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**LIFE EXPECTANCY AND EDUCATION:
EVIDENCE FROM THE
CARDIOVASCULAR REVOLUTION**

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Life Expectancy and Education: Evidence from the Cardiovascular Revolution*

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Abstract. This paper exploits the unexpected decline of deaths from cardiovascular diseases since the 1970s as a large positive health shock that affected predominantly old-age mortality; i.e., the fourth stage of the epidemiological transition. Using a differences-in-differences estimation strategy, we find that U.S. states with higher levels of cardiovascular-disease mortality prior the 1970s experienced greater increases in adult life expectancy and higher education enrollment. Our estimates suggest that the cardiovascular revolution caused an increase in life expectancy of 1.5 years and an increase in education enrollment of 9 percentage points, i.e. 52 percent of the observed increase from 1960 to 2000.

Keywords: adult life expectancy; higher education; cardiovascular diseases; differences-in-differences strategy.

JEL: I15; J24; N30; O10; O40.

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1. INTRODUCTION

In this study we investigate the fourth stage of the epidemiological transition, specifically the unexpected decline of deaths from cardiovascular diseases since the 1970s. Using a differences-in-differences estimation strategy, we find that U.S. states with higher levels of cardiovascular-disease mortality prior to the 1970s experienced greater increases in adult life expectancy and greater increase in tertiary education enrollment. While these results are remarkable per se, we take them one step further and use the health shock as an instrument for mortality and identify a strong positive effect of adult life expectancy on higher education.

The existence of a positive association between life expectancy and education is a well documented fact. According to one popular study, US Americans aged 25 in the year 2000 lived 7 years longer with any college education compared to those with only high school education or less (Meara et al., 2008). In many other countries a similar association between education and health has been observed. One of the major problems, however, hampering identification of the impact of longevity on education is reverse causality. Several mechanisms are conceivable generating a positive impact of education on longevity (e.g., Grossman, 2006; Strulik, 2013) and there exists a rich microeconomic literature suggesting that indeed a part of the observable positive correlation is explained by a causal impact of education on health and longevity (see Cutler et al., 2011 for a survey of the literature).

In order to uncover the causal effect of life expectancy on education and GDP per capita, Acemoglu and Johnson (2006; 2007) suggested to exploit the third stage of the epidemiological transition, i.e. the diffusion of antibiotics and new vaccines in the 1940, as an exogenous health shock. Their seminal approach has triggered a series of follow-up studies and extensions, mainly with the focus on GDP or GDP growth, which either challenged (Aghion et al., 2011; Cervellati and Sunde, 2011; Bloom et al., 2014) or confirmed (Acemoglu and Johnson, 2014; Hansen 2014; Hansen and Lønstrup, 2015) the original results. These studies vary in their specific estimation strategies but so far no attempts have been made in order to exploit a different health shock.

Concerning adult life expectancy, however, the third stage of the epidemiological transition is a less convincing health shock for identification. The diffusion of antibiotics and other new drugs in the 1940 had a large impact on death from *communicable* diseases, in particular tuberculosis, pneumonia, and malaria. It thus affected predominantly infant and child mortality (WHO, 2008). Adult mortality, in contrast, is mainly determined by degenerative, *non-communicable*

diseases caused by the aging body. In the U.S., for example, heart disease has been the leading cause of death since 1921 until the present day (DHHS, 1999a; American Heart Association, 2014). Worldwide, heart disease is the leading cause of death for adults aged 60 and above and it comes second (after HIV) for those aged 15-59 (WHO, 2008). Naturally, the health shock of the third stage of the epidemiological transition in the 1940s had no impact on deaths from cardiovascular disease, which were continuously on the rise from the beginning of the 20th century until about 1970, see Figure 1 (taken from Cutler et al., 2006).

[Figure 1 about here]

The focus of the earlier literature on communicable diseases and infant and child mortality, which follows inevitably from using the third stage of the epidemiological transition as a health shock, appears to be less appropriate to uncover the nexus between longevity and education for several reasons. Firstly, it is already conceivable from theory that lower child mortality causes human capital per capita to decline. This may be the case because of the scarring effect of diseases such that cured survivors perform worse at school than non-infected children (Aksan and Chakraborty, 2014, see e.g. Snow et al., 2003, and Bleakley, 2007 for evidence). Secondly, education of children is either compulsory or decided upon by their parents. According to standard economic theory parents would decide jointly on the number and education of their children. In this respect the conventional overlapping generation model predicts that improving child mortality should leave net fertility and thus education unchanged (Galor, 2005, 2011) while the Becker-Barro model as well as extensions of the OLG model predict increasing net fertility (Doepke, 2005; Strulik and Weisdorf, 2014), a response which is confirmed by time series analysis (Eckstein et al., 1999; Herzer et al., 2012). Increasing net fertility, in turn, would delay the fertility transition and the take-off of education and economic growth. These considerations are consistent with results from Cervellati and Sunde (2011) showing that increasing life expectancy had a positive effect on income per capita only in those countries, in which net fertility declines as a response to declining child mortality, i.e. in countries where the demographic transition had advanced far already in 1940. One interpretation of their result could be, in light of the present paper, that life expectancy at birth in these countries was strongly correlated with adult life expectancy because child mortality was already very low.

A paper which, like us, focusses on adult mortality is provided by Lorentzen et al. (2008).

This study, however, refrains from exploiting a health shock for identification and uses instead malaria ecology, climatic variables, and geographic features as instruments. Yet again, these instruments capture predominantly exogenous influences on deaths from communicable diseases and are presumably less suitable to capture the influence of natural aging and non-communicable diseases. Lorentzen et al. (2008) find that adult mortality, instrumented in this way, exerts a significant effect of on growth, investment, and fertility but, like Acemoglu and Johnson (2006), they fail to establish a significant effect on average years of schooling in the population. A recent study by Hansen (2013a), in contrast, demonstrates a positive effect of increasing life expectancy on years of schooling using a cohort based measure and the identification strategy of Acemoglu and Johnson (2006, 2007). Inspecting different potential drivers of these results, the study concludes that declines in pneumonia mortality seem to be mainly responsible. Interestingly, lower respiratory infections (including pneumonia) come fourth, after the cardiovascular diseases, as the leading cause of death for adults aged 60+ (WHO, 2008).

The question, however, remains what can be learned from the epidemiological transition of the 1940s or, more generally, from the historical decline of communicable diseases for the future of longevity and higher education in contemporary developed countries. In developed countries, primary and secondary education is largely compulsory and tertiary education, i.e. the decision to go to college or university is presumably made by adults on their own behalf. These adults experience little threat of dying prematurely from communicable diseases, which are affecting predominantly the mortality of children in developing countries. Instead they face the risk of dying in old age by non-communicable diseases, most importantly by heart attack and stroke.

Inspired by these insights we develop a new instrument, namely the strong, permanent, and unexpected decline of death from heart disease that began in the 1970, and has been dubbed the cardiovascular revolution (Foege, 1987; Vallin and Mesle, 2009; Thelle, 2011). We focus on education enrollment of 18 to 24 old US-Americans and on life expectancy at 30 (or, alternatively, at 50). This means that there is already by design relatively little concern for reverse causality, i.e. that the education decision of 18-24 year old may affect life expectancy of the contemporaneously 30 or 50 year old. Measurement error and time varying omitted variables, however, are still an issue, which we attempt to take care of with our instrumental variable approach, which follows the same principal logic as the empirical strategies employed in, e.g., Bleakley (2007), Acemoglu and Johnson (2007), Hansen (2014), Albanesi and Olivetti (2014),

and Bhalotra and Venkataramani (2015). Our focus on the relationship between adult life expectancy and education in the US also puts us in line with a recent micro study by Oster et al. (2013). Using individual data on the risk of developing Huntington disease, which is realized late in life, they establish that life expectancy has a strong positive impact on human capital investments, consistent with our evidence.

