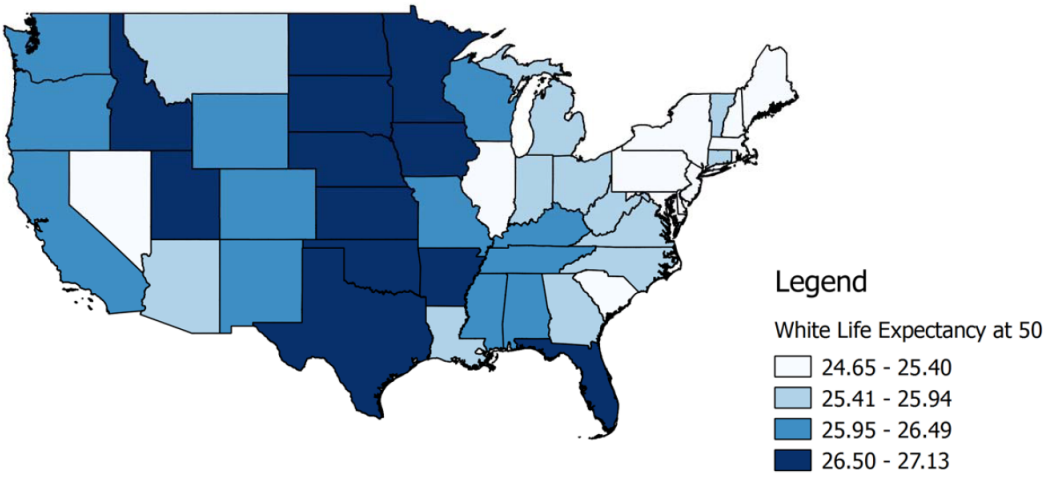


(a) United States (b) States

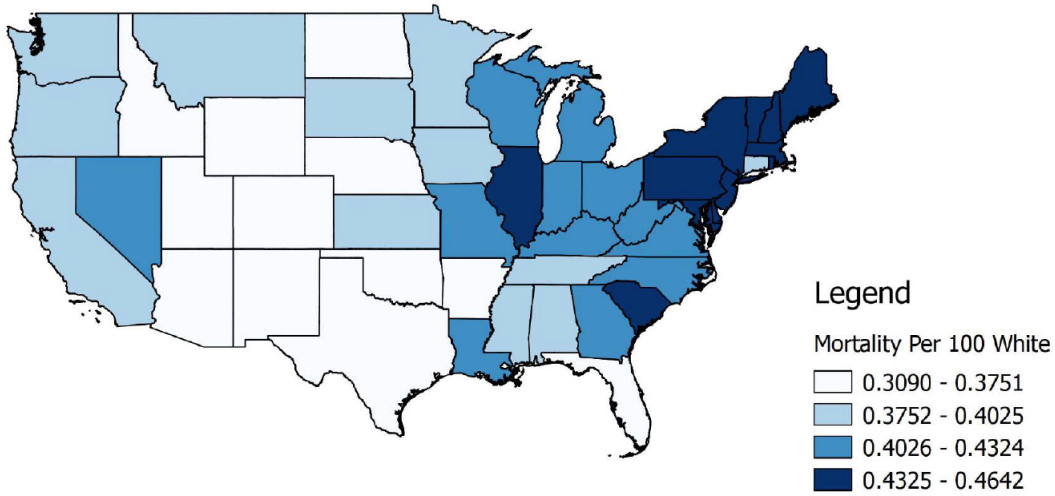
Figure 5: Life Expectancy at Age 50 in the United States

Data sources: National Center for Health Statistics (2017b).

The cardiovascular revolution was successful in considerably reducing the mortality rates from a broad spectrum of cardiovascular illnesses; for example, coronary heart disease, which in 2000 still accounted for approximately twelve percent of total deaths in the U.S. (National Center for Health Statistics, 2017a), and which in 2004 still was the most common cause of death in high-income countries (WHO, 2008). Figure 4 showcases how powerful the decline in mortality from cardiovascular diseases was: Between the peak levels in 1968 and the year 2000, mortality from coronary heart disease fell by roughly two thirds for both, men and women. As Figure 3 portrays, the number of incidents dropped for all age groups, except for infants and those above age 85, although the median age of the population had increased from 29.5 to 35.3 years during this period (Hobbs and Stoops, 2002). The decline was especially pronounced for individuals in the age range 35–84, thus especially boosting adult life expectancy as illustrated by Figure 5. In



(a) Life Expectancy at 50



(b) Mortality from Cardiovascular Diseases

Figure 6: Mortality from Cardiovascular Diseases and Life Expectancy at 50

Data source: Grove and Hetzel (1968).

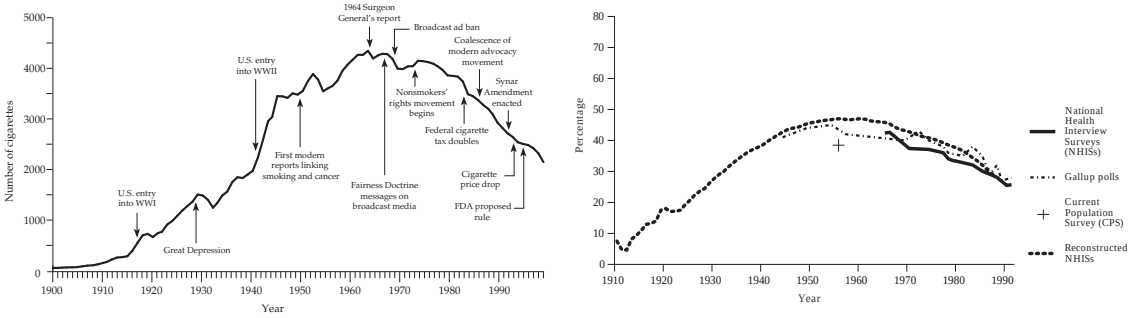
contrast, for the group above age 85, the number of incidents more than doubled during this period; however, the overall share of deaths that is attributable to cardiovascular diseases halved from almost 80 to slightly below 40 percent. One reason was that newly introduced drugs and treatment methods delayed the critical point at which the cardiovascular disease became lethal, so that incidents occurred either at a higher age, or death originated from other sources as, for example, cancer. Importantly, there have been striking geographic differences in the prevalence of cardiovascular diseases across U.S. states, which above all were rooted in social, cultural, and environmental factors (CDC, 1999b). The initial prevalence of cardiovascular diseases determined how beneficial the treatment was for states. Hence, the decline in mortality and consequently the increase in adult life expectancy varied across states. Figure 6 displays the geographic differences in life expectancy at 50 and mortality from cardiovascular diseases in the year 1960.

Reductions in mortality from cardiovascular diseases arrived through two channels. First, a number of medical innovations between the years 1960 and 1970 allowed to prevent certain diseases or to treat the symptoms. The most remarkable among these inventions were the artificial cardiac pacemaker, which was first implanted in 1958; the application of chest compression to restore blood circulation in a person that is in cardiac arrest beginning in 1960; the invention of the beta blocker in 1962, which is used to lower blood pressure and to treat cardiac arrhythmia; the invention of the portable defibrillator in 1959 and its application in the U.S. from 1966 onward; and the first adult human heart transplantation in the U.S. in 1968. Subsequent innovations include first thrombolytic therapies in 1986 to treat myocardial infarction, stroke, and pulmonary embolisms; the invention of cholesterol lowering statins, first marketed in 1987; and beginning in 1988, the implantation of intravascular stents to address acute closure of arteries and blood vessels. These new treatments improve health relatively quickly, as, for example, serum cholesterol reducing drugs which achieve their full effect within five years (Law, Wald, and Thompson, 1994). These advances in the available technology were complemented by an increasing number of specialists and care centers for cardiovascular diseases (CDC, 1999b).

The second channel for the decline in mortality constituted increased awareness of major individual risk factors and changes in behavior. Research results by Keys et al. (1963), Keys (1980), and Dawber (1980) established, among others, high blood cholesterol, high blood pressure, physical inactivity, smoking, obesity, and unbalanced diet as major risk factors for cardiovascular diseases.[5] The federal government initiated national programs to educate specialists and the general public about risks of high blood pressure in 1972; of high blood cholesterol in 1985; and of the importance of cardiovascular health in 1989 (CDC, 1999a). This increased awareness helped raise the share of patients with too high blood pressure who have their condition treated and under control (CDC, 1999b). Moreover, the report of the Surgeon General in 1964 (U.S. Department of Health, Education, and Welfare, 1964) highlighted the adverse effects of smoking on health, later followed by increased cigarette taxes in the 1980s (CDC, 1999a). Preventive measures against smoking were particularly successful, as Figure 7 shows. The share of smokers in the adult population was declining from 1960 onward, while the per capita cigarette consumption started to rapidly fall during the 1970s. Due to the cumulative damage from smoking, however, it takes about ten years after cessation until the risk of cardiovascular disease for former smokers reaches the same level as for non-smokers (Oza et al., 2011). Alcohol consumption, another risk factor if enjoyed in excess (Marmot and Brunner, 1991; Murray et al., 2002), only started to decline after 1980 as Figure 8 reveals. Therefore, the positive effects of behavioral changes probably started only as early as the 1980s. Public health education, however, also had its limits. Even though the health risks were known, physical activity declined between 1970 and 2010, while the share of obese persons doubled (Flegal et al., 1998; CDC, 2001; Kohl and Cook, 2013).

The unexpected and concentrated surge of medical breakthroughs in the 1960s and the massive preventive efforts thereafter motivate a pre-treatment period until 1960 and post-treatment from 1970 onward in the estimation sample at hand. The next section discusses the empirical framework and the data.

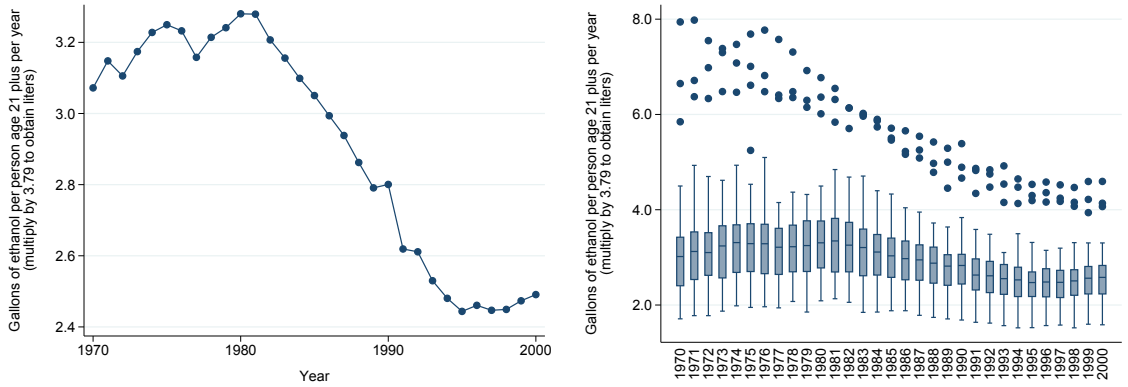
[5] According to Ezzati and Riboli (2012), high blood pressure and high blood cholesterol alone account for one half of the global incidence of coronary heart disease. Too high body weight and smoking are responsible for another 20 and 13 percent.



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

Figure 7: Smoking in the United States

Sources: U.S. Department of Health and Human Services (1998, 2000).



(a) United States (b) States

Figure 8: Alcohol Consumption in the United States

Data source: Haughwout and Slater (2017)

3 Data and Empirical Framework

3.1 Data

The empirical analysis is based on a balanced ten-year panel of the 48 contiguous states of the U.S. for the period 1940–2000. Correspondingly, the estimation sample comprises 336 observations in total. Alaska and Hawaii are excluded because of missing data for early periods, the District of Columbia is omitted due to its special nature of a metropolitan region. Since life expectancy in 1940 is only available for whites, the entire sample is restricted to the white population.[6]

Data on gross wages, labor market outcomes, and educational attainment stem from individual data in decennial U.S. censuses (IPUMS) by Ruggles et al. (2015). Wages are adjusted for inflation and measured in logarithms. The variable comprises wages, salaries, commissions, cash

[6]Table A1 in the Appendix reports descriptive statistics for age-specific groups as well as the entire population.

bonuses, tips, and other money income received from an employer. Labor market outcomes cover individual labor force participation; usual hours worked per week; usual weeks worked per year; and usual hours worked per year, which are constructed by multiplying weekly hours with work weeks. Working weeks are not available as continuous measures in 1960 and 1970, while the series on usual working hours only starts in 1980. Intervalled hours and weeks, however, are available throughout all time periods. For this reason, I construct a continuous measure for weeks and hours from bivariate regressions of the continuous on the intervalled measure. For the cohort-specific analysis, hours and weeks are constructed based on age-specific regressions.[7] The share of college graduates is constructed from the number of individuals who attended at least four years of college in their life relative to the entire number of individuals in the sample.[8] To ensure representativity, the data are collapsed to the state level using person sample weights.

Data on life expectancy are obtained from the U.S. decennial life tables and vital statistics provided by the National Center for Health Statistics (2017b) of the Centers for Disease Control and Prevention (CDC). Adult life expectancy enters the estimation equation in logarithms.[9] In 1960, adult life expectancy differed considerably between U.S. states as shown by Panel (a) of Figure 6. Notably, white life expectancy at age 50 varied by 2.48 years between Florida, the state with the highest value, and Nevada, the state with the lowest value. Moreover, adult life expectancy was high in the West North Central and West South Central census regions, whereas it was comparatively low in New England and the Atlantic regions.

In order to capture the exogenous increase in adult life expectancy due to innovations in medical technology, the analysis exploits state differences in mortality from cardiovascular diseases prior to their introduction, i.e., in 1960, as instrument for adult life expectancy. Age-adjusted cardiovascular mortality in 1960 is obtained from Grove and Hetzel (1968) and expressed in deaths per 100 whites.[10] Panel (b) of Figure 6 illustrates spatial differences in the prevalence of cardiovascular diseases as measured by mortality in 1960. The data reveal a strong negative unconditional correlation between adult life expectancy and mortality from cardiovascular diseases: Life expectancy at 50 was high in the census regions, where mortality rates were comparatively low, and vice versa. As shown in Section 2, age-adjusted mortality from coronary heart disease did not decrease until shortly before 1970 – in fact, it even slightly increased between 1950 and 1968. Only from this point on, mortality from coronary heart disease decreased substantially.[11] For the baseline specification, innovations in medical technology are, thus, coded to occur from 1970 onward. This designates the time intervals 1940–1960 as pre-treatment and 1970–2000 as post-treatment periods ('differences-in-differences model'). In a more flexible specification,

[7] This procedure will lead to downward-biased standard errors in the labor supply regressions, because the missing data points are replaced by fitted values from the corresponding regressions. The respective estimates, however, reveal no significant (positive) effect of adult life expectancy on labor supply, so that this bias does not translate to inference.

