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HISTORICAL ORIGINS OF A MAJOR KILLER: CARDIOVASCULAR DISEASE IN THE AMERICAN SOUTH

Richard H. Steckel
Garrett Senney

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Historical Origins of a Major Killer: Cardiovascular Disease in the American South
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ABSTRACT

When building major organs the fetus responds to signals via the placenta that forecast post-natal nutrition. A mismatch between expectations and reality creates physiological stress and elevates several noninfectious chronic diseases. Applying this concept, we investigate the historical origins of cardiovascular disease (CVD) in the American South using rapid income growth from 1950 to 1980 as a proxy for socioeconomic forces that created unbalanced physical growth among southern children born after WWII. Using state-level data on income growth, smoking, obesity and education, we explain over 70% of the variance in current CVD mortality rates across the country.

Richard H. Steckel
Department of Economics
Ohio State University
410 Arps Hall, 1945 North High Street
Columbus, OH 43210-1172
and NBER
steckel.1@osu.edu

Garrett Senney
Economics Department
Ohio State University
1945 N. High St.
Columbus, OH
senney.3@osu.edu

*

I. Introduction

The American South has long had poor health outcomes, but satisfying explanations of the disparity remain elusive (Pickle 1996, Lynch, Harper et al. 2004). As can be seen from Table 1, eight of the top ten states with the highest average age-adjusted, all-causes mortality rate from 1999 to 2010 are located in the South; a result that also holds for heart disease, the nation's leading cause of death. The regional pattern of cardiovascular disease (CVD) mortality is even more striking when viewed on the map (Figure 1).

Some argue that the region's poor health follows from concurrent conditions such as low education, lack of exercise, a poor diet, and so forth. While not denying the relevance of these factors, the Developmental Origins of Health and Disease hypothesis (DOHaD), also called the fetal origins hypothesis, as proposed by Barker, Thornburg and others is gaining favor (Barker 1995, Barker 2002, Gluckman, Hanson et al. 2008, Barker and Thornburg 2013).

This approach to understanding chronic adult health conditions has attracted the interest of economists but they have not applied the concept to understand the outlier of cardiovascular disease (CVD) in the American South. A recent summary of research observes that the fetal origins hypothesis has survived scrutiny by economists (Almond and Curri 2011). Development economists have been active in exploring the fetal origins hypothesis, as discussed by (Schultz and Strauss 2008). This type of work also involves economic history (Fogel and Costa 1997, Bleakley 2007); environmental economics (Deschenes, Greenstone et al. 2009); and family economics (Del Bono and Ermisch 2009, Del Bono, Ermisch et al. 2012), We foresee a large potential for these ideas in economic history.

[Insert Table 1]

[Insert Figure 1]

According to the hypothesis, the developing fetus responds to signals about current and past intergenerational nutritional states when building organs and other biological structures. Poor nutrition and stress compromises organ integrity and degrade processes that regulate physiological systems in later life (Gluckman, Hanson et al. 2008, Barker and Thornburg 2013),

making them vulnerable for diseases as adults. This process is known as programming. Evidence shows that individuals are predisposed to cardiovascular disease (CVD) if the heart, vascular tree, kidneys and pancreas are modified in the womb in response to maternal social stress and poor nutrition. If people rendered vulnerable then face a second hit in the form of an energy rich diet or stress in later life, pathophysiological processes are set into motion that lead to CVD.

In this view, intergenerational transitions from persistent poverty to prosperity may promote chronic non-infectious diseases in adults. Here we employ developmental origins concepts to argue that the CVD mortality is elevated in the South by rapid economic growth that began in the 1950s, which in turn created unbalanced physical growth in many children, or a clash between the anticipated and the realized environments of the developing child. Our proxy for rapid socioeconomic change is median household income at the state level. The discussion section elaborates on mechanisms by which this led to increased mortality rates from CVD.

While being the nation's leading cause of death, CVD is a very costly health problem that absorbs nearly $450 billion annually in health care expenditures.[1] Heart disease and stroke can lead to serious illness, disability, and a significant decline in the quality of life. More than three million Americans report some impairment caused by CVD, making it the leading cause of disability in the U.S. Nearly one-quarter of all deaths in the U.S. stemmed from heart disease in 2010 (Heron 2010). Since individuals with ongoing cardiovascular conditions (like coronary heart disease, angina, or atherosclerosis) are more than twice as likely to have a stroke than those who do not, one could plausibly add stroke deaths, which would make the total approximately 30 percent.[2] Diagnosis is further complicated by the fact that diabetes (about 2.8 percent of deaths) contributes to arthrosclerosis, and hypertension is a source of kidney failure (about 2 percent of deaths). People with diabetes also tend to develop heart disease or have strokes at an earlier age than other people. The chances of a heart attack for a middle-aged individual with type 2 diabetes are as high as someone without diabetes who has already had one heart attack.[3] With

[1] Refer to http://www.cdc.gov/chronicdisease/resources/publications/aag/pdf/2011/heart-disease-and-stroke-aag-2011.pdf

[2] Refer to http://www.strokeassociation.org/STROKEORG/LifeAfterStroke/HealthyLivingAfterStroke/UnderstandingRiskyConditions/How-Cardiovascular-Stroke-Risks-Relate_UCM_310369_Article.jsp#

[3] Refer to http://diabetes.niddk.nih.gov/dm/pubs/stroke/#connection

this perspective, perhaps one third of all deaths in the U.S. may be reasonably attributed to CVD and its associated conditions.