The obvious (but non-exclusive) interpretation of our results is as supportive evidence for the Ben-Porath (1967) model. According to this model, increasing adult life expectancy exerts a higher incentive to invest in education. In theory this mechanism was established more than 40 years ago. In practice, however, it has been recently challenged by Hazan’s (2009) study, which then triggered a series of refinements of the original theory (Hansen and Lønstrup, 2012; Strulik and Werner, 2012; Cervellati and Sunde, 2013). In a nutshell, these refinements offer alternative explanations of how increasing life-expectancy may trigger more education without necessarily increasing the age of retirement or life-time labor supply. We think that our approach captures the ideas of the Ben-Porath model and its refinements more closely than the existing empirical literature (e.g., Acemoglu and Johnson, 2007; Lorentzen et al., 2008; Cervellati and Sunde, 2011) because of our focus on the impact of adult longevity on higher education. However, there are other channels conceivable. For example, the middle-aged may experience an increase in expected life-time income through declining risk of death from heart disease, which may motivate them to spend more on college education of their children. Quantitatively, however, this channel seems to be of second order since most heart disease is experienced after retirement.¹

The paper is organized as follows. The next section introduces the cardiovascular revolution. Section 3 and 4 describe the data and estimation strategy. Section 5 establishes the impact of the cardiovascular revolution on adult adult life expectancy and on tertiary education. Section 6 presents our results on the impact on adult longevity on higher education enrollment. Section 7 concludes.

2. BACKGROUND: THE CARDIOVASCULAR REVOLUTION

It may be illuminating to begin with some insights from gerontology in order to contrast our approach with the existing health-shock literature. In gerontology there exists a very strong

¹For example, for white U.S. American men, the incidence of heart attack or fatal coronary heart disease is about 4 percent in the 55-64 age bracket and almost 8 and 12 percent in the 65-74 and 75-84 age brackets, respectively; see Mozaffarian et al. (2015)

association between age and the force of mortality, i.e. the conditional probability to die in the next period (e.g., Arking, 2006). This association, known as Gompertz-Makeham law, is formally given by $\mu = A + Re^{\alpha x}$, in which x is age and μ is the force of mortality. The parameter A captures “background mortality”, i.e. age-unrelated causes of death, whereas the parameters R and μ capture the impact of aging on mortality. In human history, background mortality has come down big time, particularly during the epidemiological transition of the 1940s, and it is now close to zero in developed countries (Gavrilov and Gavrilova, 1991). In developed countries most people die from degenerative, aging related diseases whose impact is covered by initial frailty R and the speed of increasing mortality with age α .

It is easy to see that any positive trends of life expectancy would soon come to an end if the trend were solely determined by falling background mortality, including the prevention and treatment of communicable diseases. Life expectancy would converge towards a constant life span determined by the aging parameters R and α . The observation that life expectancy does not (yet) seem to converge, i.e. that we observe “broken limits to life expectancy” (Oeppen and Vaupel, 2002) indicates that we, unlike all other animals, managed to manipulate the aging process itself. Inspecting the aging parameters we find indeed evidence that since the 1970s not only life expectancy (a population-specific characteristic) but also human life span (a species-characteristic) is on the rise (Strulik and Vollmer, 2013). The most likely driver of this “manufactured life-time” (Carnes and Olshansky, 2007) is medical technological progress that interfered with the natural way of human aging. The observation that human life span seems to be manipulated for the first time in the 1970 inspired our research on the cardiovascular revolution.

”Man is as old as his arteries”. This old aphorism, cited according to Dantas et al. (2012), describes succinctly that cardiovascular diseases are degenerative and inevitably related to the “natural” process of human aging.² Our arteries are simply not built to last. They dilate and stiffen through natural physical stress and fatigue (Ungvari et al., 2010). The most common type of heart disease and cause of heart attacks is coronary heart disease also known ischemic heart disease. It is caused by plaque building up along the inner walls of the arteries of the heart, which narrows the arteries and reduces the blood flow to the heart.

²In biology aging is understood as the “intrinsic, cumulative, progressive, and deleterious loss of function that eventually culminates in death” (Arking, 2006). For an introduction to the evolutionary foundations of human aging see Kirkwood (1999). For a detailed formal description of the aging process by reliability theory see Gavrilov and Gavrilova (1991).

Given that background mortality is continuously declining such that humans die increasingly of degenerative, aging-related diseases, we would expect that the incidence of death from cardiovascular diseases increases over time. Indeed, extrapolating historical trends for US-Americans, the National Heart, Lung, and Blood Institute expected about 1.6 million deaths of coronary heart disease (i.e. an age-adjusted death rate of 0.55 percent) in 2008 while actually it were “only” 0.4 million (a death rate of 0.15 percent), see NHLBI (2012, chart 3-24). The beforehand increasing trend was reverted around the year 1970. Our Figure 2 shows a replication of this finding. The death rate is measured in per 100,000 and displayed in logs. For both men and women the positive trend was reverted in about 1970.

[Figure 2 about here]

The trend reversal was preceded by a series of medical innovations, notably the pacemaker (in 1958), the cardiopulmonary resuscitation through external chest compressions (1960), the beta blocker (1962), the artificial heart (1963), the portable defibrillator (1965) and the heart transplant (1967). While there was comparatively little medical breakthroughs concerning cardiovascular diseases before the 1960s, medical progress continued afterwards, notably with the introduction of cholesterol lowering drugs (1987) and the intravascular stent (1988). The decline was also promoted by changing behavior, notably the decline in smoking since the mid 1960s (DHHS, 1999b). These changes in smoking behavior were presumably triggered by the first publications establishing a link between smoking and cancer appearing in the 1950s and the United States Surgeon General’s Report on Smoking and Health from 1964, which led to the banning of certain advertising, warning labels on tobacco products, and large scale anti-smoking campaigns (USPHS, 1964). Similar to the 1940s, the health shock starting in the 1960s could thus be conceptualized as a compound of medical and institutional innovations, improving prevention and treatment. The main difference is that the cardiovascular revolution mainly affected health of the elderly, which makes it an appropriate instrument to investigate the impact of adult longevity on education.

Another perspective to compare our contribution with is the literature on the “epidemiological transition”, originally proposed by Omran (1971) as a three-stage theory of historical health transitions, from the age of pestilence to the age of receding pandemics to the age of degenerative diseases. The unexpected decline of death from cardiovascular diseases after the 1970s

inspired sociologists and gerontologists to introduce a fourth stage, the “age of delayed degenerative diseases” (Olshansky and Ault, 1986), in which declining death rates are predominantly concentrated at advanced ages. Omran (1998) acknowledged this phenomenon and included the fourth stage in his revised theory of health transitions. The related literature on the longevity-education nexus thus focusses on the historical third stage of epidemiological transition while our approach focusses on the still ongoing fourth stage. Taking a within country perspective we expect that those US American states in which the pre-shock mortality from these diseases were highest benefitted the most from the cardiovascular revolution .

3. DATA AND PRETREATMENT DIFFERENCES

3.1. Data and Descriptive Statistics. This section describes the construction of the dataset and reports descriptive statistics. In the main analysis, the units of observation are the 48 contiguous US states for each decade throughout the period 1940–2000.

We construct two measures of higher education enrollment using individual Census data from IPUMS (Ruggles et al., 2008). The first measure, which is denoted *College Enrollment I*, is constructed using the variable called GRADEAT from IPUMS, which reports the grade or level of recent schooling for people who attended school/college. We count the number of white people aged 18-24, who report college attendance (i.e., undergraduate or graduate).³ This number is divided by the total number of white people in this age group, which is also obtained via IPUMS. Because GRADEATT is available only in the years 1960–1980, and 2000 in the Censuses, we construct a second measure of enrollment, denoted *College Enrollment II*, using the fact that the Census provides information on the respondents’ highest educational attainment throughout the period 1940–2010. Since our purpose is to derive a measure of attendance, we construct the higher education enrollment rate in, e.g., 1940 by counting the number of white people age 28-34 with 4 years (or more) of college attainment in 1950 and dividing this number with the total number of people in this group (also measured in 1950).⁴ As seen from Table 1, the first and second moments are within the same range of these two measures of higher education enrollment.

The second type of outcome variable is life expectancy for the white population at various ages, obtained on a decennial basis (1939–1941, 1949–1951, . . . , 1999–2001) from the US Decennial

³We apply the personal weight (PERWT) to ensure representatively.

⁴Notice, data on *College Enrollment II* in 1960 are missing for the states: Delaware, Idaho, Montana, North Dakota, South Dakota, Vermont, and Wyoming

Life Tables (NCHS, 2014). Due to data restrictions in the early years, we use the unweighted average of male and female life expectancy. The state average life expectancy at age 30 has increased 8.40 years from 1940 to 2000, while the corresponding increase in life expectancy at age 50 is 6.99 years.