[8] Results are qualitatively and quantitatively unchanged, if educational attainment contains all individuals who enrolled in college for at least one year.

[9] Results are qualitatively and quantitatively similar if, instead of a log specification, life expectancy enters the estimation framework directly.

[10] Age-adjustment allows to compare the mortality rates between states even if they have different age structures. Due to the adjustment, the mortality rates should not be interpreted as crude rates, unless a state exhibits the same age structure as the standard population. For this reason, not the absolute figures of mortality from cardiovascular diseases are of importance but the relative change over time.

[11] Declines in mortality rates and, thus, improvements in adult life expectancy slightly lag behind the actual development for the average person since medical innovations might come too late for the very ill and the very old.

mortality from cardiovascular diseases in 1960 is interacted with a full set of year dummy variables ('flexible model').

Importantly, adult life expectancy and mortality rates provide a conservative view on the effect of health improvements on wages, because they cannot fully capture morbidity reductions following the cardiovascular revolution. In the absence of better health measures across U.S. states and time, they nonetheless represent the best option.

In order to ensure that initial mortality from cardiovascular diseases is as good as randomly assigned, further controls interacted with the treatment indicator are added. These controls comprise initial life expectancy by the CDC; initial income and the initial share of college graduates, both obtained from IPUMS by Ruggles et al. (2015); initial population density by Hobbs and Stoops (2002); and initial mortality from non-cardiovascular diseases by Grove and Hetzel (1968). Current values of the corresponding variables are not included, because they might themselves be affected by treatment and, thus, constitute bad controls.

Due to the collapsing process, wages, education, and labor supply are grouped on the state level. For this reason, I weight all regressions by the group size, i.e., the initial white population of a specific age cohort or of the total population.[12]

Finally, I further exploit the Health and Retirement Study (HRS, 2017) to investigate whether improvements in adult health contributed to wage increases. This data set 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. Values are adjusted for inflation using the annual urban Consumer Price Index by the Bureau of Labor Statistics and measured in logarithms. Individual health is proxied by binary indicators, which indicate whether study participants have ever been diagnosed with heart problems or high blood pressure.

3.2 Empirical Framework

This section introduces the empirical framework to study the effect of changes in adult life expectancy on wages per worker and labor market outcomes. The structural model reads

$$y_{s,t} = \alpha x_{s,t} + w_s' \mathcal{I}_t^{1960} \beta + \gamma_s + \delta_t + \zeta_{r,t} + \varepsilon_{s,t}, \tag{1}$$

where $y_{s,t}$ denotes the outcome measure of interest (e.g., wages) for state s and time period t; $x_{s,t}$ represents log life expectancy of the age group under consideration; w_s is a vector of controls measured in 1960, interacted with the indicator matrix $\mathcal{I}_t^{1960}$, whose values take unity from 1970 onward and zero else; γ_s and δ_t denote state-fixed and time effects; $\zeta_{r,t}$ describes region-year interactions, which control for differential development trends across the nine U.S. census-regions r; and $\varepsilon_{s,t}$ constitutes an idiosyncratic error term.

Due to omitted variables and reverse causality, log life expectancy is likely endogenous. In order to uncover the causal link between adult health and average wages, I exploit heterogeneity

[12] Since the population equation of interest is the effect of improved health conditions on individual wages and labor supply, 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).

in the prevalence of cardiovascular diseases across U.S. states as exogenous source of variation for instrumentation. The first-stage equation is given by

$$x_{s,t} = \eta z_s d_t^{1960} + w_s' \mathcal{I}_t^{1960} \theta + \kappa_s + \lambda_t + \mu_{r,t} + \xi_{s,t}, \tag{2}$$

where mortality from cardiovascular diseases in 1960, z_s, is interacted with the post-treatment indicator d_t^{1960}; κ_s and λ_t denote state-fixed and time effects; $\mu_{r,t}$ describes census-region-year effects; and $\xi_{s,t}$ constitutes the error term.

Conceptually, the first stage equation compares differences in the increase of adult life expectancy to differences in the decline of mortality from cardiovascular diseases between the pre-treatment and post-treatment period across states. For this reason, it corresponds to a differences-in-differences approach, where all states are treated but with different treatment intensities. Moreover, the first stage has a natural interpretation in this context: A decline in the mortality rate from cardiovascular diseases initiates an increase in adult life expectancy, which, in turn, affects the economic outcomes of interest in the structural model.

For initial mortality from cardiovascular diseases to be a valid instrument, several conditions must be fulfilled. First, initial mortality must be as good as randomly assigned conditional on covariates. This assumption requires the instrument (initial cardiovascular mortality interacted with the treatment indicator) to be independent of potential outcomes and potential treatment assignments, given the complete set of covariates. To this end, the baseline specification contains controls for initial state levels of income, the share of college graduates, and population density. These controls take up state-level selection toward more health, which is attributable to disparities in income, educational attainment, or the rural-urban discrepancy between densely populated states at the coasts and spacious states in the middle of the country. Moreover, initial non-cardiovascular mortality and initial life expectancy control for the health environment prevailing before new medical treatment technologies for cardiovascular diseases were introduced. Finally, state-fixed, time, and region-year fixed effects eliminate systematic state- and region-level variation due to differences in further social, cultural, or environmental factors that do not vary concomitantly over states and time, or that possess time-varying influences on mortality from cardiovascular diseases. In particular, these trends cancel out differentials between census regions.

Furthermore, mortality from cardiovascular diseases must affect outcomes only through the first stage; that is, through the channel of health and longevity. Accordingly, mortality from cardiovascular diseases is not part of the structural model. This exclusion restriction is fundamentally untestable. It would be violated if changes in cardiovascular mortality rates were to affect the outcome of interest through a channel other than adult health as measured by adult life expectancy. Since the instrument is specific to the channel of health on the aggregate level, however, this assumption should plausibly be fulfilled in the context of this paper. Additionally, the empirical model accounts for initial differences in non-cardiovascular mortality to prevent the instrument from taking up beneficial effects attributable to medical advancement in the treatment of other diseases.

Finally, changes in mortality from cardiovascular diseases must be predictive of changes in adult life expectancy. Sufficiently high values of the first-stage F-statistics demonstrate that this assumption is fulfilled for the differences-in-differences model.

I also report results for a flexible model, in which the instrument is interacted with year dummies instead of the post-1960 treatment indicator. In this case, the first stage corresponds to

$$x_{s,t} = \sum_{\tau=1940}^{2000} \eta_\tau z_s d_t^\tau + w_s' \mathcal{I}_t^\tau \theta + \kappa_s + \lambda_t + \mu_{r,t} + \xi_{s,t}. \tag{3}$$

4 Results

This section presents the empirical results. First, I report evidence on the first-stage correlation between mortality from cardiovascular diseases and adult life expectancy as well as evidence on the reduced form effect of mortality on average wages. After having statistically established these relationships, I show results from two-stage least squares estimates for wages by age group and the total workforce.[13] Finally, this section concludes by investigating potential channels for a causal positive link between health improvements and wages.

4.1 First-Stage Evidence: Mortality and Adult Life Expectancy

First, I investigate the first-stage association between mortality from cardiovascular diseases and adult health conditions, proxied by log life expectancy at age 50. The analysis is based on the ten-year panel of the 48 contiguous U.S. states from 1940 to 2000 described in Section 3.1 with pre-treatment periods 1940–1960 and post-treatment periods 1970–2000. Table 1 reports least squares results for the differences-in-differences model from (2) in Panel (a) and for the flexible model from (3) in Panel (b).[14]

Column (1) shows results without covariates. In this case, initial mortality from cardiovascular diseases (interacted with the treatment indicator) and life expectancy at 50 correlate positively. This result is, however, driven by the omission of initial life expectancy. Based on how life tables are constructed, mortality rates and life expectancy must correlate negatively. Moreover, given better initial health conditions, there is less scope for future reductions in the mortality rates and, consequently, less potential for future improvements in life expectancy. Correspondingly, changes in life expectancy and initial life expectancy also correlate negatively. Therefore, the respective estimate is biased upward. Once, initial life expectancy is included as additional control for Columns (2) to (5), the sign turns negative. Accordingly, the larger the decline of cardiovascular mortality, the higher the gain in life expectancy. Column (3) adds initial mortality from non-cardiovascular diseases as control, which improves the fit of the first stage. Furthermore, this measure precludes that the instrument takes up health improvements that cannot be attributed to the cardiovascular revolution. This leads to a slightly more negative point estimate. Column (4) adds region-year fixed effects that eliminate systematic trends reflecting economic, social, or cultural differences across U.S. census regions. Finally, the full specification in Column (5) adds additional controls for the initial share of college graduates, initial population density, and initial income. These variables cancel out any variation in life expectancy originating from initial differences in education and development. The corresponding point estimate takes a value of

[13] Table A2 in the Appendix reports estimates from ordinary least squares (OLS).

[14] Since life expectancy at age 50 on average provides the most accurate picture of health for workers around that age, the sample is weighted by the initial white population of the 45 to 54 year olds. The results are unaltered if, instead, weighted for the entire white population.

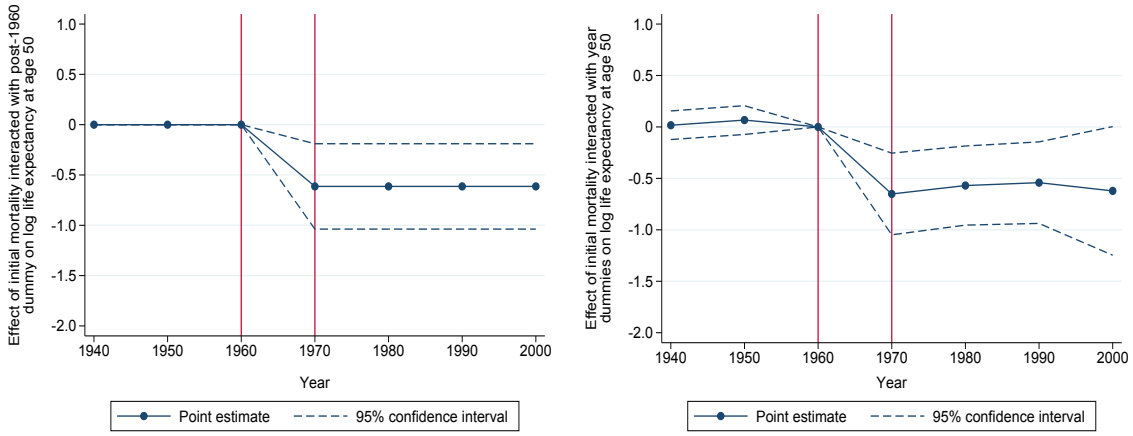
Table 1: First Stage: Effect of Mortality on Adult Life Expectancy

	Dependent variable: log life expectancy at age 50				
	(1)	(2)	(3)	(4)	(5)
	(a) Differences-in-differences model				
Mortality × Post 1960	0.50***	-0.53***	-0.86***	-0.77***	-0.61***
	(0.11)	(0.15)	(0.17)	(0.20)	(0.21)
	(b) Flexible model				
Mortality × 1940	-0.13*	-0.13*	-0.15*	0.04	0.02
	(0.08)	(0.08)	(0.08)	(0.08)	(0.07)
Mortality × 1950	-0.05	-0.05	-0.05	0.10	0.07
	(0.05)	(0.05)	(0.05)	(0.06)	(0.07)
Mortality × 1970	0.21***	-0.82***	-1.16***	-0.78***	-0.65***
	(0.05)	(0.16)	(0.17)	(0.19)	(0.20)
Mortality × 1980	0.34***	-0.69***	-1.03***	-0.70***	-0.57***
	(0.07)	(0.15)	(0.17)	(0.19)	(0.19)
Mortality × 1990	0.50***	-0.54***	-0.88***	-0.64***	-0.54***
	(0.09)	(0.16)	(0.18)	(0.19)	(0.20)
Mortality × 2000	0.71***	-0.32*	-0.65***	-0.75***	-0.62*
	(0.15)	(0.18)	(0.21)	(0.25)	(0.31)
Controls in 1960 × Post 1960:					
Initial life expectancy		✓	✓	✓	✓
Initial mortality (not CVD)			✓	✓	✓
Initial share college					✓
Initial population density					✓
Initial income					✓
Region-year FE				✓	✓
FE & TE	✓	✓	✓	✓	✓
States	48	48	48	48	48
Observations	336	336	336	336	336

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of 45 to 54 year olds. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

-0.61 and is significant at the one percent level. Given the quasi-natural source of variation, the parameter has a quantitative interpretation: A reduction of mortality from cardiovascular diseases of one person per 1,000 whites leads to an increase in white life expectancy at 50 of approximately 6.1 percent. Taken at face value, the reduction in cardiovascular mortality by two persons per 1,000 whites (50 percent of its initial value) between 1960 and 2000, thus, led to an increase in life expectancy at 50 of approximately 3.16 years, or two thirds of the overall increase over this time period.[15] This number conforms closely to the increase in life expectancy of 3.27 years, which, according to Cutler, Deaton, and Lleras-Muney (2006), can be attributed to medical advancement in the treatment of cardiovascular diseases between 1960 and 2000.

[15] To arrive at these figures, compute $\Delta_x = \widehat{\eta} \cdot \Delta_z \cdot \bar{\mu}_x = (-0.61) \cdot (-0.20) \cdot 25.91 \approx 3.16$, where $\bar{\mu}_x$ is evaluated at the sample mean in 1960, and $\Delta_x / (x^{2000} - x^{1960}) = 3.16/4.76 \approx 0.66$.



(a) DD Model (Table 1a, Column 5)

(b) Flexible Model (Table 1b, Column 5)

Figure 9: Illustration: First Stage

The flexible model in Panel (b) reports point estimates for the instrument interacted with year dummies for every period and the year 1960 as reference category. Mortality affects life expectancy negatively in the post-treatment period 1970 to 2000 for all specifications that control for initial longevity. In Column (5), the parameters are estimated to be of similar quantity and significantly different from zero. In contrast, there is no effect of mortality from cardiovascular diseases in 1960 on life expectancy for the pre-treatment periods 1940 to 1960.[16] Figure 9 plots the point estimates from the full specification in Column (5) with the corresponding 95-percent confidence interval for both models. The displayed coefficients of the flexible model in Panel (b) show a stable pattern for the pre-treatment and post-treatment period so that the assumption of a constant effect for each period in the differences-in-differences model appears appropriate.

4.2 Reduced-Form Evidence: Mortality and Average Wages

Table 2 reports reduced form estimates for the effect of mortality on wages for the 45 to 54 year olds. This age group is of particular interest for two reasons. On the one hand, these workers are usually considered the most productive group of the workforce for their high participation rate and their considerable experience. Therefore, these workers are usually at the peak of their life-cycle earnings profile as illustrated in Figure 1. On the other hand, they become increasingly susceptible to cardiovascular diseases due to aging and behavioral risk factors, while still being young enough to profit quite considerably from new treatment possibilities and changed behavior. For these reasons, this is one of the age groups, which might profit from medical innovations in terms of both, health and economic outcomes.[17]

Panel (a) shows the results for the differences-in-differences model. As for the first stage, additional controls are included for initial values of life expectancy, mortality from non-cardiovascular diseases, region-year fixed effects, the share of college graduates, population density, and income. The parameter estimate in the full specification in Column (5) takes a value of -1.56 and is

[16] Single outliers do not drive these partial correlations as Figure A1 in the Appendix shows.
[17] Table A3 in the Appendix reports qualitatively similar results for the entire workforce.

Table 2: Reduced Form: Effect of Mortality on Average Wages of Workers Aged 45–54

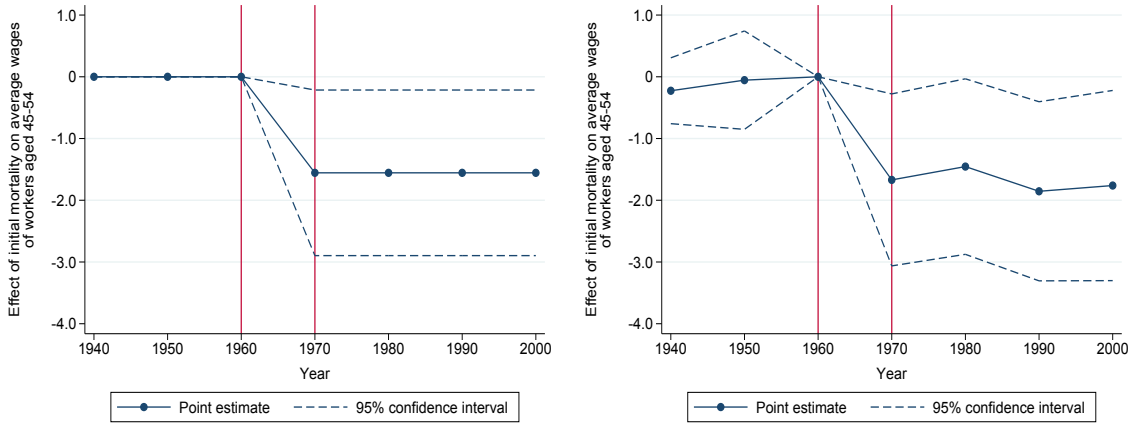
	Dependent variable: log wages of whites 45–54				
	(1)	(2)	(3)	(4)	(5)
	(a) Differences-in-differences model				
Mortality × Post 1960	-0.87***	-1.20**	-1.50***	-1.39***	-1.56**
	(0.21)	(0.45)	(0.53)	(0.49)	(0.67)
	(b) Flexible model				
Mortality × 1940	0.50*	0.50*	0.43	-0.43**	-0.23
	(0.27)	(0.27)	(0.27)	(0.20)	(0.27)
Mortality × 1950	0.38	0.38	0.34	0.23	-0.05
	(0.36)	(0.36)	(0.35)	(0.41)	(0.40)
Mortality × 1970	-0.42***	-0.76**	-1.06**	-1.39***	-1.67**
	(0.13)	(0.38)	(0.42)	(0.47)	(0.69)
Mortality × 1980	-0.85***	-1.18***	-1.50***	-0.98**	-1.45**
	(0.23)	(0.41)	(0.44)	(0.48)	(0.71)
Mortality × 1990	-0.42	-0.75	-1.09*	-1.70***	-1.85**
	(0.33)	(0.55)	(0.63)	(0.58)	(0.72)
Mortality × 2000	-0.60*	-0.93*	-1.30**	-1.73***	-1.76**
	(0.35)	(0.54)	(0.62)	(0.62)	(0.77)
Controls in 1960 × Post 1960:					
Initial life expectancy		✓	✓	✓	✓
Initial mortality (not CVD)			✓	✓	✓
Initial share college					✓
Initial population density					✓
Initial income					✓
Region-year FE				✓	✓
FE & TE	✓	✓	✓	✓	✓
States	48	48	48	48	48
Observations	336	336	336	336	336

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of 45 to 54 year olds. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

significant at the five percent level.[18] Given that the instrument is as good as randomly assigned, the coefficient estimate reflects the intention-to-treat effect. It measures the effect of being offered the treatment. Since not all individuals decide to take up the treatment (e.g., some patients do not take a beta blocker although they belong to high risk groups for stroke or cardiac arrest), the intention-to-treat effect is too low relative to the average treatment effect on the treated (Angrist and Pischke, 2009). According to the point estimate, a reduction of mortality from cardiovascular diseases by one person per 1,000 whites leads to a wage increase of 15.6 percent for the group of the 45 to 54 year olds.

In the full specification of the flexible model in Panel (b), there is no significant effect

[18]The sample is again weighted by the initial population of the 45 to 54 year olds. The resulting coefficient is quantitatively similar if weighted by the entire white population.



(a) DD Model (Table 2a, Column 5)

(b) Flexible Model (Table 2b, Column 5)

Figure 10: Illustration: Reduced Form

of cardiovascular mortality on average wages in the pre-treatment period, though the effect is significantly negative in the post-treatment period.[19] Figure 10 depicts the reduced-form estimates for the differences-in-differences and the flexible model. The flexibly estimated coefficients show again a stable pattern over time giving credibility to the simpler differences-in-differences model.[20]

4.3 Life Expectancy and Average Wages

The last two sections have established the existence of a first-stage correlation between mortality from cardiovascular diseases and adult life expectancy, and a reduced-form effect of mortality on wages for the 45 to 54 year olds. Now, I turn to the average treatment effect on the treated, which corresponds to the ratio of the intention-to-treat effect from the reduced form, and the first-stage estimand, which corresponds to the compliance rate. Using two-stage least squares (2SLS), this quantity can also be directly estimated by instrumenting log life expectancy at age 50 with mortality from cardiovascular diseases in 1960 interacted with the treatment indicator. Table 3 reports the estimated effect of life expectancy on wages of the 45 to 54 year olds.[21] Section 4.4 discusses the corresponding estimates for all age groups and the entire workforce.

The first column of Panel (a) shows estimates for the differences-in-differences model without any additional controls except state-fixed and time effects. In this case, an increase in life expectancy leads to a decline in wages of workers aged 45 to 54. As argued by Aghion, Howitt, and Murtin (2011) and Bloom, Canning, and Fink (2014), however, this specification is misspecified, because it omits initial life expectancy. In particular, initial life expectancy correlates with initial mortality from cardiovascular diseases and subsequent improvements in life expectancy. Furthermore, it concomitantly affects prospective wage gains. For a given reduction in mortality rates, the first stage, therefore, underestimates the corresponding improvement in life expectancy; in fact, the model suggests smaller improvements in life expectancy for states with higher initial prevalence of cardiovascular diseases. In addition, the reduced form underestimates

[19] Single outliers do not drive these partial correlations as Figure A2 in the Appendix shows.
[20] Figure A3 in the Appendix plots the corresponding reduced form parameters for the entire workforce.
[21] The sample is weighted by the initial white population of the 45 to 54 year olds.

Table 3: Adult Life Expectancy and Average Wages of Workers Aged 45–54

	Dependent variable: log wages of whites 45–54				
	(1)	(2)	(3)	(4)	(5)
	(a) Differences-in-differences model (2SLS)				
Log life expectancy at 50	-1.73***	2.26***	1.73***	1.81***	2.54***
	(0.40)	(0.84)	(0.61)	(0.57)	(0.95)
First stage F–statistic	62.5	26.6	44.6	36.6	14.2
	(b) Flexible model (2SLS)				
Log life expectancy at 50	-1.36***	0.69	0.75**	1.73***	2.35***
	(0.37)	(0.44)	(0.38)	(0.53)	(0.89)
First stage F–statistic	15.3	13.2	19.9	7.4	3.5
Hansen test p–value	0.09	0.03	0.04	0.2	0.8
	(c) Flexible model (LIML)				
Log life expectancy at 50	-1.47***	0.92	0.91**	2.05***	2.57**
	(0.41)	(0.57)	(0.44)	(0.66)	(1.00)
First stage F–statistic	15.3	13.2	19.9	7.4	3.5
Hansen test p–value	0.09	0.03	0.05	0.2	0.8
Controls in 1960 × Post 1960:					
Initial life expectancy		✓	✓	✓	✓
Initial mortality (not CVD)			✓	✓	✓
Initial share college					✓
Initial population density					✓
Initial income					✓
Region-year FE				✓	✓
FE & TE	✓	✓	✓	✓	✓
States	48	48	48	48	48
Observations	336	336	336	336	336

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of 45 to 54 year olds. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

the associated wage gains following the health improvements of the cardiovascular revolution. In combination, the omission of initial life expectancy results in a downward bias of the estimates. Correspondingly, initial life expectancy is included in all remaining specifications. The third column adds initial mortality from non-cardiovascular diseases to improve the fit of the first stage, leading to an increase of the Kleibergen-Paap F-statistic from 26.6 to 44.6. Moreover, this control prevents the mortality instrument from taking up beneficial effects of medical innovations that work through health channels other than the cardiovascular revolution. The last two columns additionally contain region-year fixed effects that take up differential trends in wages and life expectancy across U.S. census regions.

Finally, the full specification in Column (5) adds initial values of the share of college graduates, population density, and income – all interacted with the Post-1960 treatment indicator. Because there is a strong link between education and health (Grossman and Kaestner, 1997; Lleras-Muney, 2005), the initial share of college graduates is added to take up variation of life expectancy that is attributable to initial disparities in state-level education. Population density is included to account for the rural-urban gradient in cardiovascular diseases with a particularly high prevalence of coronary heart disease and hypertension in non-metropolitan areas (Pickle and Gillum, 1999; Cooper et al., 2000). Finally, initial income is added to account for a potential feedback effect from income to health (Ettner, 1996; Frijters, Haisken-DeNew, and Shields, 2005; Lindahl, 2005; Chetty et al., 2016). The corresponding coefficient estimate takes a value of 2.54 and is significant at the one percent level. Therefore, an increase in life expectancy at age 50 by one percent, ceteris paribus, causes a wage hike of 2.54 percent for the group of the 45 to 54 year olds. Thus, the average treatment effect on the treated is approximately 60 percent larger than the intention-to-treat effect from the reduced form. Taken at face value, the increase in life expectancy at 50 between 1960 and 2000 led to a hike in average gross wages of the 45 to 54 year olds of approximately 9,762$ per year, or around 47 percent of the overall increase over this time period.[22] The first-stage F-statistic shows a value of 14.2, which indicates a sufficiently strong instrument given the conventional cutoff level of 10.

Panels (b) and (c) present results for the flexible model, estimated with two-stage least squares and with a heteroskedasticity-robust version of limited information maximum likelihood (LIML) due to the small value of the F-statistic. The full specification in Column (5) reports point estimates that are quantitatively similar to those of the differences-in-differences model. Since the p-value of the Hansen test for overidentification takes a value of 0.8, the null hypothesis that all instruments provide the same information is maintained.

4.4 Heterogeneity Across Age Groups and Measurement

Before turning to the effect of adult life expectancy for other age groups and the overall workforce, consider measurement of health improvements in light of the demographic structure of the population; a point that has so far largely gone unnoticed by the literature. The convention is to use a specific measure to capture the effect of a positive health shock, e.g., life expectancy at a given age. By construction, this measure encompasses the expected remaining lifetime including all following age groups. Thus, it provides a gross approximation of expected health over the remaining part of the life cycle. Accordingly, one implicitly assigns the same health to all individuals in the population of interest. This assumption is overly restrictive and masks heterogeneous effects across age cohorts. Moreover, it may introduce a systematic measurement error into the empirical model that cannot be solved by the instrumentation strategy.

This deficit becomes clear in light of the cohort structure of the labor force. On average, young workers have a relatively good health and can still expect to live a high number of years. Meanwhile, average health of older workers is lower due to aging, so that their remaining expected

[22] To arrive at these figures, compute $\Delta_y = \widehat{\alpha} \cdot \Delta_x / x^{1960} \cdot \bar{\mu}_y \approx 2.54 \cdot 3.16/25.91 \cdot 31515.35 \approx 9762.82$ with $\Delta_y/(y^{2000} - y^{1960}) \approx \frac{9762.82}{20666.62} \approx 0.47$, and $\bar{\mu}_y$ evaluated at the mean in 1960. The corresponding estimates based on the reduced-form estimate are 5,996$, or around 29 percent of the overall wage hike. Table A4 in the Appendix reports similar results for an empirical model in which adult life expectancy enters linearly.

years of life are considerably smaller than for young workers. Correspondingly, a measure that predominantly captures the health of young workers would overstate the health of older workers and vice versa. Hence, there would be systematic measurement error.

Suppose, for example, one is interested in estimating the effect of health changes on average wages of the total workforce. In order to capture improvements in the health of the workforce, one might use the (log-) change in life expectancy at age 30. By assumption, every worker is assigned the expected health improvement of a thirty-year-old. This measure may over- or understate the average health improvement, depending on how the health shock under consideration affects the average health status of the different age groups. Hence, one does not use the exact measure $x_{s,t}$ in the empirical framework but

$$p_{s,t} = x_{s,t} + \nu_{s,t}, \tag{4}$$

where $p_{s,t}$ is the observed proxy in the sample and $\nu_{s,t}$ is a measurement error. Whether this measure correctly captures the average health improvement of the workforce depends on the demographic structure. For example, in the case of the cardiovascular revolution, health gains concentrated among older adults with gains in life expectancy increasing with age. If, without loss of generality, the number of young workers is small, the average worker is older than 30. Therefore, the measure assigns a too pessimistic figure of the health improvement to the workforce for all observational units s with the extent of the error depending on state-level variation in the demographic structure of the workforce. Hence, the health proxy $p_{s,t}$ and the measurement error $\nu_{s,t}$ correlate negatively. Plugging the expression from (4) into the regression model yields

$$y_{s,t} = \alpha p_{s,t} + w_s' \mathcal{I}_t^{1960} \beta + \gamma_s + \delta_t + \zeta_{r,t} + \varepsilon_{s,t} - \alpha \nu_{s,t}. \tag{5}$$

Define $e_{s,t} = \varepsilon_{s,t} - \alpha \nu_{s,t}$ as the composite error term and suppose that there is no correlation between the proxy $p_{s,t}$ and the idiosyncratic error $\varepsilon_{s,t}$. Given a non-negative α and the negative correlation between $p_{s,t}$ and $\nu_{s,t}$ due to systematic error, the health proxy $p_{s,t}$ must correlate positively with the composite error. Hence, the point estimate $\widehat{\alpha}$ for this model will be biased upward. The same logic applies to a too optimistic measure of average health with the only difference being that $p_{s,t}$ and $\nu_{s,t}$ correlate positively in this case. Accordingly, systematic mismeasurement of the health status leads to downward-biased estimates of the population parameter if the health proxy overstates gains in average health, while estimates are biased upward if the health proxy understates the improvement in average health. The more proxy and true health diverge, the more severe this bias will be.