We capture the intensity of the health shock by the number of deaths due to major cardiovascular diseases per 100 white people (age adjusted), measured prior to the breakthroughs in the understanding of cardiovascular diseases (i.e., in 1960). This variable is gathered from Grove and Hetzel (1968) and is referred to as *CVD*. Figure 3 depicts the spatial pattern across US states, and it reveals that Northeastern states generally had higher levels of *CVD* in 1960.

Motivated by the evidence described in Section 2, we code the (medical) interventions to occur after 1960, implying that 1940–1960 is designated as the pretreatment period and 1970–2000 as the post treatment period. We use the interaction between *CVD* and a full set of year fixed effects, as well as the interaction between *CVD* and an indicator variable (*Post*) which equals one from 1970 onwards, to estimate how the cardiovascular revolution impacted the subsequent development of adult life expectancy and higher education.

To control for the possible influence of non-cardiovascular mortality on life expectancy, we also construct the variable *Initial Mortality*, which is the age-adjusted mortality rate from the causes of death that are *not* attributable to cardiovascular diseases in 1960. This variable is also obtained from Grove and Hetzel (1968) and interacted with the time indicator.

We furthermore derive an income measure (*Log income, age 30 plus*), exploiting again micro data from IPUMS. This variable is available 1950–2000, but we mainly use the 1960 variation interacted with the indicator variable as a control. Table 1 provides summary statistics for the all the variables explained so far.

In addition, we consider the following variables (measured in 1960): average years of schooling in the workforce and log capital per worker. These variables are taken from Turner et al. (2007), and again, to account for this variation in our regressions, they are also interacted with the indicator.

[Table 1 about here]

[Figure 3 about here]

3.2. Pretreatment Differences in the Outcomes. Because our empirical strategy exploits a differences-in-differences (DD) application to estimate the effect of the cardiovascular revolution on life expectancy and education (see also the next section), the identifying assumption is identical pretreatment trends in the outcomes between treatment and control states. Due to the continuous nature of our treatment measure, this is the same as saying that *CVD* should be uncorrelated with changes in the outcomes before treatment. Therefore, as an initial step of testing this assumption, we now explore pretreatment differences in the level and the change of the outcomes for states with different levels of *CVD*. In the level specification, we regress the outcome j in state s (Y_s^j) on the cardiovascular mortality rate in 1960 (CVD_s):

$$Y_{s1960}^j = \alpha + \beta_j CVD_s + \mathbf{X}_s' \gamma + \varepsilon_s, \quad (1)$$

where \mathbf{X}_s' is vector of controls and ε_s is the error term. The estimated β_j reflects pretreatment (conditional) differences in the outcome j for states with different mortality rates from cardiovascular diseases in 1960. Before turning to our change specification, it is important to note that $\hat{\beta}_j \neq 0$ does necessarily imply that the identifying assumption is violated as the DD model allows us to non-parametrically control for state fixed effects.

In the change specification, we regress the pretreatment changes in the outcome between 1940 and 1960 (ΔY_s^j) on CVD_s and \mathbf{X}_s' :

$$\Delta Y_s^j = \bar{\alpha} + \beta_j^\Delta CVD_s + \mathbf{X}_s' \gamma + \varepsilon_s. \quad (2)$$

The estimate of β_j^Δ provides the same information as a falsification exercise testing if the CVD-shock variable ($CVD \times Post$) in the DD model has any predictive power for the development of the outcomes in the pretreatment period (i.e., 1940–1960). If $\hat{\beta}_j^\Delta = 0$ cannot be rejected, the falsification test would support the identifying assumption of common pretrends.

Table 2 reports weighted least squares estimates of β_j and β_j^Δ in equations (1) and (2), respectively. Columns (1) and (3) report the unconditional estimates, whereas columns (2) and (4) report these estimates conditional on two state-level controls.⁵ Unsurprisingly, the relationship between life expectancy at age 30 (or 50) in 1960 and *CVD* is negative and statistically significant at the 1 percent level (columns 1 and 2). Thus, states with higher death rates from *CVDs* also had lower adult life expectancies, suggesting that in fact *CVD* mortality was quantitatively

⁵The covariates are: initial mortality and income

important. More importantly for our analysis, however, we also find that *CVD* is completely unrelated to changes in life expectancy between 1940 and 1960, which implies that the *CVD* shock has no predictive power for life expectancy in the pretreatment period (columns 3 and 4). Figure 4 graphically shows that this "lack" of relationship is *not* driven by some outlier states.

Furthermore, the estimates reported in Table 2 reveal that states with different rates of *CVD* mortality were similar in terms of higher education enrollment in the pretreatment year 1960 (columns 1 and 2). These states also experienced similar changes in the *College Enrollment I* between 1940 and 1960; see columns (3) and (4), and Figure 5. Overall, these findings suggest that the *CVD*-shock variable is not likely to pick up preexisting trends in health or education.

[Table 2 and Figures 4 and 5 about here]

4. EMPIRICAL FRAMEWORK

This section explains our empirical strategy, which starts out by studying the effects of the cardiovascular revolution on the development of adult life expectancy and higher education. We hereafter apply these findings in an instrumental-variable approach to identify the reduced-form impact of adult life expectancy on higher education.

By comparing the relative change in the outcomes of interest in the pretreatment period to the posttreatment period between states with different treatment intensities, as measured by *CVD_s*, the empirical strategy follows a DD type of estimation approach. The identifying assumption is, accordingly, common trends in the observables prior to treatment. The baseline estimation equation in (3) allows us to test this assumption while at the same time providing insights into the dynamics of the shock:

$$Y_{st} = \sum_{k=1940}^{2000} \pi_k (CVD_s \times Post_t^k) + (\mathbf{X}'_s \times Post_t) \eta + \lambda_s + \tau_t + \epsilon_{st}, \quad (3)$$

where the outcome of interest, denoted by Y_{st} , is either adult life expectancy or the higher education enrollment rate (i.e., *College Enrollment I and II*) for the white population in state s at time t , CVD_s is the age-adjusted death rate from major cardiovascular diseases for the white population in 1960, which is interacted with a full set of year fixed effects; the omitted year of comparison is 1960. We also include a vector of controls (\mathbf{X}'_s), containing the age-adjusted mortality rate from non-CVD causes, the initial enrollment rate, initial personal income. These

cross-sectional variables are measured in 1960 and interacted with an indicator ($Post_t$) turning on after 1960. The panel structure of the data allows us to non-parametrically control for state (λ_s) and time (τ_t) fixed effects. We cluster the error term (ϵ_{st}) at the state level, ensuring that the standard errors of our estimates are robust to arbitrary serial correlation in the states (e.g., Cameron and Miller, 2013). We estimate 10-year panel models starting (at the earliest) in 1940 and ending in 2000 for the 48 contiguous states.

While the specification in equation (3) allows us to investigate the assumption of identical pretrends, along with the dynamic behavior of the outcome due to the shock, we also estimate a model, where the time-period fixed effects in the first term on the right-hand side are replaced with the indicator variable. The estimates from such a model specification can be used to quantify the average effect of the cardiovascular revolution over the 30-year period from 1970 to 2000.

At this point one should note that as the uptake of the new cardiovascular-medical technologies is endogenous, our strategy exploits the fact that some states stood to benefit more from the breakthroughs—in terms of potential health improvements—as measured by *CVD*-mortality in 1960 prior to shock.⁶ Thus, we are measuring the *intention-to-treat* effect, which is smaller than the average causal effect of on those who were actually treated (Angrist and Pischke, 2008). This means that we provide a conservative quantification of how the cardiovascular revolution influenced these outcomes. This type of empirical strategy has been deployed before by, e.g., Bleakley (2007), Acemoglu and Johnson (2007), Albanesi and Olivetti (2014) and Bhalotra and Venkataramani (2015).