For the age-group analysis, this problem can be solved by using age-specific life expectancy as right-hand variable. Panel (a) of Table 4 reports the corresponding results for the differences-in-differences model.[23] Columns (1) to (5) show results for age cohorts from 15 to 64 in ten-year intervals. Column (6) reports the estimated effect of adult life expectancy on wages for old-age workers above the age of 65. Finally, Column (7) provides the parameter estimates for the entire workforce.[24] To this end, life expectancy for the total workforce is approximated by the arithmetic mean over life expectancy at birth and all following age cohorts, thus providing a

[23] Table A5 in the Appendix presents estimates for the flexible model.

[24] Regressions are weighted by the initial white population of each specific age cohort in 1960 in the first six columns and by the entire white population for the last column.

Table 4: Adult Life Expectancy and Average Wages by Age Cohorts

	Differences-in-differences model (2SLS)						
	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Life expectancy of specific age group						
Log life expectancy	3.66	4.18***	2.82***	2.54***	1.09	5.21***	3.62***
(of specific age group)	(3.10)	(1.61)	(0.88)	(0.95)	(0.95)	(1.98)	(1.09)
First stage F–stat	10.8	22.5	46.9	14.2	26.0	16.0	31.7
	(b) Naive model: life expectancy at age 30						
Log life expectancy at 30	1.19	4.18***	4.30***	4.46***	2.26	11.47***	3.67***
	(1.77)	(1.61)	(1.48)	(1.53)	(1.66)	(3.73)	(1.31)
First stage F–stat	24.0	22.5	21.4	21.9	22.5	24.3	23.3
	(c) Naive model: life expectancy at birth						
Log life expectancy at birth	0.72	3.79**	3.46**	4.15**	3.15	15.26**	4.00**
	(1.78)	(1.73)	(1.64)	(1.77)	(2.06)	(6.10)	(1.56)
First stage F–stat	11.6	11.4	10.4	10.0	9.6	9.3	11.0
States	48	48	48	48	48	48	48
Observations	336	336	336	336	336	336	336
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Control variables are measured in 1960 and interacted with the Post-1960-treatment dummy. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

health indicator closer to the average person of the workforce.[25]

The effect of life expectancy is positive and significant at the five percent level for the prime-age groups of the working-age population from 25 to 54 and old-age workers above 65. A one percent increase of life expectancy, ceteris paribus, induces a wage hike of roughly 3.5 percent with coefficients ranging from 2.54 to 5.21. The values of the Kleibergen-Paap F-statistic show a strong first-stage correlation between adult life expectancy and mortality from cardiovascular diseases. For very young workers, the effect of life expectancy on wages is large and positive but insignificant due to high standard errors. A potential explanation for this finding is that positive effects of health improvements are counteracted by an out-selection of the most productive workers into college. Moreover, the explanatory power of the instrument is weakest among all age groups with a value of the F-statistic of 10.8. Likewise, for the group of the 55 to 64 year olds, health improvements do not significantly affect wages. The effect, however, is considerably stronger for those workers who decide to work even after age 65. One reason for this finding might be selection into retirement starting around age 60. In order to save enough for retirement,

[25]Similar results obtain for life expectancy at age 30 or 40 as health measure for the total workforce.

workers who optimally would like to retire early due to bad health or outdated human capital continue to work, thus lowering productivity for the 55 to 64 year olds. Above age 65, only the healthy, motivated, and productive workers remain in the workforce: The overall labor force participation rate drops to slightly below 20 percent for this group. These workers are also likely those who gain most from improvements in health innovations. Hence, life expectancy shows the largest effect on wages with a coefficient of 5.21. Taken together, this evidence indicates a sizable positive effect of adult life expectancy on wages for workers in their prime-age and those above 65. Graphically, this translates into a shift toward steeper life-cycle wage profiles consistent with the unconditional evidence presented in Figure 1.

Panel (b) reports estimation results for a naive version of the differences-in-differences model, where life expectancy at age 30 is used for every age cohort. As outlined above, using a mismeasured proxy for health conditions leads to downward-biased estimates for age groups whose health gain is overstated by the measure, i.e., the 15 to 24 year olds, and upward-biased estimates for age groups whose health gains are understated, i.e., all groups above age 35. Correspondingly, the resulting point estimates would suggest a too large effect of life expectancy on average wages for older workers. For the 25 to 34 year olds, the model is identical to Panel (a). Panel (c) repeats this exercise for life expectancy at birth, which is the most common health indicator in cross-country studies. For the age groups above 35, where changes in life expectancy at birth understate the actual change, the point estimates are overestimated. In contrast, for the age group 25 to 34, the results show slightly smaller effects of health innovations on average wages compared to the results in Panel (a), although gains in life expectancy are quantitatively similar for this age group. Since the first stage of life expectancy at birth is considerably weaker compared to Panel (a), this finding might be due to a loss of precision in the estimation. Compared to the more adequate specifications in Panel (a), the naive model with life expectancy at birth suggests a too large effect of health gains on average wages, because the health gains are relatively larger at higher ages.

Importantly, the instrumentation strategy cannot eliminate this measurement error, because the correlation between adult life expectancy and mortality depends on age. If measured at higher ages, gains in life expectancy do not contain health improvements resulting from reduced mortality at younger ages. In contrast, if life expectancy is measured at a too young age, the instrument assigns individuals beneficial effects from health innovations that do not apply to them due to their age. Therefore, the first stage again systematically over- or understates the average health improvement, if the wrong age-specific proxy for health conditions is assigned.

This subtle point is of significant practical importance for a large number of published work. For example, a branch of the growth literature has investigated the effect of health and life expectancy on long-run growth of output per capita. The conventional measure employed in these studies is life expectancy at birth. Long-run changes in output are, thus, explained by improvements of health conditions of infants which might overstate the improvements for the workforce. In such a case, the corresponding point estimates would be biased downward. How severe the bias from measurement error is, depends on how well changes in life expectancy at birth capture changes in the health status of the workforce. For example, if improvements in medical technology mostly help infants and young children, as it was the case for the epidemiological

transition, the gains in life expectancy at birth and for higher ages will differ greatly. In this case, the bias is most pronounced. This might explain why Acemoglu and Johnson (2007), Acemoglu and Johnson (2014), Bloom, Canning, and Fink (2014), and Hansen (2014) have not found a substantial positive effect of health and life expectancy on growth for the reduction in mortality from infectious diseases. If, in contrast, increases in life expectancy at birth mostly reflect improvements in health at older ages, as it is case for the cardiovascular revolution, the bias should be comparatively small, because changes in life expectancy at birth provide still a reasonable approximation of improvements in adult health.

4.5 Accounting for Inter-State Migration

The analysis so far has investigated the causal relationship between adult life expectancy and average wages, while treating states as closed entities. A potential concern relates to workforce migration between states. About 1.5 percent of the total U.S. population move between states per year and one third of the citizens do not live in the state, where they were born (Molloy, Smith, and Wozniak, 2011). If, on average, high productivity workers migrate into states where life expectancy is higher, parameter estimates might be biased upward. In order to address this problem, individuals in the census data are dropped, if they do not live in the state, where they were born.[26] Panel (a) of Table 5 presents the respective results of the differences-in-differences model for state-level regression.[27]

The corresponding point estimates indicate a strong positive effect of increased life expectancy on average wages, which is statistically significant at the five percent level, except for the age group of the 15 to 24 year olds. For all age groups above 25, the resulting coefficients are quantitatively larger than in the baseline model in Table 4. The evidence from the non-migrant sample, therefore, conflicts with an upward bias due to migrant workers, unless indirect effects owing to the complementarity between domestic and migrating workers distinctly outweigh the direct effects. Hence, it is unlikely that the considerable positive effect of adult life expectancy on average wages in the baseline model is driven solely by workforce migration, and it appears conservative given the evidence presented in Table 5. Moreover, this evidence tends to the concern whether health should be measured by state of birth or state of residence: The results establish a causal link between adult life expectancy and average wages in the non-migrant sample, for which state of birth and state of residence coincide.

Furthermore, a frequent phenomenon is migration after retirement. While in the 1950s, fewer than one million people above age 60 moved from one state to another, the corresponding number had increased to 1.6 million between 1975 and 1980; whereupon, Florida, California, and Arizona were the most popular destination targets (Rogers and Watkins, 1987). High values of life expectancy at higher ages in these states reflect this popularity. If it is predominantly rich pensioners, who move for retirement, old-age migration might act as a positive demand shock to the destination states. If, at the same time, these pensioners are healthier than the average retiree, migration of the elderly might bias upward the estimated effect of health gains on average wages. Panel (b) of Table 5 presents results from the state panel without Florida, California,

[26] Note, however, that the effect of migrants cannot be deducted from the life expectancy measure.
[27] Estimates for the flexible model are reported in Table A6 in the Appendix.

Table 5: Robustness: Migration

	Differences-in-differences model (2SLS)						
	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) No inter-state migration of workers						
Log life expectancy	2.21	5.02***	4.65***	3.91***	2.56**	6.61**	4.78***
(of specific age group)	(2.76)	(1.51)	(1.05)	(1.24)	(1.01)	(2.73)	(1.18)
First stage F–stat	13.4	22.6	37.4	13.3	32.9	15.5	31.0
States	48	48	48	48	48	48	48
Observations	336	336	336	336	336	336	336
Full controls	✓	✓	✓	✓	✓	✓	✓
	(b) No old-age migration						
Log life expectancy	8.22	5.01**	3.11***	2.43**	1.07	5.21***	3.67***
(of specific age group)	(7.48)	(2.40)	(1.12)	(1.01)	(0.83)	(1.94)	(1.19)
First stage F–stat	2.7	11.7	33.7	12.4	43.8	18.6	24.2
States	45	45	45	45	45	45	45
Observations	315	315	315	315	315	315	315
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Average wages in Panel (a) are confined to workers who work in the same state they were born in. Panel (b) excludes Arizona, California, and Florida from the sample. Control variables are measured in 1960 and interacted with the Post-1960-treatment dummy. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

and Arizona. The parameter estimates are quantitatively similar to the baseline results, thus revealing again a positive link between adult life expectancy and wages per worker.[28] Overall, the evidence indicates that old-age migration does not cause a spurious correlation between life expectancy and wages.

4.6 Heterogeneity along Further Dimensions: Metropolitan Areas, Occupational Choice, and Educational Attainment

The analysis so far has investigated the causal relationship between adult life expectancy and average wages on the state level. As noted by Cooper et al. (2000), however, there might be disparities in the prevalence of cardiovascular diseases, in particular, between rural and metropolitan areas that might not be fully taken up by controlling for population density. Therefore, this section analyzes the causal link between life expectancy and wages only for metropolitan areas in the corresponding states. To this end, census data are collapsed on the metropolitan-area level, and each area is assigned the corresponding state-level value of life

[28] Results for the flexible model are reported in Table A7 in the Appendix.

Table 6: Adult Life Expectancy and Average Wages: Metropolitan Areas

	Differences-in-differences model (2SLS)						
	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Log life expectancy	9.91**	6.23***	4.09***	5.40***	3.05**	6.89*	6.31***
(of specific age group)	(3.88)	(1.78)	(1.11)	(1.86)	(1.32)	(3.53)	(1.45)
First stage F–stat	12.2	37.3	65.1	12.6	25.1	6.3	44.2
States	33	33	33	33	33	33	33
Metropolitan Areas	89	89	89	89	89	89	89
Observations	623	623	623	623	623	623	623
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: All regressions include metropolitan-area-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Control variables are measured in 1960 and interacted with the Post-1960-treatment dummy. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

expectancy.[29] Since metropolitan areas changed over time, only those areas that are consistently defined throughout all time periods from 1940 to 2000 enter the estimation sample. This leaves 623 time-year observations for 89 metropolitan areas in 33 states. Table 6 reports results for age groups and the workforce for the differences-in-differences model including a full set of controls.[30]

Throughout all age groups, the estimated coefficients are larger compared to state-level estimates. According to the workforce estimate in Column (7), a one-percent gain in life expectancy leads to a 4.17-percent increase in average wages per worker. All coefficient estimates are statistically significant at the five percent level, and the F-statistic indicates a strong first-stage correlation between life expectancy and mortality from cardiovascular diseases. This finding is in line with a rural-urban gap in health improvements related to cardiovascular diseases as, for example, found by Kulshreshtha et al. (2014). One explanation for this divide is that behavioral risk factors such as smoking, drinking, obesity, and physical inactivity are more common in rural areas (CDC, 2017). Another potential reason is that access to treatment for cardiovascular diseases is more readily available in urban areas due to returns to scale. Overall, this evidence suggests that the positive effect of adult life expectancy on average wages is not an artifact of comparing rural with urban states. Moreover, the gains from health innovations are larger in metropolitan areas compared to the state-level estimates.

Occupational choice constitutes another dimension of heterogeneity. Blue-collar workers, on the one hand, execute tasks that are physically demanding, whereas white-collar jobs, on the other hand, require minimal physical labor but usually more investment in educational attainment. With increasing age, blue-collar workers are, thus, more vulnerable to negative income shocks as a consequence of worsened health status compared to white-collar workers. Hence, innovations

[29] The motivation for using state-level health measures in this context is twofold: First, data on adult life expectancy are not available below the state level. Second, more disaggregated measures for age-specific life expectancy and disease-specific mortality rates may suffer from low quality, as relatively rare chance events may generate spurious patterns in small populations.

[30] Table A8 in the Appendix shows results for the flexible model.

Table 7: Heterogeneity: White-Collar and Blue-Collar Workers

	Differences-in-differences model (2SLS)						
	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) White-collar workers						
Log life expectancy	3.83	8.99***	4.08***	1.36	0.05	5.83**	4.51***
(of specific age group)	(6.76)	(3.27)	(1.58)	(1.29)	(0.95)	(2.85)	(1.68)
First stage F–stat	2.8	9.8	28.5	8.4	14.6	14.2	18.1
	(b) Blue-collar workers						
Log life expectancy	8.10	3.45	3.17*	4.07*	3.62**	0.35	4.15**
(of specific age group)	(6.36)	(2.85)	(1.64)	(2.17)	(1.67)	(2.60)	(1.99)
First stage F–stat	4.5	9.3	24.4	5.7	13.0	11.4	16.8
States	48	48	48	48	48	48	48
Observations	288	288	288	288	288	288	288
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Average wages contain observations from white-collar workers in Panel (a) and from blue-collar workers in Panel (b). Control variables are measured in 1960 and interacted with the Post-1960-treatment dummy. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

in medical technology should benefit blue-collar workers especially at advanced working age. In contrast, higher prospective health encourages potential white-collar workers to invest more in human capital, thus resulting in higher productivity particularly among younger age cohorts.

In order to test these hypotheses, workers in the decennial U.S. census are categorized as blue-collar or white-collar, based on the occupation coding guidelines for the 1970 U.S. census (U.S. Bureau of the Census, 1972, pp. 152–154). Specifically, this coding classifies workers as white-collar, if they belong to the group of professional, technical and kindred workers; managers and administrators except farm; sales workers; or clerical and kindred workers. In contrast, blue-collar occupations comprise 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. Since occupational status was first reported in the U.S. census in 1950, the number of state-year observations decreases to 288.

Table 7 presents results for a regression of average wages per worker on adult life expectancy for a subsample consisting of white-collar workers in Panel (a) and blue-collar workers in Panel (b).[31] Adult life expectancy affects average wages of white-collar workers in the age group 25 to 44 positively and significantly, whereas the effect vanishes for the more advanced workers in the age range from 45 to 64. This finding points to better educated young white-collar workers.[32] Conversely, wages of older white-collar workers did not increase significantly following

[31] Table A9 in the Appendix reports results for the flexible model.

[32] The effect is slightly more pronounced if adult life expectancy is lagged. Results are available upon request.

Table 8: Heterogeneity: College and Non-College Workers

	Differences-in-differences model (2SLS)						
	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Workers with some college education						
Log life expectancy	-0.34	3.08	2.27**	1.50	0.41	13.67**	2.22**
(of specific age group)	(4.41)	(2.01)	(1.13)	(1.03)	(1.40)	(5.68)	(1.10)
First stage F–stat	8.5	20.9	45.5	16.6	26.5	16.9	29.1
	(b) Workers without college education						
Log life expectancy	2.14	1.69	0.91	1.53*	0.82	2.66	1.35
(of specific age group)	(3.07)	(1.35)	(0.85)	(0.88)	(1.06)	(1.76)	(0.94)
First stage F–stat	11.1	22.8	47.1	13.8	26.1	16.1	31.7
States	48	48	48	48	48	48	48
Observations	336	336	336	336	336	336	336
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Average wages contain observations from workers with at least some college education in Panel (a) and from workers without any college education in Panel (b). Control variables are measured in 1960 and interacted with the Post-1960-treatment dummy. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

the cardiovascular revolution. Only for the selected group above age 65, life expectancy and average wages are again positively and significantly associated. This result is consistent with reduced sorting-out of productive workers due to improved health.

The coefficient estimates for the blue-collar workers in Panel (b) show a mirror image of the results for white-collar workers. Adult life expectancy and average wages show no statistically significant correlation for the young age groups and old-age workers above 65. Health gains, however, caused a significant wage rise for more experienced workers in the age range from 35 to 64. Hence, this finding supports the hypothesis that especially blue-collar workers were to benefit from improved health conditions due to the demanding physical activities they execute.

Finally, educational attainment represents another potential dimension of heterogeneity. Typically, wages of college-educated workers grow faster with every additional year of work experience compared to workers without any college education (see, for example, Ashenfelter and Rouse, 1999). If, for example, health gains at higher ages prolong the work life, wage gains would, ceteris paribus, be larger for college workers as a result of their relatively more favorable earnings trajectory. At the same time, however, wages might show little growth for young college-educated workers since individuals must invest additional time on acquiring college education. Lastly, gains in adult life expectancy likely raise the share of college enrollment in the population. If, at the margin, individuals, who would have otherwise not chosen this option, select into college because of higher prospective health, average wages for college workers remain unchanged or even decline. Hence, the extent of heterogeneity regarding educational attainment is a priori unclear.

In order to test whether adult life expectancy affects average wages differently along educational attainment, the sample is split into workers with at least some college education and those with at most a high-school degree. Individuals who did not report on their educational attainment are excluded from the sample. Table 8 reports parameter estimates for college workers in Panel (a) and non-college workers in Panel (b).[33] Overall, the results reveal no statistically significant association between adult life expectancy and average wages within educational groups. Therefore, health improvements do not alter the within-educational-group earnings trajectories. This finding does, however, not preclude wage increases as a consequence of structural shifts toward a more highly educated workforce. The next section highlights this point more closely in the context of potential mechanisms that explain the wage hikes observed in the baseline sample.

4.7 Channels

What are the channels through which innovations in understanding and treatment of cardiovascular diseases affect average wages? This section discusses four potential channels that may explain how the treatment translates into higher wages: labor supply, educational attainment, behavioral changes and improved health.

First of all, consider the possibility that wage hikes may result from changes in labor supply. At the intensive margin, individuals, who know about their improved health prospects, might decide to work more hours per week or more weeks per year and, thus, earn higher wages. Alternatively, at the extensive margin, workers might feel healthier particularly at higher ages and, thus, decide to remain in the workforce. Since workers typically earn higher wages with increasing experience and age, increased labor force participation at advanced ages might keep productive workers in the workforce and, thus, push average wages up. Table 9 reports the estimated semi-elasticities for the labor force participation rate (measured as 0 to 100 percent) in Panel (a), usual hours worked per week in Panel (b), usual weeks worked per year in Panel (c), and usual hours worked per year (derived from individual hours and weeks) in Panel (d).[34]

Strikingly, labor supply increased neither at the extensive nor at the intensive margin. In fact, the estimates in Panel (a) show that labor force participation rates decreased by roughly 1 to 1.5 percentage points for most age groups and for the total workforce. Moreover, the decrease was strongest among the 25 to 34 year olds with no significant effect for the 45 to 54 year olds. Hence, higher wages for prime-age workers cannot be the result of increased labor force participation. Furthermore, the evidence also precludes the possibility that cohort wages increased unilaterally as a consequence of lower labor supply. If wages and labor force participation were negatively correlated, one should observe an increase of wages for the 55 to 64 but not the 45 to 54 year olds; however, this is not the case as shown in Table 4. The specifications in Panels (b) to (d) reveal moderate negative effects of higher life expectancy on usual hours or weeks worked. For the entire population, a one percent increase in life expectancy leads to a decline of usual working hours per week by 0.16, or 10 minutes per week. Likewise, usual working weeks shrunk by 0.17, or roughly one workday per year. Neither effect is statistically significant at the conventional significance levels. Again, there is no clear pattern, which explains wage hikes as a consequence

[33] Table A10 in the Appendix presents estimates for the flexible model.
[34] Tables A11 and A12 in the Appendix present estimates for the flexible model.

Table 9: Adult Life Expectancy and Labor Supply by Age Cohorts

	Differences-in-differences model (2SLS)						
	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Labor force participation (0 to 100 percent)						
Log life expectancy (of specific age group)	-86.15 (95.18)	-223.01*** (61.93)	-92.35*** (30.32)	-21.20 (30.71)	-116.37** (48.82)	-140.11** (59.82)	-149.56*** (36.88)
	(b) Usual hours per week						
Log life expectancy (of specific age group)	-129.42** (54.81)	-64.50*** (24.25)	-28.72* (15.24)	-1.77 (13.08)	-35.32* (19.86)	-38.76* (20.92)	-15.95 (14.57)
	(c) Usual weeks per year						
Log life expectancy (of specific age group)	-91.90* (54.38)	-86.82*** (31.75)	-33.48** (16.58)	0.08 (14.45)	-43.91* (22.95)	-55.42** (25.55)	-17.21 (15.58)
	(d) Labor supply of those working (weeks × hours)						
Log life expectancy (of specific age group)	-3138.79 (2183.64)	-2584.40* (1322.99)	-1147.17 (801.69)	350.41 (655.21)	-1780.77* (986.28)	-1741.36* (896.19)	-387.51 (736.01)
First stage F–stat	10.8	22.5	46.9	14.2	26.0	16.0	31.7
States	48	48	48	48	48	48	48
Observations	336	336	336	336	336	336	336
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: The dependent variable is the labor force participation in Panel (a), usual hours worked per week in Panel (b), usual weeks worked per year in Panel (c), and hours worked per year of those working in Panel (d). All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Control variables are measured in 1960 and interacted with the Post-1960-treatment dummy. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

of reduced labor supply. Therefore, adjustments along the intensive margin of workers' labor provision cannot explain higher wages either. Finally, the combination of raised wages and stable or reduced labor supply implies an increase of productivity, as measured by wages per workers, per working hours, or per working weeks. In sum, labor supply cannot account for the observed increase in average wages.

Education provides an alternative channel through which gains in adult life expectancy might affect average wages per worker. Between 1960 and 2000, the share of U.S. whites who enrolled into college at least once roughly doubled from 15 to 30 percent. At the same time, the share of graduates with at least four years of college education tripled from 5 to 15 percent. Based on a prototype Ben-Porath model of human capital and life-cycle earnings, Cervellati and Sunde (2013) show that an increase in survival rates during working ages may raise the benefits of education relative to its costs. Health gains that take place at sufficiently young ages may, consequently, increase individuals' educational investment. Hansen and Strulik (2017) find that college enrollment increases by roughly ten percentage points as a consequence of

higher life expectancy following the cardiovascular revolution in U.S. states. Reproducing their specification with college enrollment of the 15 to 24 year olds as dependent variable in the empirical framework presented in this paper yields a quantitatively similar estimate of roughly nine percentage points.[35] Therefore, educational attainment is one possible channel through which adult life expectancy affects average wages. Human capital is, however, tied to the person, who acquired it. Consequently, upskilling of older worker groups takes time. For example, if individuals around age 20 enroll in college due to the treatment in 1970, the direct benefits of education for wages of 45 to 54 year olds will only take full effect after 20 to 30 years, when these individuals enter this age group. Hence, education may play a key role in explaining wage hikes, though, only after sufficient time has elapsed.[36] This timing structure will help in confining direct health from gains through educational and behavioral changes.

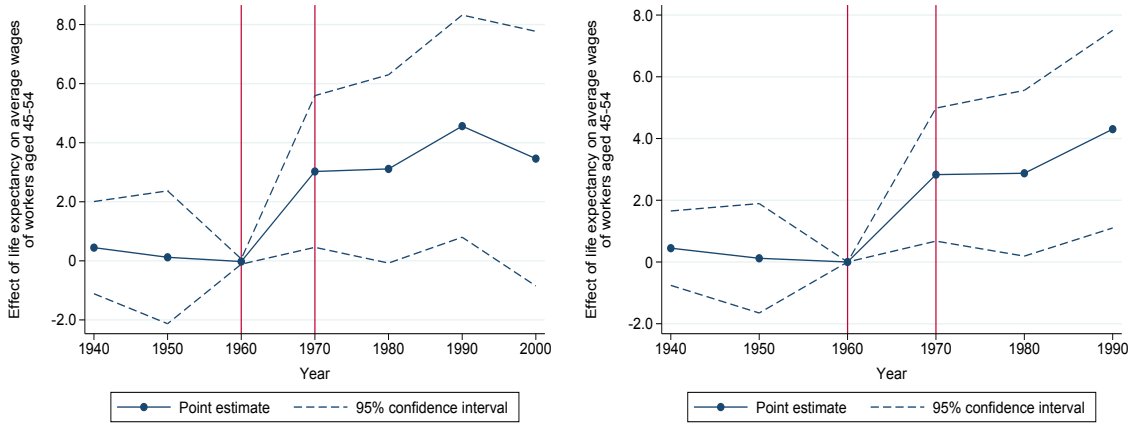
In combination, the results for education and labor supply indicate that increased life-time labor supply is not a necessary condition for higher educational attainment, as was claimed by Hazan (2009). In contrast, the evidence confirms simulation results of Cervellati and Sunde (2013) and Strulik and Werner (2016). They argue that higher educational attainment and lower life-time labor supply are compatible, if the income effect of higher life expectancy is large enough to afford both, increased life-time consumption and leisure time while at work.

Changes in individual behavior constitute another potential channel through which health may affect wages. For example, preventive measures against smoking following the report of the Surgeon General in 1964 (U.S. Department of Health, Education, and Welfare, 1964) have reduced smoking among U.S. adults considerably after 1970, as exemplified by Figure 7. Due to the cumulative damage of smoking, however, cessation requires up to ten years to take the full beneficial health effects (Oza et al., 2011). Consequently, health improvements from reduced smoking should show the full positive effect on wages per worker only starting from the 1980s. Behavioral changes that have more immediate positive effects are increased physical activity, reduced alcohol consumption, and a more healthy diet. Physical activity, however, has gradually declined between 1970 and 2010, while the share of obese persons doubled (Flegal et al., 1998; CDC, 2001; Kohl and Cook, 2013). At the same time, the consumption of alcohol started to decline only after 1980, as shown in Figure 8. Hence, behavioral changes due to a more healthy lifestyle do not cause immediate improvements in health conditions among U.S. adults. Nevertheless, there should be positive long-run effects from reduced drinking and smoking.

Taken together, the presented evidence suggests that labor supply cannot explain the observed wage hikes. Meanwhile, higher college enrollment and more healthy behavior only unfold a positive effect on productivity starting in the 1980s. By eliminating these channels, short-run

[35] Table A13 presents the effect of a health shock on college enrollment for different measures of adult life expectancy. Following Hansen and Strulik (2017), the estimation equation is weighted by the population at risk, i.e., the initial population of 15 to 24 year olds. To arrive at the numbers, compute $\Delta_y = \frac{\widehat{\alpha}}{100} \cdot \Delta_x = \frac{0.77}{100} \cdot 12.20 \approx 0.09$ with $\Delta_x = \frac{3.16}{25.91} \cdot 100 \approx 12.20$ and $\widehat{\pi} \cdot \Delta_z \cdot \bar{\mu}_x = (-0.61) \cdot (-0.20) \cdot 25.91 \approx 3.16$ computed from the first stage estimates in Table 1, where $\bar{\mu}_x$ is evaluated at the mean in 1960.