Finally, in an application of our findings, we exploit the cardiovascular revolution to identify the effect of adult life expectancy on higher education. The second-stage regression takes the following form:

$$College\ enrollment_{st} = \theta Life\ Expectancy_{st} + (\mathbf{X}'_s \times Post_t)\eta + \lambda_s + \tau_t + \epsilon_{st}. \quad (4)$$

Thus, in this part of the analysis, we focus on the relationship between higher education and adult life expectancy. The identifying assumption in this 2SLS strategy would be $Cov(CVD_s \cdot Post_t, \epsilon_{st}) = 0$. Since it can plausibly be argued that the timing of the cardiovascular revolution is largely orthogonal to state-specific economic developments, the potential threat to identification

⁶This has the additional advantage that we do not need to collect data on actual adoption rates, which seems broadly unavailable.

is that *CVD* is related to economic changes before the shock, which we tested in the first part of the analysis. That being said, we cannot exactly identify the mechanism through which adult life expectancy influences higher education. As discussed in the Introduction, it is possible that the cardiovascular revolution was associated with an income gain, which potentially could have been transferred from one generation to the next in order to buy more higher education.

5. THE CARDIOVASCULAR REVOLUTION, LIFE EXPECTANCY, AND HIGHER EDUCATION

5.1. Adult Life Expectancy. This subsection presents the results from estimating the effect of the cardiovascular revolution on adult life expectancy. Throughout the analysis, the regressions include state and year fixed effects and are weighted by the white population aged 18-24 in 1960.⁷

Panel A of Table 3 reports the results from estimating equation (3). Columns (1)–(3) report estimates for life expectancy at age 30, while columns (4)–(6) report estimates for life expectancy at age 50. In column (1), we see that $\hat{\pi}_{1940}$ and $\hat{\pi}_{1950}$ are basically zero, indicating that before treatment life expectancy was not trending any differently between treatment and control states. By 1970, in contrast, we find a positive and significant relationship between *CVD* and adult life expectancy. This effect accumulates over time: $\hat{\pi}_{1970}$ is equal to 4.97 (s.e. = 1.40), whereas $\hat{\pi}_{2000}$ is equal to 13.96 (s.e. = 4.37).

Columns (2) and (3) add controls for the initial mortality environment, initial income, and the initial enrollment rate. In order to estimate the contribution of these cross-sectional measures, they are interacted with the time indicator as all the specifications include state-fixed effects. We find that the observed pattern from column (1) stays intact, which is also evident in Figure 6 where the estimates and 95 percent confidence intervals are plotted from column (3).⁸

Columns 4–6 report the evidence for life expectancy at age 50. We arrive at the same conclusion that there are no trends of the estimated interaction effects prior to the cardiovascular revolution, whereas by 1970 “more treated” states begin to experience greater increases in life expectancy at age 50. Overall, the identifying assumption in the DD framework of common pretrends is supported by the evidence reported in Table 3.

⁷In general, similar conclusions are obtained using the entire white population as an alternative weight or from unweighted estimation.

⁸It should also be noted that the same pattern, though slightly more *precisely* estimated, is obtained by controlling flexibly for the cross-sectional measures, that is, interacting them with time-period fixed effects instead of the indicator.

Panel B of Table 3 reports the results from estimating equation (3), where *CVD* is interacted with the indicator variable instead of the time-period fixed effects. Thus, this model provides an average effect of the shock. Column (3), which includes the baseline control interactions, shows an estimated coefficient of the CVD shock of 7.62 (s.e. = 2.62). This suggests that in states with a higher death rate from cardiovascular diseases in 1960, life expectancy at age 30 was growing significantly faster during the period 1970–2000. The coefficient estimate implies that increasing the treatment intensity from the lowest to the highest level is associated with an increase in life expectancy of 1.18 years.

In terms of the average decrease in cardiovascular-disease mortality between 1960 and 2000,⁹ our baseline estimate suggests that the cardiovascular revolution contributed to an increase in life expectancy with about 1.53 years. We believe that this is within a reasonable range since Cutler et al. (2006) argues that the drop in CVD mortality was associated with an increase in life expectancy at birth with about 4.9 years, where as much as 3.27 years can be attributed to medical advances within CVD diseases (i.e., excluding the possible contribution from changes in smoking behavior). Thus, taken these numbers at face value, and remembering that we are measuring an ITT effect, would indicate a adoption rate of around 47 percent.

[Table 3 about here]

[Figure 6 about here]

5.2. Higher Education. Table 4 documents the relationship between the CVD shock and higher education enrollment. In columns (1)-(3), we report the results for the outcome variable *College Enrollment I*, which is only available in the years 1960-1980 and 2000, while columns (4)-(6) provides estimates for *College Enrollment II*; available all the years, although data from seven states are missing in 1960. Panel A reveals that before 1970 college enrollment was not trending differently by the treatment-intensity measure (i.e., *CVD*). However, consistent with the argument that the cardiovascular revolution led to increase in the higher education enrollment rate, the estimates generally become positive after the 1960s. Compared to results for adult life expectancy, the effect seems to be one decade delayed, since first by 1980 the estimates

⁹Data from the National Heart, Lung, and Blood Institute indicate that the cardiovascular disease mortality rate decreased by about 50 percent from 1960–2000.

become statistically significant. This is likely to be explained by the fact that people need to realize that adult life expectancy has suddenly gone up. Figure 7 visualize the estimates from the specification reported in column (6).

[Figure 7 about here]

Panel B repeats the exercise (from Table 3) of estimating the average effect of the cardiovascular revolution over the period. In line with the flexible estimates, we first observe that the point estimates on *College Enrollment II* are somewhat larger than the point estimates on *College Enrollment I*, however, these are within the same 95 percent confidence bands. Considering *College Enrollment II*, we find an effect of 0.47 (s.e. = 0.12), suggesting that the average decline in cardiovascular was associated with an increase in the college enrollment rate of about 9 percentage points, which can be compared to an observed total increase of about 52 percentage points from 1940 to 2000.

[Table 4 about here]

6. THE OF EFFECT OF ADULT LIFE EXPECTANCY ON HIGHER EDUCATION

While the analysis so far has established that the cardiovascular revolution led to substantial increases in adult life expectancy and higher education, this section uses this evidence to study the reduced-form impact of life expectancy on education. Thus, the estimation equation is (4), and now we exploit the CVD-shock to instrument life expectancy at age 30; similar results are obtained using life expectancy at age 50.

Panel A of Table 5 reports the least-square (LS) estimates. We find positive and statistical significant effects. Taken at face value, the baseline LS estimate of θ , reported in column (3), suggests that for one additional year of life expectancy *College Enrollment I* increases by about 2 percentage points. As seen from columns (4)-(6), the point estimates for *College Enrollment II* are generally larger in magnitude, although not statistically.

Due to the usual endogeneity concerns, such as time varying omitted variable, reverse causality, and measurement error, the LS estimates cannot be interpreted causally. For these reasons, we next exploit the cardiovascular revolution as an exogenous source of within-state variation in life expectancy as documented in the previous section.

The 2SLS estimates, reported in panel B of Table 5, show that the effect of life expectancy on the higher education enrollment rate is positive and highly significant. The 2SLS estimates are also larger in magnitude compared to the corresponding LS estimates, although not statistically. Accordingly, a one year increase in life expectancy translates into a 3.8 percentage points higher college enrollment rate (column 3), whereas for our second measure of enrollment the corresponding number is 6.1 percentage points. It should be noted, however, that these numbers are not statistically significant different from each other.

The instrument quality in terms of the Kleibergen-Paap F-statistic is reported at the bottom of the table. Because the F-statistics are around the rule-of-thumb level of 10 in all the specifications, concerns about weak-instrument bias are moderate. Nevertheless, Anderson-Rubin p-values are also reported in the table.

[Table 5 about here]

Table 6 considers various extensions to the baseline specification with *College Enrollment I* as the outcome variable, and Table 7 reports the same robustness tests for *College Enrollment II*. The first three columns of these tables add potential confounders. Firstly, Columns (1) and (2) demonstrate that our conclusion is robust to initial variation in average years of schooling in the workforce and physical capital per worker (both interacted with the indicator). Secondly, even though *Log income, age 30 plus* is likely to be a bad control variable, and our baseline model already includes the variation in this variable in 1960 (i.e., *Initial income*) interacted with the time indicator, column (3) reveals that the effect of life expectancy on enrollment is quantitatively and qualitatively robust to the inclusion of this variable. Moreover, the effect of the CVD shock on life expectancy seems also robust to these additional controls as the first-stage F-statistics are at the least about nine. Thus, our findings seems not to be driven by some convergence process in education related to the initial level of enrollment, years of schooling in the workforce, or income.