[36] Training constitutes another dimension of educational attainment, which might raise worker productivity and wages more immediately, because it is mostly directed at prime-age workers (Carnevale, Strohl, and Gulish, 2015). Public expenditures on training, however, are quantitatively small compared to spending on tertiary education and cover less than one-tenth of a percent of U.S. GDP in the year 2000 (OECD, 2018a; OECD, 2018b). Private expenditures on formal training appear quantitatively more sizable with two to three per mill of U.S. GDP between 2010 and 2015, though, they are again minor in comparison to spending on tertiary education (Training Magazine, 2015; OECD, 2018a).



(a) 1940–2000 (Table 10, Column 1)

(b) 1940–1990 (Table 10, Column 2)

Figure 11: Illustration: Effect of Life Expectancy Over Time (45–54 Year Olds)

effects of the treatment, thus, likely reflect health improvements. For example, gains in individual health status arise through new drugs and treatment possibilities such as the beta blocker or the cardiac pacemaker, which allow patients to continue to work only with minor restrictions.

In order to understand how the cardiovascular revolution affected wages over time consider the following fully-flexible model

$$y_{s,t} = \sum_{\tau=1940}^{2000} \alpha_\tau x_{s,t} d_t^\tau + w_s' \mathcal{I}_t^\tau \beta + \gamma_s + \delta_t + \zeta_{r,t} + \varepsilon_{s,t}, \tag{6}$$

where log life expectancy is interacted with a full set of time dummies. This model allows to estimate the effect of life expectancy on wages for all six time periods relative to the reference year 1960. Using the flexible first stage from equation (3), the model is just-identified. The estimated coefficients for the 45 to 54 year olds are reported in Table 10 and plotted in Figure 11 for the time periods 1940–2000 and 1940–1990. Due to the increased number of instruments, the value of the F-statistic falls to a level of below one for the model from 1940–2000. If observations in the year 2000 are dropped, the model is somewhat better identified with an F-statistic of approximately 7.6. The results are qualitatively consistent with moving-window and long-differences models, which add one additional year at a time. Therefore, it seems reasonable to provide a qualitative interpretation of the patterns shown in Figure 11.[37]

Panel (a) shows the results for the time period 1940–2000. In the pre-treatment periods, the effect of life expectancy is statistically insignificant and very close to zero. After the treatment in 1970, the estimated effect is positive, significant at the five percent level, and takes a value of 3.03. The estimated parameter does not change much between 1970 and 1980 but becomes slightly less precise. Between 1980 and 1990, the effect increases by approximately 50 percent to 4.56 before it slightly declines thereafter. The model for the period 1940–1990 in Panel (b), which shows a higher value of the F-statistic, confirms these patterns. According to the channels outlined in this section, the immediate increase in 1970 and 1980 is likely due to health improvements.

[37] Parameter estimates for the moving-window and long-differences models are reported in Tables A14 and A15 in the Appendix.

Table 10: Effect of Treatment Over Time

	45–54 Year Olds		Total Workforce	
	1940–2000	1940–1990	1940–2000	1940–1990
	(1)	(2)	(3)	(4)
Log life expectancy × 1940	0.45	0.45	0.21	0.21
	(0.80)	(0.61)	(1.81)	(1.41)
Log life expectancy × 1950	0.12	0.12	-0.20	-0.20
	(1.15)	(0.90)	(1.44)	(1.10)
Log life expectancy × 1970	3.03**	2.83***	5.26***	5.20**
	(1.31)	(1.10)	(1.97)	(2.22)
Log life expectancy × 1980	3.11*	2.87**	5.84**	5.76**
	(1.63)	(1.37)	(2.50)	(2.88)
Log life expectancy × 1990	4.56**	4.30***	7.73**	7.65**
	(1.92)	(1.63)	(3.32)	(3.68)
Log life expectancy × 2000	3.46	—	6.15**	—
	(2.20)	—	(2.39)	—
First stage F–statistic	0.6	7.6	2.2	2.0
States	48	48	48	48
Observations	336	288	336	288
Full controls	✓	✓	✓	✓

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of 45 to 54 year olds. Log life expectancy of the respective age group is interacted with time dummies. Initial log life expectancy is measured in 1960 and interacted with the Post-1960-treatment dummy. All other control variables are measured in 1960 and interacted with time dummies. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Finally, the gains from higher educational attainment and behavioral changes materialize in the data from 1990 onward, thus explaining an increase in the effect of life expectancy on wages of the 45 to 54 year olds.

Longitudinal data from the Health and Retirement Study provide another piece of evidence that health effects contributed to wage increases following the cardiovascular revolution. For approximately 22,000 respondents, the data set contains up to twelve observations from biannual interviews between 1992 and 2014. Table 11 shows results for the effect of individual health status on wages. Estimates are obtained from an OLS regression of log wage $y_{i,t}$ from individual i at interview wave t on a binary indicator for health status $h_{i,t}$; its interaction with a dummy, $h_{i,t}b_i^\tau$, which takes a value of one, if individual i has been born before a certain cutoff year τ; a quartic age trend $a_{i,t}^k$, $k \in \{1, 2, 3, 4\}$; and a set of fixed effects. Specifically,

$$y_{i,t} = \vartheta h_{i,t} + \rho h_{i,t} b_i^\tau + \phi_1 a_{i,t} + \phi_2 a_{i,t}^2 + \phi_3 a_{i,t}^3 + \phi_4 a_{i,t}^4 + \iota_i + \chi_t + \psi_{s,t} + \omega_{r,t} + \epsilon_{i,t} \tag{7}$$

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. In particular, the individual-fixed effect eliminates time-invariant heterogeneity in pivotal dimensions such as ability, educational attainment, and occupational choice. Moreover, state-fixed effects control for state-specific intercepts that pertain to individuals who migrate to another state. Finally, wave and census-region-wave effects address wage differentials that result from general wage trends over time.

Table 11: Effect of Individual Health on Wages

	Dependent variable: respondents' log wages					
	(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

Notes: All regressions include individual-fixed, state-fixed, wave and census-region-wave effects as well as 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 has been born before a certain threshold level, e.g., 1910, and zero else. Standard errors are clustered at the individual level. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

The parameter ϑ describes the direct effect of having ever been diagnosed with a negative health status such as 'having heart problems'; in this case $h_{i,t}$ takes a value of one. The respective parameter estimate corresponds to roughly 0.06 throughout all specifications except Column (5). Taken at face value, workers who have been diagnosed with heart problems, thus, earn on average six percent lower wages compared to workers without heart problems. The causal effect of adverse health effects, however, is likely even more negative. Since the analysis examines variation in wages at the intensive margin, it cannot capture particularly severe cases of heart problems, which end lethally or in disability. Accordingly, cases in which the negative health effect corresponds to 100 percent are, by construction, omitted from the regression.

Furthermore, the parameter ρ captures heterogeneity with respect to the health effect for different birth cohorts. Individuals who have been born before the year 1910 were already around age 60, when new drugs and treatment procedures for cardiovascular diseases became available. Hence, the cardiovascular revolution came too late for them to affect most of their work life, or to provide significant incentives for further investment in educational attainment. The corresponding estimate shows a large and significant negative effect for this group compared to younger cohorts; however, a word of caution is needed. Due to the small number of only 71 observations before 1910, the resulting coefficient may be plagued by both, small sample properties and unobserved selection of individuals within this age group. Even though the results from Column (2) conform with the hypothesis of higher productivity and wages due to improved adult health, they should be seen as suggestive and not conclusive. Columns (3) and (4) illustrate that once the cutoff year is shifted toward younger cohorts for which new drugs

and treatments for cardiovascular diseases were at least partly available, 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 wages for birth cohorts that are young enough to fully harness beneficial effects of the cardiovascular revolution. Concretely, the direct effect of suffering from heart problems becomes statistically insignificant, once the interaction term splits the sample into individuals, who could not or only partly profit from the treatment, and individuals of the reference group who could fully avail of it. Overall, this evidence suggests that health innovations have marginalized negative effects of cardiovascular diseases on individual productivity and income over time.[38]

4.8 Discussion

The preceding sections argued that productivity gains from improved health and higher educational attainment explain the observed wage hikes. Here, I discuss to what extent the evidence is consistent with general equilibrium effects and alternative wage theories, in particular: positive demand effects, agglomeration economics, efficiency wages, and compensating wage differentials.

First, newly available drugs and health services reduce the frequency of severe courses of disease and, thus, costs for patients and relatives. Therefore, individuals may reallocate income to commodities or additional, previously unaffordable health services, boosting overall demand and wages. Catlin and Cowan (2015) show that national health expenditures continuously increased over the period 1960–2000, and that annual growth rates of national health expenditures exceeded GDP growth rates in all but three years over this time period. As a result, the share of national health expenditures to GDP increased substantially. Hence, individuals devoted rather more than less resources to health services. This finding, thus, conflicts with a positive demand shock on commodity markets, but it conforms with a positive demand shock on the health sector.

Agglomeration economies and local multipliers constitute another potential source of prolonged income growth (see, for example, Moretti, 2010, and Kline and Moretti, 2013). Specifically, the demand for local goods and services increases with the equilibrium wage and the number of skilled workers in a city or economically-integrated area. The existence of such multiplier effects is consistent with the evidence presented above. Better health conditions and more training increase the number of skilled workers and raise average productivity and, therefore, wages per worker. Consequently, demand for local goods and services increases, thus providing further support for higher wages. Because skilled workers concentrate in metropolitan areas, agglomeration economies may explain heterogeneity in the size of health effects between rural and urban areas. Agglomeration economies, however, constitute a second-round effect that requires initial improvements in productivity or the distribution of skills within an area. Hence, they fail as the sole determinant of the observed positive effect of adult health on average wages.

The discussion so far implicitly assumed that workers be remunerated according to their marginal product on a competitive labor market. Efficiency wage theories depart from this

[38] Similar findings apply, if negative health status is measured by 'high blood pressure' as Table A16 in the Appendix shows. For 'stroke' as proxy of negative health status, the results show qualitatively similar though statistically insignificant effects. This finding, however, is not surprising insofar that the sample contains considerably fewer observations for stroke and that selection out of the labor market is particularly strong for this group.

assumption by allowing wages above the market rate, as this profits the firm (see, e.g., Katz 1986, Stiglitz, 1986, and Krueger and Summers, 1988). In the context of cardiovascular diseases, for example, firms might find it profitable to pay healthy workers above their marginal product in order to reduce turn-over costs from replacing ill workers. This argument, however, is contradicted by the finding of a larger beneficial effect of health innovations in states with a high initial prevalence of cardiovascular diseases. Improved health conditions and better treatment possibilities, in particular through new drugs, lower firms' incentives to pay wages above market clearing. Therefore, efficiency wage arguments cannot explain the observed wage increase.

Finally, the theory of compensating wage differentials suggests that jobs with less favorable job characteristics must be remunerated with higher wages as "[t]he whole of the advantages and disadvantages of the different employments of labour and stock must, in the same neighbourhood, be either perfectly equal or continually tending to equality. If in the same neighbourhood, there was any employment evidently either more or less advantageous than the rest, so many people would crowd into it in the one case, and so many would desert it in the other, that its advantages would soon return to the level of other employments" (Smith, 1776, Book 1, Chapter 10). According to this prediction, workers would, ceteris paribus, demand higher wages for jobs and states, which pose more disadvantages due to higher risk of cardiovascular diseases. This reasoning, however, conflicts with a larger beneficial effect of health innovations in states with high initial prevalence of cardiovascular diseases. Following health innovations, the compensating wage differentials should collapse, thus implying lower, not higher wage growth in states with high initial prevalence of cardiovascular diseases, as shown by the baseline results. Hence, compensating wage differentials cannot explain the observed raise in average wages.

5 Conclusion

This paper establishes a positive causal link between adult health and average wages per worker by exploiting the sharp decline in mortality from cardiovascular diseases in U.S. states after the 1960s. This drop in mortality, also known as the cardiovascular revolution, provides a well-suited source of quasi-experimental variation for several reasons. First, since cardiovascular diseases become more likely with increasing age, they predominantly affect adult health conditions and, thus, adult life expectancy. Second, the decline in mortality rates was initiated by a number of unexpected, path-breaking medical innovations during the 1960s. Lastly, treatment intensities vary across states due to heterogeneity in the prevalence of cardiovascular diseases due to social, cultural, and environmental reasons. Hence, this variation allows to estimate a differences-in-differences model where all states are treated but with varying treatment intensities. In order to account for endogeneity, adult life expectancy is instrumented by mortality from cardiovascular diseases prior and post the medical advancements in the 1960s.

The results suggest that the cardiovascular revolution was responsible for an increase of life expectancy at 50 of approximately 3.16 years, or roughly two thirds of the increase between 1960 and 2000. This rise in life expectancy can account for roughly 47 percent of the wage increase observed between 1960 and 2000 for workers aged 45 to 54. In particular, the results reveal that the gains concentrate on the prime-age workers between 25 and 54 as well as old-age workers above 65. Correspondingly, the life-cycle earnings profile for an average worker increases more

steeply at younger ages, whereas it flattens out more slowly at higher ages. Overall, this pattern is consistent with a workforce that over time becomes healthier at any given age, and at higher ages in particular.

The paper's main finding of a positive causal link between adult life expectancy and average wages also maintains for empirical models that exploit metropolitan-area variation in wages or account for interstate migratory patterns. Adjustments in labor supply cannot explain the estimated wage increase, because labor force participation rates, working hours, and working weeks either declined or remained unchanged during the treatment period. Moreover, age group estimates preclude the possibility of unilateral indirect wage effects through out-selection or increased bargaining power. Furthermore, the analysis reveals that there exists heterogeneity in the beneficial effects of health improvements on average wages between rural and metropolitan areas as well as different occupational groups. The timing of the wage hikes suggests that potential channels are health improvements, in particular in the short-run, and higher educational attainment and potential adoption of a more healthy individual lifestyle in the long-run. Evidence based on micro data further suggests that health innovations have marginalized negative effects of cardiovascular diseases on individual income over time. Overall, the evidence demonstrates that thanks to better adult health, workers earn more, work slightly less, and invest more in human capital.

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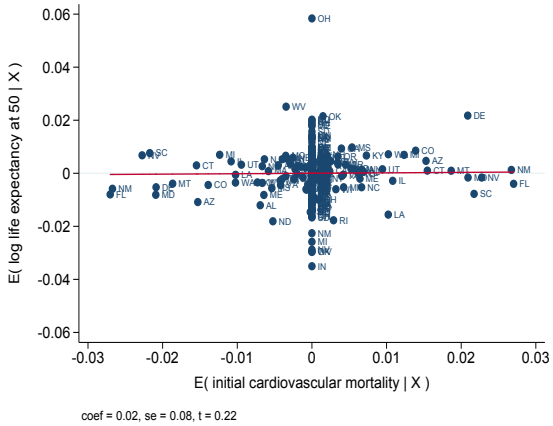
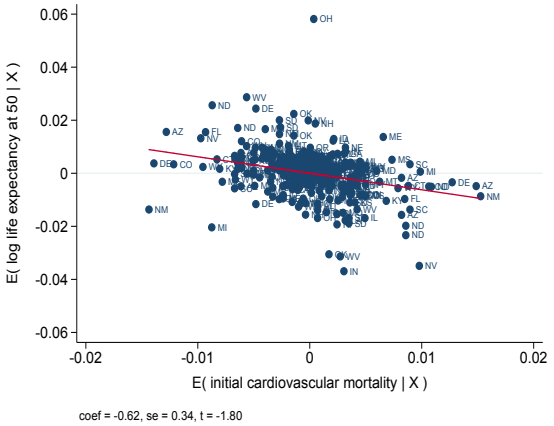
Appendix

Additional Figures

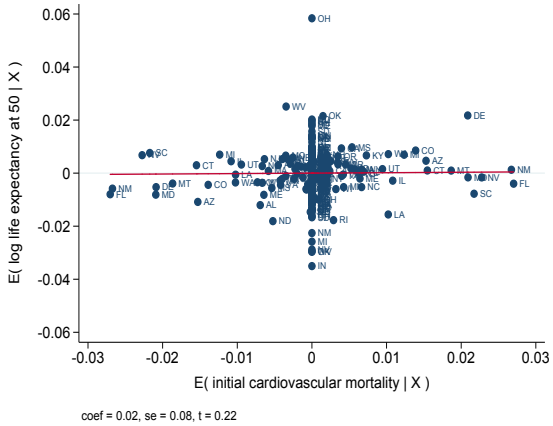


(a) Pre Treatment (Table 1b, Column 5)

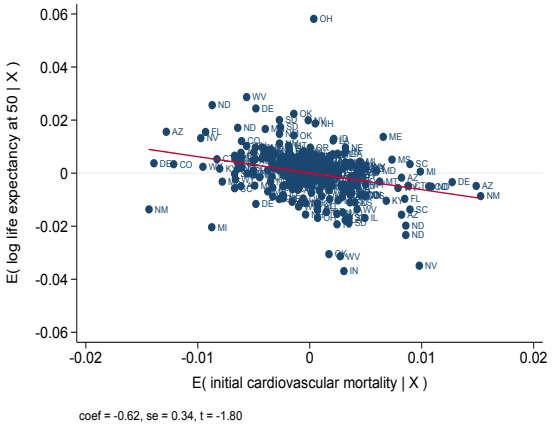


(b) Post Treatment (Table 1b, Column 5)

Figure A1: Partial Correlation Plots: First Stage (Flexible Model)

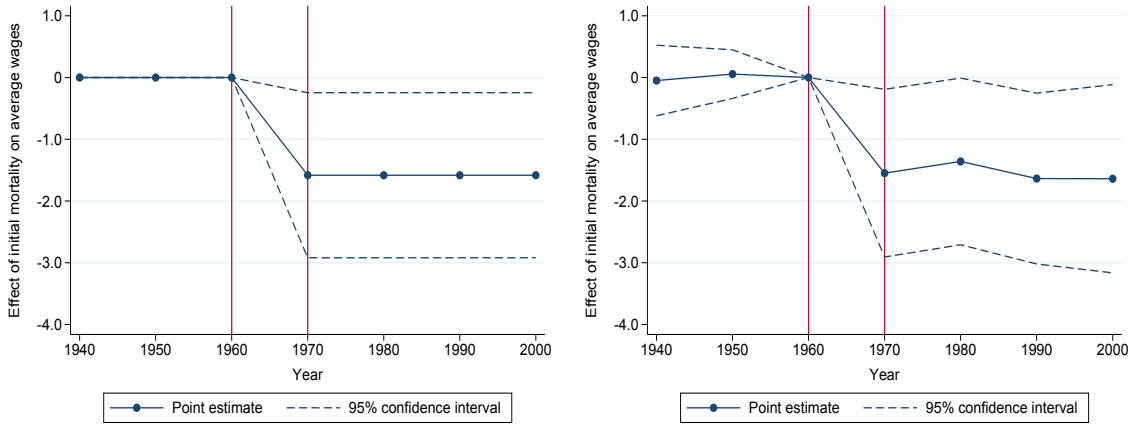


(a) Pre Treatment (Table 2b, Column 5)



(b) Post Treatment (Table 2b, Column 5)

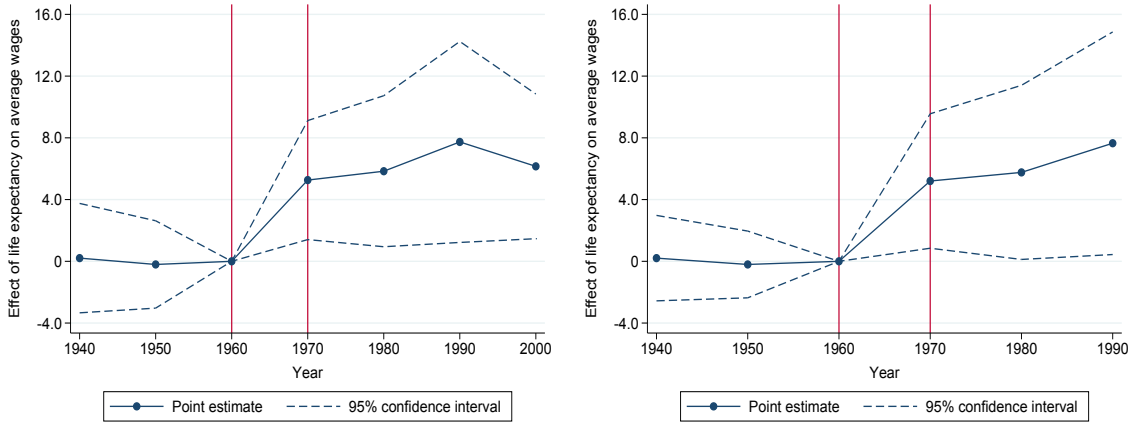
Figure A2: Partial Correlation Plots: Reduced Form (Flexible Model)



(a) DD Model (Table A3a, Column 5)

(b) Flexible Model (Table A3b, Column 5)

Figure A3: Robustness: Reduced Form Total Workforce



(a) 1940–2000 (Table 10, Column 3)

(b) 1940–1990 (Table 10, Column 4)

Figure A4: Illustration: Effect of Life Expectancy Over Time (Total Workforce)

Additional Tables

Table A1: Descriptive Statistics by Age Group

	Total	15–24	25–34	35–44	45–54	55–64	65+
Log wages	10.20	9.41	10.24	10.44	10.46	10.36	9.90
	(0.37)	(0.26)	(0.35)	(0.37)	(0.40)	(0.40)	(0.31)
Labor force participation	58.43	56.78	71.71	73.26	71.23	56.42	17.86
	(6.25)	(9.90)	(9.81)	(9.54)	(9.03)	(5.51)	(5.79)
Usual work hours per week	17.68	19.19	29.59	30.32	29.47	23.23	6.84
	(3.63)	(4.85)	(5.76)	(5.29)	(5.03)	(3.21)	(1.93)
Usual work weeks per year	18.78	19.99	31.27	31.99	31.50	25.46	8.03
	(5.97)	(6.29)	(10.18)	(10.44)	(9.76)	(6.93)	(2.43)
Average work hours per year	723.93	641.58	1250.97	1304.04	1283.79	993.46	254.08
	(254.28)	(240.45)	(445.50)	(452.71)	(424.22)	(282.46)	(87.69)
Log life expectancy	3.65	4.00	3.81	3.58	3.29	2.95	2.74
	(0.07)	(0.05)	(0.06)	(0.07)	(0.09)	(0.11)	(0.12)
Mortality from CVD in 1960	0.23	—	—	—	—	—	—
× Post 1960	(0.20)	—	—	—	—	—	—
Controls in 1960 × Post 1960:							
Initial log life expectancy at 50	2.07	2.27	2.16	2.03	1.86	1.66	1.53
	(1.80)	(1.97)	(1.88)	(1.76)	(1.61)	(1.44)	(1.33)
Initial mortality other than CVD	0.21	—	—	—	—	—	—
	(0.18)	—	—	—	—	—	—
Initial share college graduates	0.03	—	—	—	—	—	—
	(0.03)	—	—	—	—	—	—
Initial share college enrollment	0.07	—	—	—	—	—	—
	(0.06)	—	—	—	—	—	—
Initial population density ($\times\frac{1}{100}$)	0.75	—	—	—	—	—	—
	(1.60)	—	—	—	—	—	—
Initial log wages	5.80	—	—	—	—	—	—
	(5.03)	—	—	—	—	—	—
Sample weights:							
Initial white population ($\times\frac{1}{100000}$)	32.95	4.36	4.17	4.47	3.83	2.94	3.18
	(34.26)	(4.31)	(4.42)	(4.84)	(4.18)	(3.27)	(3.41)

Notes: Descriptive statistics for balanced panel of the 48 contiguous states from 1940–2000 with a total number of 336 observations. Numbers are means for the respective variable in the total population or a specific age group. Standard deviations are in parentheses. CVD is an abbreviation for cardiovascular diseases.

Table A2: OLS: Adult Life Expectancy and Average Wages of Workers Aged 45–54

	Dependent variable: log wages of whites 45–54				
	(1)	(2)	(3)	(4)	(5)
	Ordinary Least Squares				
Log life expectancy at 50	-0.43	-0.05	-0.04	0.23	-0.01
	(0.26)	(0.37)	(0.38)	(0.41)	(0.37)
Controls in 1960 × Post 1960:					
Initial life expectancy		✓	✓	✓	✓
Initial mortality (not CVD)			✓	✓	✓
Initial share college					✓
Initial population density					✓
Initial income					✓
Region-year FE				✓	✓
FE	✓	✓	✓	✓	✓
States	48	48	48	48	48
Observations	336	336	336	336	336

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of 45 to 54 year olds. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A3: Robustness: Reduced Form for Total Workforce

	Dependent variable: log wages of the total workforce				
	(1)	(2)	(3)	(4)	(5)
	(a) Differences-in-differences model				
Mortality × Post_1960	-0.62***	-0.65	-1.34**	-1.18**	-1.58**
	(0.19)	(0.43)	(0.61)	(0.56)	(0.66)
	(b) Flexible model				
Mortality × 1940	0.36	0.36	0.35	-0.33	-0.05
	(0.23)	(0.23)	(0.24)	(0.22)	(0.28)
Mortality × 1950	0.13	0.13	0.12	0.11	0.06
	(0.17)	(0.17)	(0.17)	(0.21)	(0.20)
Mortality × 1970	-0.05	-0.08	-0.75	-1.11*	-1.55**
	(0.17)	(0.41)	(0.58)	(0.56)	(0.68)
Mortality × 1980	-0.79***	-0.81	-1.49**	-0.77	-1.36**
	(0.27)	(0.50)	(0.64)	(0.54)	(0.67)
Mortality × 1990	-0.42	-0.44	-1.15	-1.49**	-1.64**
	(0.35)	(0.54)	(0.72)	(0.64)	(0.69)
Mortality × 2000	-0.57	-0.60	-1.34*	-1.67**	-1.64**
	(0.39)	(0.57)	(0.76)	(0.72)	(0.76)
Controls in 1960 × Post 1960:					
Initial life expectancy		✓	✓	✓	✓
Initial mortality (not CVD)			✓	✓	✓
Initial share college					✓
Initial population density					✓
Initial income					✓
Region-year FE				✓	✓
FE & TE	✓	✓	✓	✓	✓
States	48	48	48	48	48
Observations	336	336	336	336	336

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of 45 to 54 year olds. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A4: Robustness: Linear Specification of Life Expectancy

	Dependent variable: log wages of whites 45–54				
	(1)	(2)	(3)	(4)	(5)
	(a) Differences-in-differences model (2SLS)				
Life expectancy at 50	-0.08***	0.39	0.22**	0.10***	0.15**
	(0.02)	(0.29)	(0.10)	(0.03)	(0.06)
First stage F–statistic	46.8	2.1	6.8	35.5	9.4
	(b) Flexible model (2SLS)				
Life expectancy at 50	-0.05***	0.03	0.04*	0.09***	0.12*
	(0.02)	(0.02)	(0.02)	(0.03)	(0.06)
First stage F–stat	10.9	7.7	9.8	7.9	2.0
Hansen test p–value	0.04	0.002	0.004	0.2	0.8
	(c) Flexible model (LIML)				
Life expectancy at 50	-0.06***	0.12	0.10*	0.12***	0.15*
	(0.02)	(0.14)	(0.06)	(0.04)	(0.09)
First stage F–stat	10.9	7.7	9.8	7.9	2.0
Hansen test p–value	0.04	0.05	0.05	0.3	0.9
Controls in 1960 × Post 1960:					
Initial life expectancy		✓	✓	✓	✓
Initial mortality (not CVD)			✓	✓	✓
Initial share college					✓
Initial population density					✓
Initial income					✓
Region-year FE				✓	✓
FE & TE	✓	✓	✓	✓	✓
States	48	48	48	48	48
Observations	336	336	336	336	336

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of 45 to 54 year olds. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A5: Adult Life Expectancy and Average Wages by Age Cohorts

	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Flexible model (2SLS)						
Log life expectancy	1.00	3.61***	2.71***	2.35***	0.84	0.66	3.13***
(of specific age group)	(2.05)	(1.29)	(0.80)	(0.89)	(0.74)	(1.10)	(0.94)
First stage F–stat	3.4	5.3	8.5	3.5	5.9	2.4	5.8
Hansen test p–value	0.4	0.8	1.0	0.8	0.7	0.02	0.8
	(b) Flexible model (LIML)						
Log life expectancy	1.13	3.84***	2.75***	2.57**	0.90	1.48	3.30***
(of specific age group)	(2.52)	(1.40)	(0.81)	(1.00)	(0.78)	(2.80)	(1.00)
First stage F–stat	3.4	5.3	8.5	3.5	5.9	2.4	5.8
Hansen test p–value	0.4	0.8	1.0	0.8	0.7	0.02	0.8
States	48	48	48	48	48	48	48
Observations	336	336	336	336	336	336	336
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Control variables are measured in 1960 and interacted with a full set of time dummies with the year 1960 as reference category. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A6: Adult Life Expectancy and Average Wages: No Migration