The remaining columns report the results from various sample splits. Columns (5)–(7) make the split by geographical area. This evidence demonstrates that our results seem to be driven by Northern and Southern states as we find positive and statistical significant relationships in these sub samples: the 2SLS estimates, as well as the first-stage F-statistics, are very similar for these states in both tables, whereas the impact of life expectancy on enrollment is basically

zero for Western states, which seems to be related to the fact that the first-stage is almost non-existing (see column 7); in an unreported specification, we show that the LS estimate for the Western states is positive and highly significant. Nevertheless, as an alternative check along the same lines, estimating a model for the full sample of the 48 contiguous US states, which non-parametrically takes into account the variation occurring between the North, South, and West over time by including region-by-year fixed effects, we find that the estimate of the first-stage estimates of the CVD shock in life expectancy continues to be positive and statistically significant, and the corresponding 2SLS estimate remains also quantitatively and qualitatively robust.

The final two columns of the table report the results from splitting the sample into rich and poor states as measured by median income in 1960. Considering Table 6, the impact on enrollment is largely the same in initially "poor" compared to initially "rich" states, but the first stage appears to be stronger for the former group of states. The same pattern emerges for *College Enrollment I* in Table 7, although the 2SLS point estimate is larger for rich states. As with the geographical sample splits, we have also estimated models that only exploit variation within the two groups for the full sample of states with results close to our baseline findings.

[Tables 6 and 7 about here]

7. CONCLUSION

This paper documents positive effects of the so-called cardiovascular revolution on adult life expectancy and higher education, strongly suggesting a positive effect of life expectancy on education. While many existing studies exploit the decline in infectious-disease mortality—known as the third stage of the epidemiological transition—to study the relationship between health and economic outcomes, we focus on the cardiovascular revolution as the incidence of cardiovascular diseases is concentrated in adulthood and, thus, more suitable for testing the relationship between adult life expectancy and higher education. Our findings are supportive of the so-called Ben-Porath mechanism according to which higher life-expectancy induces more education. The cardiovascular revolution, however, may also have increased income of the middle-aged, a part of which could have been used to finance more higher education of the next generation. We believe that this type of argument is not a threat to identification but rather

another possible channel that could explain our findings. Accordingly, we interpret the 2SLS estimates as reduced-form evidence on the effect of adult life expectancy on higher education.

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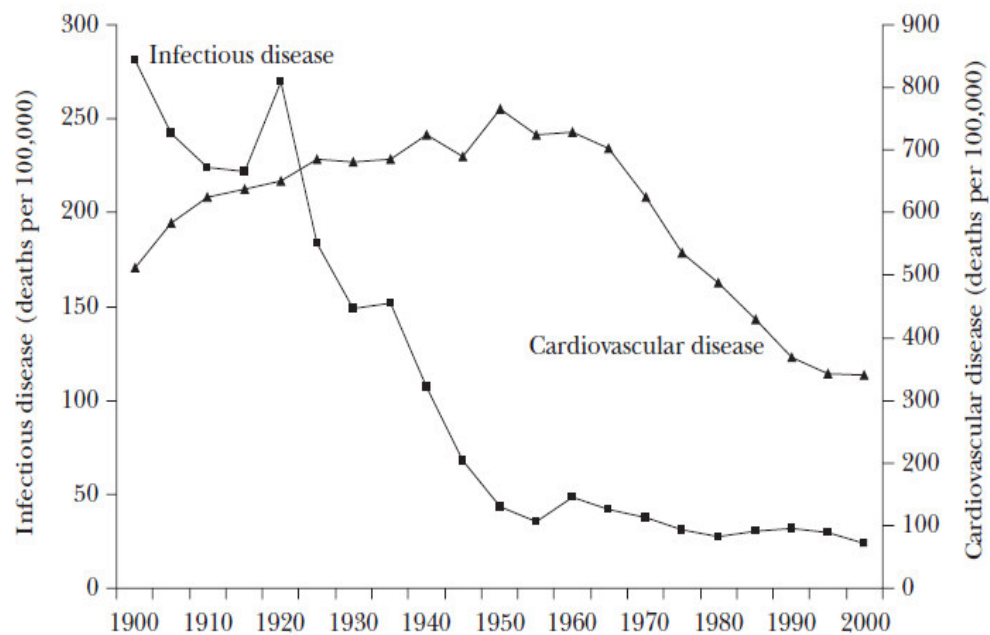
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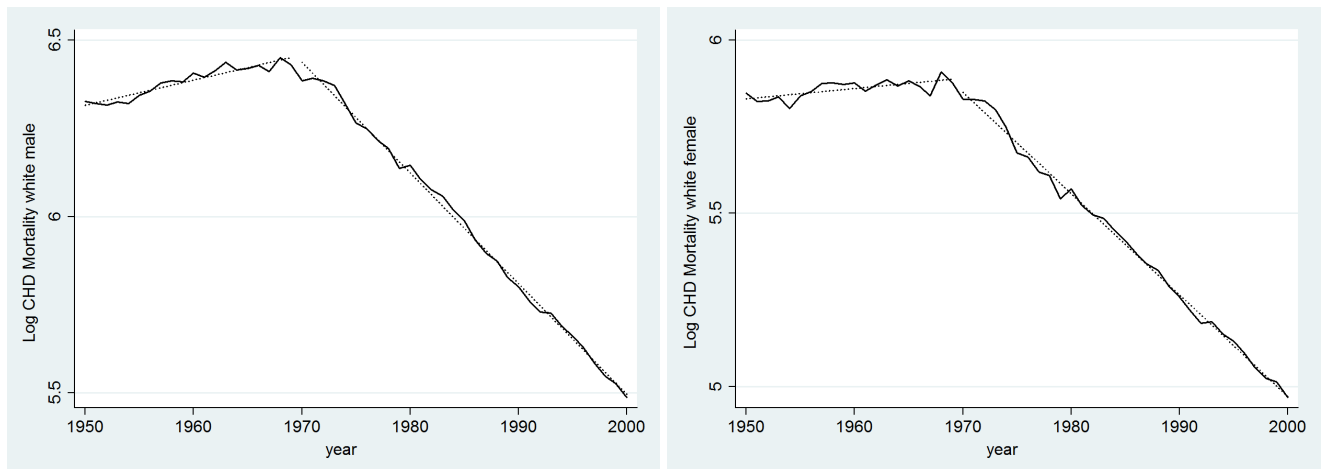
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Figure 1: Mortality from Infectious Diseases and Cardiovascular Diseases, United States, 1900-2000



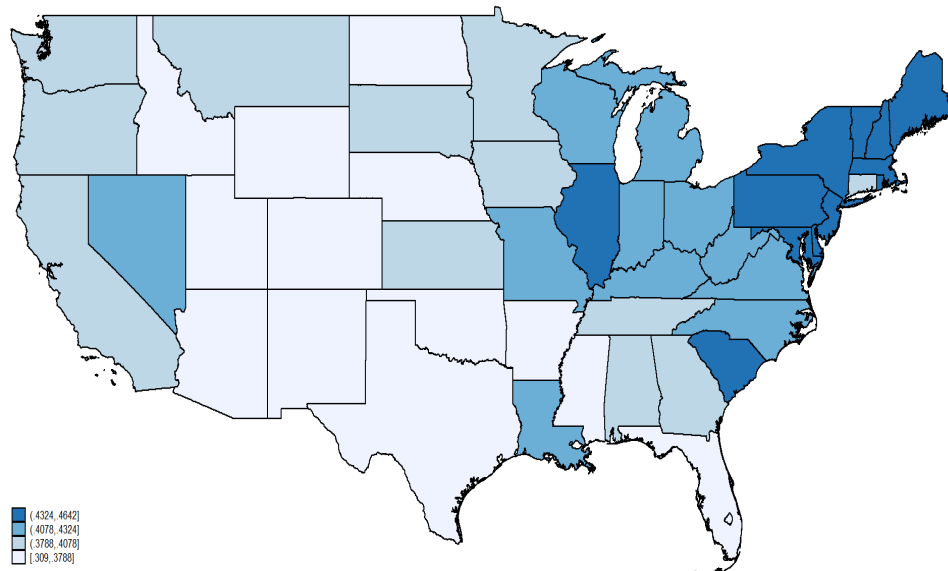
Notes: This figure shows the age-adjusted mortality rate from infectious diseases and cardiovascular diseases for the US over the 20th century. Source: Cutler et al. (2006).

Figure 2: Annual Development of US CVD-mortality 1950–2000 (Coronary Heart Disease Mortality by Sex)



Notes: The left (right) figure shows the annual development in the log age-adjusted mortality rate from Coronary Heart Disease, CHD, for white males (females) in the US. Data source: National Heart, Lung, and Blood Institute.

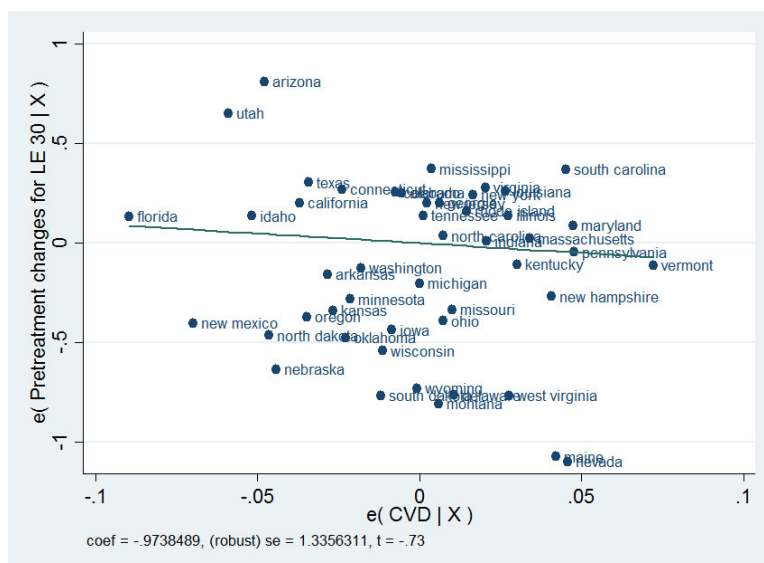
Figure 3: Spatial Distribution of 1960-CVD Mortality



Notes: This map pictures the geographical distribution of *CVD* for the 48 contiguous states.

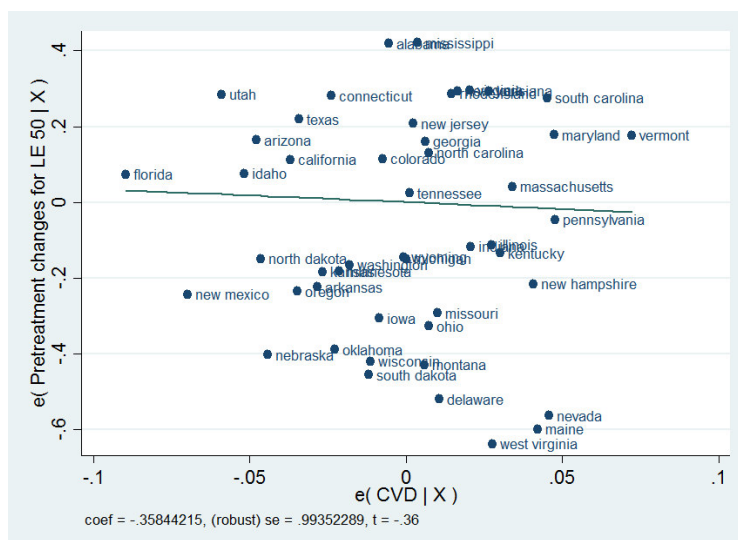
Figure 4: Pretreatment Changes in Adult Life Expectancy by CVD

Panel A: Life Expectancy at age 30



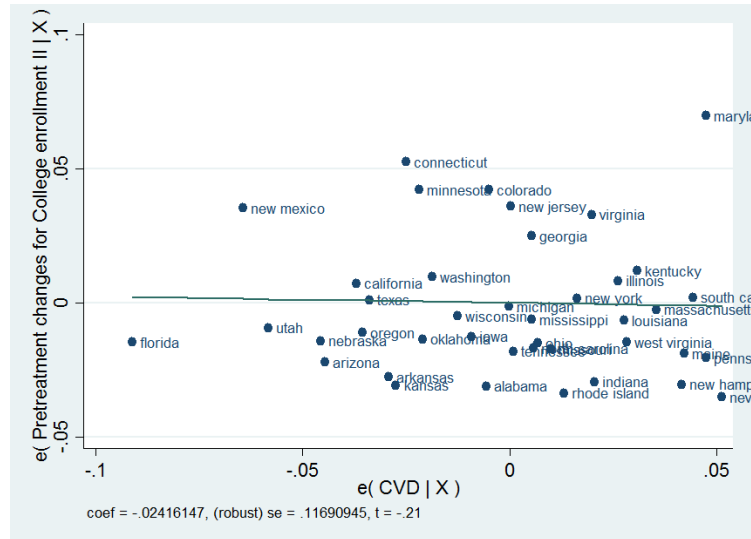
Notes: This figure shows the relationship between the change in pretreatment (i.e, 1940-1960) life expectancy at age 30 and the CVD variable. Source is column 4 of Table 1.

Panel B: Life Expectancy at age 50.



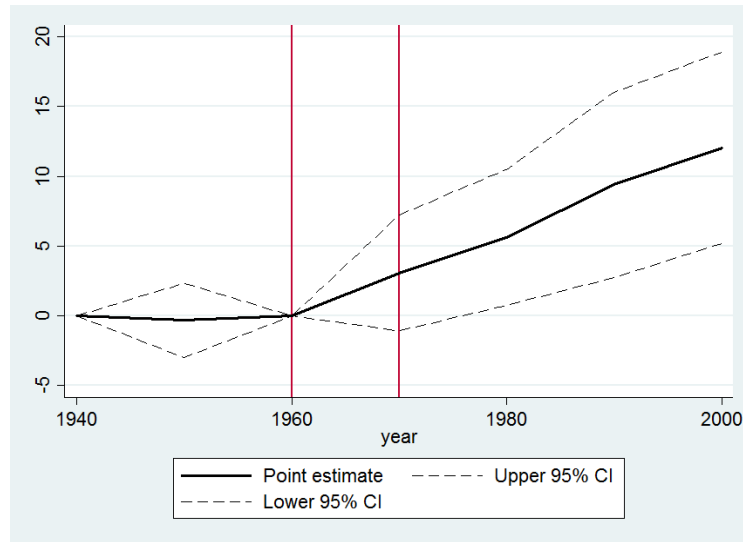
Notes: This figure shows the relationship between the change in pretreatment (i.e, 1940-1960) life expectancy at age 50 and the CVD variable. Source is column 4 of Table 1.

Figure 5: Pretreatment Changes in College enrollment by CVD



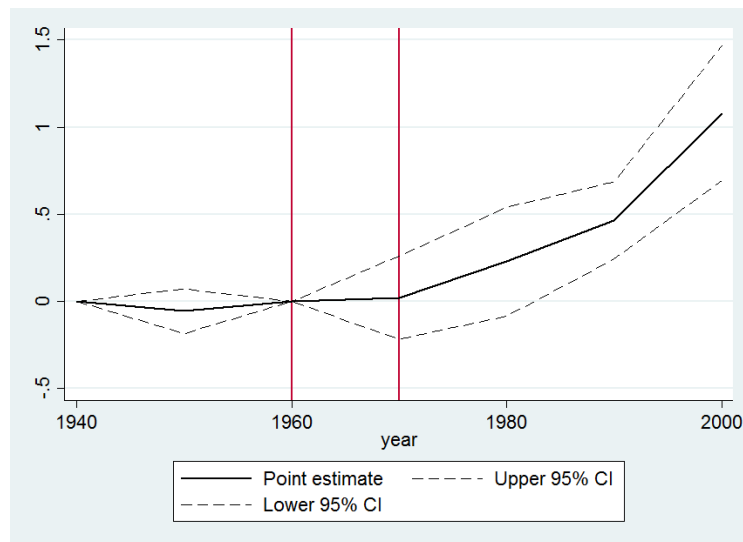
Notes: This figure shows the relationship between the change in pretreatment (i.e, 1940-1960) college enrollment and the CVD variable. Source is column 4 of Table 1.