	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Flexible model (2SLS)						
Log life expectancy	1.71	4.36***	4.83***	3.91***	1.85**	6.41**	4.88***
(of specific age group)	(1.91)	(1.19)	(0.97)	(1.20)	(0.93)	(2.84)	(1.08)
First stage F–stat	4.7	6.1	6.6	3.2	5.4	3.8	5.9
Hansen test p–value	0.01	0.008	0.009	0.02	0.006	0.1	0.002
	(b) Flexible model (LIML)						
Log life expectancy	3.67	6.24***	6.89***	5.51***	2.59**	7.44**	6.60***
(of specific age group)	(3.61)	(2.06)	(1.72)	(2.07)	(1.27)	7.44**	(1.65)
First stage F–stat	4.7	6.1	6.6	3.2	5.4	3.8	5.9
Hansen test p–value	0.02	0.03	0.03	0.07	0.009	0.1	0.008
States	48	48	48	48	48	48	48
Observations	336	336	336	336	336	336	336
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Control variables are measured in 1960 and interacted with a full set of time dummies with the year 1960 as reference category. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A7: Adult Life Expectancy and Average Wages: No Old-Age Migration

	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Flexible model (2SLS)						
Log life expectancy	0.25	3.93**	2.59***	2.22**	0.65	1.72	3.00***
(of specific age group)	(3.28)	(1.61)	(0.93)	(0.89)	(0.65)	(1.43)	(0.97)
First stage F–stat	1.4	3.2	6.3	3.9	7.8	2.7	4.7
Hansen test p–value	0.5	0.8	0.3	0.5	0.3	0.05	0.3
	(b) Flexible model (LIML)						
Log life expectancy	-0.97	4.42**	2.91***	2.63**	0.75	3.90	3.45***
(of specific age group)	(9.13)	(1.90)	(1.07)	(1.10)	(0.71)	(4.16)	(1.18)
First stage F–stat	1.4	3.2	6.3	3.9	7.8	2.7	4.7
Hansen test p–value	0.5	0.8	0.3	0.5	0.3	0.08	0.4
States	45	45	45	45	45	45	45
Observations	315	315	315	315	315	315	315
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Control variables are measured in 1960 and interacted with a full set of time dummies with the year 1960 as reference category. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A8: Adult Life Expectancy and Average Wages: Metropolitan Areas

	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Flexible model (2SLS)						
Log life expectancy	2.48	5.01***	3.73***	4.35***	2.40**	0.60	4.97***
(of specific age group)	(2.16)	(1.33)	(0.91)	(1.64)	(0.94)	(1.35)	(1.15)
First stage F–stat	6.2	12.8	15.3	4.4	7.5	3.3	11.8
Hansen test p–value	0.02	0.2	0.4	0.1	0.5	0.01	0.2
	(b) Flexible model (LIML)						
Log life expectancy	2.99	5.40***	3.82***	5.27**	2.47**	0.58	5.29***
(of specific age group)	(3.13)	(1.48)	(0.95)	(2.17)	(0.99)	(1.58)	(1.27)
First stage F–stat	6.2	12.8	15.3	4.4	7.5	3.3	11.8
Hansen test p–value	0.02	0.2	0.4	0.2	0.5	0.01	0.2
States	33	33	33	33	33	33	33
Metropolitan Areas	89	89	89	89	89	89	89
Observations	623	623	623	623	623	623	623
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: All regressions include metropolitan-area-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Control variables are measured in 1960 and interacted with a full set of time dummies with the year 1960 as reference category. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A9: Heterogeneity: White-Collar and Blue-Collar Workers

	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) White-collar workers: flexible model (2SLS)						
Log life expectancy	-3.29	6.40***	3.63**	1.00	-0.53	4.83**	3.60***
(of specific age group)	(4.66)	(2.13)	(1.45)	(1.12)	(0.74)	(2.04)	(1.29)
First stage F–stat	2.0	3.5	6.4	2.6	5.2	5.3	4.7
Hansen test p–value	0.6	0.4	0.5	0.4	0.09	0.04	0.3
	(b) White-collar workers: flexible model (LIML)						
Log life expectancy	-5.82	8.31***	3.97**	1.26	-0.60	6.74**	4.21***
(of specific age group)	(8.09)	(3.14)	(1.60)	(1.48)	(0.86)	(3.11)	(1.62)
First stage F–stat	2.0	3.5	6.4	2.6	5.2	5.3	4.7
Hansen test p–value	0.6	0.6	0.5	0.4	0.09	0.07	0.3
	(c) Blue-collar workers: flexible model (2SLS)						
Log life expectancy	5.37	4.65**	3.41**	4.12**	3.57**	-1.28	4.32**
(of specific age group)	(3.50)	(2.17)	(1.38)	(2.02)	(1.43)	(1.94)	(1.74)
First stage F–stat	2.3	3.2	5.9	2.4	5.0	5.3	4.4
Hansen test p–value	0.7	0.8	0.9	0.9	0.8	0.4	1.0
	(d) Blue-collar workers: flexible model (LIML)						
Log life expectancy	6.09	5.30**	3.62**	4.55**	3.81**	-1.35	4.44**
(of specific age group)	(4.00)	(2.50)	(1.47)	(2.31)	(1.54)	(2.21)	(1.79)
First stage F–stat	2.3	3.2	5.9	2.4	5.0	5.3	4.4
Hansen test p–value	0.7	0.8	0.9	0.9	0.8	0.4	1.0
States	48	48	48	48	48	48	48
Observations	288	288	288	288	288	288	288
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Average wages contain observations from white-collar workers in Panels (a) and (b) and from blue-collar workers in Panels (c) and (d). Control variables are measured in 1960 and interacted with a full set of time dummies with the year 1960 as reference category. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A10: Heterogeneity: College and Non-College Workers

	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) College workers: flexible model (2SLS)						
Log life expectancy	-1.54	2.63*	2.12**	1.16	-0.01	2.86	2.27**
(of specific age group)	(3.39)	(1.59)	(1.06)	(0.96)	(1.30)	(2.97)	(0.90)
First stage F–stat	3.1	5.5	8.7	3.8	6.1	2.8	5.9
Hansen test p–value	0.7	0.8	0.6	0.4	0.09	0.06	0.7
	(b) College workers: flexible model (LIML)						
Log life expectancy	-1.74	2.76	2.19**	1.28	0.01	4.52	2.34**
(of specific age group)	(3.71)	(1.70)	(1.10)	(1.10)	(1.62)	(5.69)	(0.94)
First stage F–stat	3.1	5.5	8.7	3.8	6.1	2.8	5.9
Hansen test p–value	0.7	0.8	0.6	0.4	0.09	0.08	0.7
	(c) Non-college workers: flexible model (2SLS)						
Log life expectancy	0.14	1.48	0.94	1.67*	0.88	0.28	1.20
(of specific age group)	(2.21)	(1.11)	(0.76)	(0.86)	(0.82)	(1.06)	(0.85)
First stage F–stat	3.4	5.3	8.5	3.4	5.9	2.4	5.7
Hansen test p–value	0.4	0.6	0.7	0.6	1.0	0.09	0.6
	(d) Non-college workers: flexible model (LIML)						
Log life expectancy	0.22	1.56	1.01	1.81*	0.90	0.54	1.28
(of specific age group)	(2.65)	(1.19)	(0.80)	(0.93)	(0.83)	(1.55)	(0.89)
First stage F–stat	3.4	5.3	8.5	3.4	5.9	2.4	5.7
Hansen test p–value	0.4	0.6	0.7	0.6	1.0	0.09	0.6
States	48	48	48	48	48	48	48
Observations	336	336	336	336	336	336	336
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Average wages contain observations from workers with at least some college education in Panels (a) and (b) and from workers without any college education in Panels (c) and (d). Control variables are measured in 1960 and interacted with a full set of time dummies with the year 1960 as reference category. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A11: Adult Life Expectancy and Labor Supply by Age Cohorts: Flexible Model (2SLS)

	Flexible model (2SLS)						
	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Labor force participation (0 to 100 percent)						
Log life expectancy	-92.22	-227.50***	-108.21***	-25.16	-106.01***	-63.96*	-170.43***
(of specific age group)	(71.25)	(53.62)	(29.89)	(27.30)	(40.48)	(36.56)	(41.13)
Hansen test p–value	0.5	0.4	0.3	0.7	0.10	0.2	0.7
	(b) Usual hours per week						
Log life expectancy	-83.21**	-66.01***	-32.40**	-0.84	-34.18**	-15.37	-26.91*
(of specific age group)	(36.75)	(20.93)	(13.91)	(11.40)	(17.03)	(12.83)	(14.53)
Hansen test p–value	0.05	0.5	0.9	0.5	0.01	0.2	0.5
	(c) Usual weeks per year						
Log life expectancy	-81.63**	-85.75***	-43.09***	-4.91	-48.33**	-21.19	-30.87**
(of specific age group)	(41.08)	(25.03)	(15.14)	(12.86)	(19.01)	(16.54)	(15.14)
Hansen test p–value	0.7	0.6	0.5	0.8	0.03	0.03	0.8
	(d) Labor supply of those working (weeks × hours)						
Log life expectancy	-2384.18	-2820.10***	-1519.95**	193.90	-2094.78**	-643.37	-956.40
(of specific age group)	(1466.70)	(1043.86)	(688.12)	(581.45)	(858.40)	(631.58)	(671.45)
Hansen test p–value	0.5	0.6	1.0	0.7	0.02	0.06	0.6
First stage F–stat	3.4	5.3	8.5	3.5	5.9	2.4	5.8
States	48	48	48	48	48	48	48
Observations	336	336	336	336	336	336	336
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: The dependent variable is the labor force participation in Panel (a), usual hours worked per week in Panel (b), usual weeks worked per year in Panel (c), and hours worked per year of those working in Panel (d). All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Control variables are measured in 1960 and interacted with a full set of time dummies with the year 1960 as reference category. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A12: Adult Life Expectancy and Labor Supply by Age Cohorts: Flexible Model (LIML)

	Flexible model (LIML)						
	15–24	25–34	35–44	45–54	55–64	65+	Total
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
	(a) Labor force participation (0 to 100 percent)						
Log life expectancy	-104.64	-261.05***	-118.71***	-36.96	-213.46***	-97.21	-177.53***
(of specific age group)	(85.79)	(65.51)	(33.20)	(36.26)	(80.06)	(71.79)	(43.29)
Hansen test p–value	0.5	0.5	0.3	0.7	0.2	0.4	0.7
	(b) Usual hours per week						
Log life expectancy	-132.30*	-76.23***	-33.37**	-5.18	-95.30**	-24.01	-30.39*
(of specific age group)	(73.57)	(24.70)	(14.30)	(20.70)	(47.01)	(22.59)	(16.26)
Hansen test p–value	0.1	0.5	0.9	0.5	0.08	0.3	0.5
	(c) Usual weeks per year						
Log life expectancy	-94.45*	-93.64***	-46.03***	-6.19	-88.84**	-57.58	-33.69**
(of specific age group)	(49.23)	(27.78)	(16.11)	(14.58)	(35.89)	(79.40)	(16.32)
Hansen test p–value	0.7	0.6	0.5	0.8	0.09	0.3	0.8
	(d) Labor supply of those working (weeks × hours)						
Log life expectancy	-2685.52	-3106.16***	-1543.29**	212.52	-4100.54**	-1474.06	-1056.13
(of specific age group)	(1696.27)	(1146.91)	(697.33)	(691.18)	(1750.78)	(1974.76)	(727.12)
Hansen test p–value	0.5	0.6	1.0	0.7	0.07	0.2	0.6
First stage F–stat	3.4	5.3	8.5	3.5	5.9	2.4	5.8
States	48	48	48	48	48	48	48
Observations	336	336	336	336	336	336	336
Full controls	✓	✓	✓	✓	✓	✓	✓

Notes: The dependent variable is the labor force participation in Panel (a), usual hours worked per week in Panel (b), usual weeks worked per year in Panel (c), and hours worked per year of those working in Panel (d). All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of the respective age group. Control variables are measured in 1960 and interacted with a full set of time dummies with the year 1960 as reference category. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A13: Adult Life Expectancy and College Enrollment

	Dependent variable: college enrollment 15–24					
	Diff-in-Diff. Model (2SLS)		Flexible Model (2SLS)		Flexible Model (LIML)	
	(1)	(2)	(3)	(4)	(5)	(6)
Log life expectancy at 30	1.19**		0.85*		0.91	
	(0.56)		(0.49)		(0.58)	
Log life expectancy at 50		0.77**		0.65**		0.84*
		(0.33)		(0.31)		(0.43)
First stage F–stat	22.6	15.0	5.4	3.2	5.4	3.2
Hansen test p–value	—	—	0.1	0.3	0.1	0.4
States	48	48	48	48	48	48
Observations	336	336	336	336	336	336
Full controls	✓	✓	✓	✓	✓	✓

Notes: All regressions include state-fixed and time effects. Estimates are weighted by the initial white population of 15 to 24 year olds. Control variables are measured in 1960 and interacted with the Post-1960-treatment dummy. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college enrollment, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A14: Robustness: Moving-Window Model

	Dependent variable: log wages of whites 45–54			
	1940–1970	1940–1980	1940–1990	1940–2000
	(1)	(2)	(3)	(4)
Log life expectancy at 50	3.05**	2.09**	2.45***	2.54***
	(1.37)	(0.96)	(0.87)	(0.95)
First stage F–statistic	23.6	37.5	47.3	14.2
States	48	48	48	48
Observations	192	240	288	336
Full controls	✓	✓	✓	✓

Notes: Regression results for moving window model which adds one post-treatment at a time. Estimates are weighted by the initial white population of 45 to 54 year olds. Control variables are measured in 1960 and interacted with the Post-1960-treatment dummy. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A15: Robustness: Long-Differences Model

	Dependent variable: log wages of whites 45–54			
	1960–1970	1960–1980	1960–1990	1960–2000
	(1)	(2)	(3)	(4)
Log life expectancy at 50	5.96***	2.61*	4.72***	4.13
	(2.16)	(1.57)	(1.62)	(4.38)
First stage F–statistic	13.7	24.3	14.6	0.8
States	48	48	48	48
Observations	96	96	96	96
Full controls	✓	✓	✓	✓

Notes: Regression results for long differences model. Estimates are weighted by the initial white population of 45 to 54 year olds. Control variables are measured in 1960 and interacted with the Post-1960-treatment dummy. The full set of controls comprises log initial life expectancy, initial mortality from non-cardiovascular diseases, the initial share of college graduates, initial population density, and log initial income. Standard errors are clustered on the state level and reported in parentheses. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.

Table A16: Effect of Individual Health on Wages

	Dependent variable: respondents' log wages					
	(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

Notes: All regressions include individual-fixed, state-fixed, wave and census-region-wave effects as well as 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 has been born before a certain threshold level, e.g., 1910, and zero else. Standard errors are clustered at the individual level. Asterisks indicate significance levels: * $p < 0.1$; ** $p < 0.05$; *** $p < 0.01$.