Figure 6: Estimated differences in adult life expectancy by CVD



Notes: This figure depicts the flexible estimates for life expectancy at age 30. The vertical lines indicate before and after treatment, respectively. Source: column 3 in Table 3.

Figure 7: Estimated differences in college enrollment by CVD



Notes: This figure depicts the flexible estimates for college enrollment II. The vertical lines indicate before and after treatment, respectively. Source: column 6 in Table 4.

Table 1: Descriptive Statistics

	Obs:	Mean	Std. Dev.	Min	Max
Life expectancy, age 30	336	45.13	2.835	38.68	52.15
Life expectancy, age 50	336	27.02	2.394	22.06	33.18
CVD \times Post	336	0.230	0.201	0	0.464
Initial mortality \times Post	336	0.211	0.184	0	0.468
College enrollment I	185	0.253	0.0884	0.0882	0.494
College enrollment II	329	0.203	0.110	0.00867	0.504
Log income, age 30 plus	288	9.966	0.391	8.943	10.70

Notes: This table reports descriptive statistics for the main variables used in the empirical analysis. Data on adult life expectancy, CVD*Post, Initial mortality*Post are for the years 1940-2000 for the 48 contiguous states. College enrollment I is for the years 1960-1980 and 2000 for the 48 contiguous states. College enrollment II is for the years 1940-2000, however, data on this variable in 1960 are missing for the states: Delaware, Idaho, Montana, North Dakota, South Dakota, Vermont, and Wyoming. Log income, age 30 plus is for the years 1950-2000 for the 48 contiguous states.

Table 2: Pretreatment State Characteristics by 1960-CVD Mortality

	Pretreatment levels		Pretreatment changes	
	Without Covariates	With Covariates	Without Covariates	With Covariates
	(1)	(2)	(3)	(4)
Life Expectancy, age 30	-17.24*** (1.645)	-18.05*** (1.103)	-0.0985 (1.868)	0.725 (1.286)
Life Expectancy, age 50	-18.53*** (1.397)	-18.65*** (0.884)	0.592 (1.465)	0.407 (1.071)
College enrollment I	0.0333 (0.131)	-0.0270 (0.132)	NA	NA
College enrollment II	-0.0156 (0.143)	-0.0852 (0.110)	0.00719 (0.117)	-0.0489 (0.0967)

Notes: The table reports least squares estimates (weighted by the white population age 18-24 in 1960) of CVD in equations (1) and (2). The dependent variables are: life expectancy at age 30, life expectancy at age 50, college enrollment I and II for the white population. Pretreatment level refers to the level of the dependent variable in 1960, while pretreatment changes refers to the change in the dependent variable during the pretreatment-period 1940–1960. The covariates are: the number of deaths from all other causes than CVD per 100 white population in 1960 and Log income, age 30 plus in 1960 (i.e., initial income). Constants and controls are not reported. Standard errors are robust clustered at the state level.

*** p<0.01, ** p<0.05, * p<0.1.

Table 3: The Cardiovascular Revolution and Adult Life Expectancy

	Dependent Variable:					
	Life Expectancy, age 30			Life Expectancy, age 50		
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Flexible Estimates						
CVD \times 1940	9.85e-05 (0.00187)	9.85e-05 (0.00187)	9.85e-05 (0.00187)	-0.000592 (0.00146)	-0.000592 (0.00146)	-0.000592 (0.00146)
CVD \times 1950	-0.318 (1.361)	-0.318 (1.361)	-0.318 (1.361)	-0.0562 (1.044)	-0.0562 (1.044)	-0.0562 (1.044)
CVD \times 1970	4.968*** (1.404)	2.949 (2.209)	3.083 (2.116)	4.757*** (1.199)	2.572 (1.917)	2.745 (1.866)
CVD \times 1980	7.517*** (2.235)	5.498** (2.595)	5.631** (2.489)	7.162*** (2.024)	4.977** (2.269)	5.150** (2.168)
CVD \times 1990	11.28*** (2.510)	9.258*** (3.365)	9.392*** (3.394)	11.12*** (2.555)	8.940*** (2.767)	9.113*** (2.766)
CVD \times 2000	13.96*** (4.369)	11.94*** (3.537)	12.07*** (3.497)	15.17*** (4.374)	12.99*** (3.965)	13.16*** (3.974)
Panel B: DD Estimates						
CVD \times Post	9.503*** (3.141)	7.484*** (2.635)	7.618*** (2.623)	9.771*** (3.069)	7.585*** (2.664)	7.758*** (2.626)
Controls (\times Post):						
Initial mortality	No	Yes	Yes	No	Yes	Yes
Initial enrollment	No	Yes	Yes	No	Yes	Yes
Initial income	No	No	Yes	No	No	Yes
# Observations	346	346	346	346	346	346
# States	48	48	48	48	48	48

Notes: The table reports least squares estimates weighted by the white population age 18-24 in 1960. The observation period is 1940–2000. All regressions include state and year fixed effects. The dependent variables are adult life expectancy at age 30 and 50. CVD is the age-adjusted number of deaths from cardiovascular diseases per 100 white population in 1960. Initial mortality is the number of deaths from all other causes than CVD per 100 white population in 1960. Initial enrollment is the variable college enrollment rate I measured in the year 1960. Initial income is the variable log income, age 30 plus measured in the year 1960. Panel A reports flexible estimates, where CVD is interacted with a full set of year fixed effects and the omitted year of comparison is 1960. Panel B report DD estimates, where CVD is interacted with the indicator Post which equals one after 1960. Constants are not reported. Standard errors are robust clustered at the state level.

*** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Table 4: The Cardiovascular Revolution and Higher Education Enrollment

	Dependent Variable:					
	College enrollment I			College enrollment II		
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: Flexible Estimates						
CVD \times 1940				-4.39e-06 (0.000116)	-2.03e-06 (0.000116)	-5.06e-06 (0.000116)
CVD \times 1950				-0.0552 (0.0653)	-0.0528 (0.0659)	-0.0559 (0.0651)
CVD \times 1970	0.0280 (0.0882)	0.0113 (0.0912)	0.00766 (0.0941)	0.109 (0.0819)	0.0134 (0.120)	0.0220 (0.122)
CVD \times 1980	0.298*** (0.0938)	0.281*** (0.0940)	0.277*** (0.0938)	0.316** (0.151)	0.220 (0.151)	0.228 (0.159)
CVD \times 1990				0.552*** (0.122)	0.456*** (0.123)	0.465*** (0.113)
CVD \times 2000	0.538*** (0.109)	0.522*** (0.110)	0.517*** (0.111)	1.167*** (0.217)	1.071*** (0.214)	1.080*** (0.197)
Panel B: DD Estimates						
CVD \times Post	0.291*** (0.0763)	0.280*** (0.0792)	0.270*** (0.0795)	0.556*** (0.142)	0.517*** (0.151)	0.469*** (0.124)
Controls (\times Post):						
Initial mortality	No	Yes	Yes	No	Yes	Yes
Initial enrollment	No	Yes	Yes	No	Yes	Yes
Initial income	No	No	Yes	No	No	Yes
# Observations	185	185	185	329	329	329
# States	48	48	48	48	48	48

Notes: The table reports least squares estimates weighted by the white population age 18-24 in 1960. The observation period is 1940–2000. All regressions include state and year fixed effects. The dependent variables are college enrollment I and II. CVD is the age-adjusted number of deaths from cardiovascular diseases per 100 white population in 1960. Initial mortality is the number of deaths from all other causes than CVD per 100 white population in 1960. Initial enrollment is the variable college enrollment rate I measured in the year 1960. Initial income is the variable log income, age 30 plus measured in the year 1960. Panel A reports flexible estimates, where CVD is interacted with a full set of year fixed effects and the omitted year of comparison is 1960. Panel B report DD estimates, where CVD is interacted with the indicator Post which equals one after 1960. Constants are not reported. Standard errors are robust clustered at the state level.

*** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$

Table 5: Adult Life Expectancy and Higher Education Enrollment

	Dependent Variable:					
	College enrollment I			College enrollment II		
	(1)	(2)	(3)	(4)	(5)	(6)
Panel A: LS estimates						
Life Expectancy at age 30	0.0166*** (0.00332)	0.0192*** (0.00376)	0.0212*** (0.00361)	0.0327*** (0.00641)	0.0353*** (0.00705)	0.0340*** (0.00597)
Panel B: 2SLS estimates						
Life Expectancy at age 30	0.0329*** (0.00687)	0.0395*** (0.0114)	0.0380*** (0.00978)	0.0580*** (0.0189)	0.0606** (0.0236)	0.0610*** (0.0230)
Anderson-Rubin [p-value]	[0.0005]	[0.0013]	[0.0018]	[0.0004]	[0.0021]	[0.0006]
Controls (\times Post):						
Initial mortality	No	Yes	Yes	No	Yes	Yes
Initial enrollment	No	No	Yes	No	No	Yes
Initial income	No	No	Yes	No	No	Yes
First-stage F-stat.	11.25	8.714	10.00	8.830	7.802	8.099
# Observations	185	185	185	329	329	329
# States	48	48	48	48	48	48

Notes: Panel A reports least squares estimates (weighted by the white population age 18-24 in 1960). Panel B reports two stage least squares estimates (weighted by the white population size in 1960) using the instrument CVD \times Post. The observation years are 1960-1980 and 2000 in columns 1-3 and 1940-2000 in columns 5-6. All regressions include state and year fixed effects. The dependent variables are college enrollment I and II. CVD is the age-adjusted number of deaths from cardiovascular diseases per 100 white population in 1960. Initial mortality is the number of deaths from all other causes than CVD per 100 white population in 1960. Initial enrollment is the variable college enrollment rate I measured in the year 1960. Initial income is the variable log income, age 30 plus measured in the year 1960. Post is an indicator which equals one after 1960. Constants are not reported. Standard errors are robust clustered at the state level.

*** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

Table 6: Sensitivity Tests and Sample Splits

	Dependent Variable is College enrollment I							
	Additional Controls:				Sample Splits:			
	(1)	(2)	(3)	(5)	(6)	(7)	(8)	(9)
Life Expectancy at age 30	0.0381*** (0.00895)	0.0510*** (0.0124)	0.0464*** (0.0112)	0.0356*** (0.00844)	0.0350** (0.0149)	-0.347 (0.725)	0.0394*** (0.0152)	0.0296* (0.0174)
Controls (· Post):								
Initial Mortality	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Enrollment	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Income	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Education	Yes	No	Yes	No	No	No	No	No
Initial Physical Capital	No	Yes	Yes	No	No	No	No	No
Log income, age 30 plus	No	No	Yes	No	No	No	No	No
First-stage F-statistics	12.42	9.89	10.40	8.71	13.29	0.29	2.17	5.95
Anderson-Rubin [p-value]	[0.0020]	[0.0002]	[0.0002]	[0.0065]	[0.1545]	[0.1308]	[0.1885]	[0.0255]
# Observations	185	185	185	81	63	41	90	95
# States	48	48	48	21	16	11	23	25

Notes: The table reports two stage least squares estimates, weighted by the white population size age 18-24 1960, using the instrument CVD \times Post. The observation years are 1960-1980 and 2000. All regressions include state and year fixed effects. The dependent variables are college enrollment I. CVD is the age-adjusted number of deaths from cardiovascular diseases per 100 white population in 1960. Initial mortality is the number of deaths from all other causes than CVD per 100 white population in 1960. Initial enrollment is the variable college enrollment rate I measured in the year 1960. Initial income is the variable log income, age 30 plus measured in the year 1960. Post is an indicator which equals one after 1960 Initial Education is average years of schooling in the workforce in 1960. Initial Physical Capital is log capital per worker in 1960. Post is an indicator that equals one after 1960. Northern States are states in the US North. Southern States are states in the US South. Western States are states in the US West. Rich states are states with above median income in 1960. Poor states are states with below median income in 1960. Constants are not reported. Standard errors are robust clustered at the state level.

*** p<0.01, ** p<0.05, * p<0.1.

Table 7: Sensitivity Tests and Sample Splits

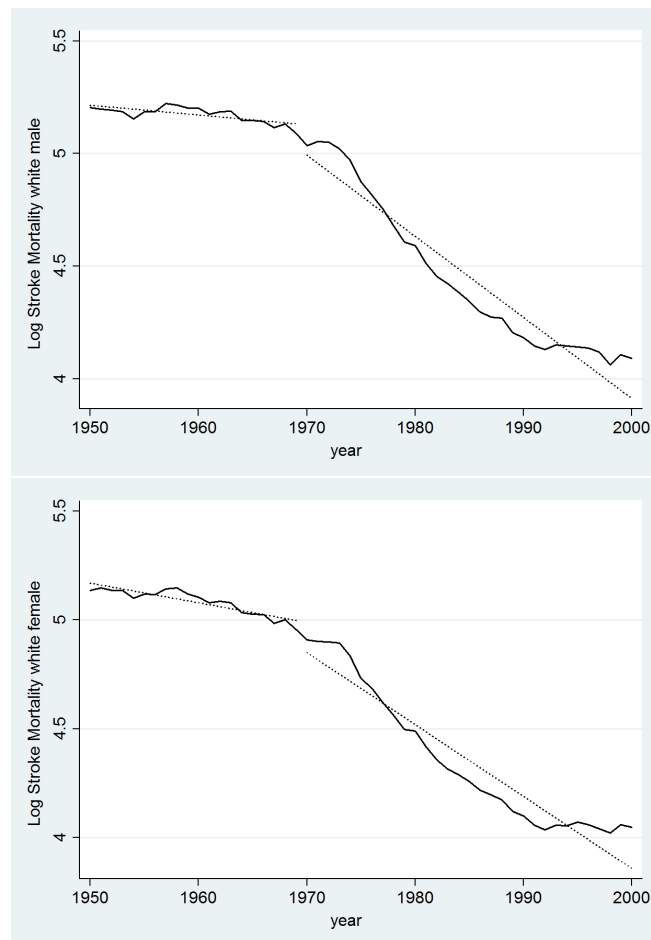
	Dependent Variable is College enrollment II							
	Additional Controls:				Sample Splits:			
	(1)	(2)	(3)	(5)	(6)	(7)	(8)	(9)
Life Expectancy at age 30	0.0613*** (0.0204)	0.0818*** (0.0280)	0.0892*** (0.0250)	0.0516*** (0.0181)	0.0655* (0.0384)	-0.0979 (0.117)	0.112* (0.0612)	0.0362 (0.0398)
Controls (· Post):								
Initial Mortality	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Enrollment	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Income	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Initial Education	Yes	No	Yes	No	No	No	No	No
Initial Physical Capital	No	Yes	Yes	No	No	No	No	No
Log income, age 30 plus	No	No	Yes	No	No	No	No	No
First-stage F-statistics	9.12	6.86	8.73	13.38	8.78	1.52	1.33	4.64
Anderson-Rubin [p-value]	[0.0004]	[0.0004]	[0.0000]	[0.0060]	[0.0106]	[0.2903]	[0.0746]	[0.2120]
# Observations	329	329	281	144	111	74	159	170
# States	48	48	48	21	16	11	23	25

Notes: The table reports two stage least squares estimates, weighted by the white population size age 18-24 1960, using the instrument CVD \times Post. The observation years are 1940-2000. All regressions include state and year fixed effects. The dependent variables are college enrollment II. CVD is the age-adjusted number of deaths from cardiovascular diseases per 100 white population in 1960. Initial mortality is the number of deaths from all other causes than CVD per 100 white population in 1960. Initial enrollment is the variable college enrollment rate I measured in the year 1960. Initial income is the variable log income, age 30 plus measured in the year 1960. Post is an indicator which equals one after 1960 Initial Education is average years of schooling in the workforce in 1960. Initial Physical Capital is log capital per worker in 1960. Post is an indicator that equals one after 1960. Northern States are states in the US North. Southern States are states in the US South. Western States are states in the US West. Rich states are states with above median income in 1960. Poor states are states with below median income in 1960. Constants are not reported. Standard errors are robust clustered at the state level.

*** p<0.01, ** p<0.05, * p<0.1.

Appendix

Figure A1: Annual Development of US CVD-mortality 1950–2000 (Stroke Mortality by Sex)



Notes: The top (lower) figure shows the annual development in the log age-adjusted mortality rate from Strokes for white males (females) in the US. Data source: National Heart, Lung, and Blood Institute.