[Insert Figure 2]

Age differences in CVD are large while gender and racial differences are rather small (Heron 2010). Figure 2 shows the gradient of death rates by age in 2009. The death rate rises sharply beyond middle age, increasing from 12.3 percent at ages 25-44, to 21.2 percent at ages 45-64 and to 26.5 percent at ages 65+.

As can be seen in Figure 3, the difference in the share of deaths from heart disease varies little by gender (24.9 percent for men versus 23.5 percent for women). The racial differences are also small, illustrated in Figure 4, although Hispanics are somewhat lower (20.8 percent) compared to non-Hispanic whites (24.6 percent) and non-Hispanic blacks (24.1 percent). With these facts in hand, here we restrict our focus to mortality patterns of whites by age but in future research we plan to investigate racial differences in CVD deaths.

[Insert Figures 3 & 4]

Researchers have identified numerous factors associated with CVD, including lifestyle; family history; and genetics, all of which ultimately operate through molecular mechanisms. Genetic and family-history research in the area has mushroomed in the past decade, and while researchers can point to progress, the information acquired explains relatively little of the variation in deaths across individuals and populations (Dandona, Stewart et al. 2010, Prins, Lagou et al. 2012, Roberts and Stewart 2012, Swerdlow, Holmes et al. 2012). Lifestyle conditions have been the most intensively studied factors such that a standard profile of risks has emerged, encompassing obesity, smoking, physical inactivity, a high-fat diet, and psychological stress (De Backer 2008, Watson and Preedy 2013, Graham 2014). Also implicated are uncontrolled hypertension and diabetes as well as low education and poor access to medical care. It is also worth noting, however, that one-half of individuals with CVD lack any of the conventional risk factors (Futterman and Lemberg 1998), and therefore much remains to be explained concerning the origins of this disease.

A growing literature supports the importance of the environments in utero and in early life on adult health, going back at least to the work of David Barker and associates (Barker and Osmond 1986, Barker 1990, Barker 1995, Barker and Thornburg 2013). DOHaD connects non-harmonious growth trajectories in early-life with CVD and other chronic adult illnesses. Such a trajectory is triggered when severe biological stress slows development and maturation in utero in anticipation of continuing stress following birth. If the poorly nourished fetus emerges to good nutritional surroundings; however, its body is maladapted to this unexpected, lush environment. Mounting evidence indicates that a range of maternal experiences can deliver cues that may be transmitted across generations to influence patterns of health and disease (Stöger 2008, Kuzawa and Sweet 2009, Thayer and Kuzawa 2011). In some cases, experiences of deprivation in one generation can be transmitted not only to offspring but to further generations (Kuzawa and Sweet 2009, Heron 2010, Thayer and Kuzawa 2011). Numerous research projects have extended this work, including (McEniry and Palloni 2010) who study a representative sample of Puerto Ricans aged 60-74. They find that after controlling for standard risk factors the probability of heart disease was 65 percent higher among individuals who were born during seasons in which the incidence of disease and poor nutrition were higher.

Finding a suitable socioeconomic proxy for non-harmonious growth (i.e. contrasting pre and post infant environmental conditions) is a challenge for this line of research. The economic history of the South, however, creates a fortunate opportunity for study. The region was poor for decades following the Civil War, but grew faster than the national average following WWII (see Figure 5).

[Insert Figure 5]

Our approach follows the methodology of papers showing that the states with higher diabetes and hypertension prevalence in 2010 correlate well with the states that had the greatest median household income growth between 1950 and 1980 (Steckel 2013, Senney, Steckel et al. 2015). If the developmental origins hypothesis has merit then the ratio of median household income in 1980 to that in 1950 should be a useful proxy for non-harmonious physical growth.

Heart disease and type 2 diabetes are of course different illnesses, but are nevertheless related; some 67% of subjects with type 2 diabetes die of heart disease (Grundy, Benjamin et al. 1999). Under the developmental origins hypothesis their causes originate from the same non-harmonious growth in early life. It is, therefore, important to determine whether mortality from

CVD responds to intergenerational changes in economic conditions in a similar way to that of type 2 diabetes. Consistent with co-morbidity, the geographic distribution of prevalence rates at the state or county level are similar for these diseases.[4]

In an effort to design remedies for heart disease, numerous studies have investigated antecedents or causes, leading to recommendations on diet, exercise, abstinence from smoking, weight control, careful monitoring of blood pressure, and so forth. While beneficial, such research does not harness intergenerational information that would be quite useful if guided by developmental origins concepts. Heart attacks and strokes often appear after CVD is well-advanced, but this problem could be lessened by knowledge of proclivity based on socioeconomic information that many patients could readily provide, such as occupations of the parents and grandparents and their counties of birth and residence in adolescence.

The DOHaD model views the fetus as an organic system that optimizes itself for the external world that it expects to inhabit; however, the only meaningful signals the developing child receives come from the mother via the placenta and from the external environment after birth. While it is unsurprising that transitioning from a good to a poor nutritional state has negative health consequences, recent literature identifies negative effects that follow a transition from a poor to a good nutritional state (Barker 2002, Hanson and Gluckman 2008, Barker and Thornburg 2013). Research suggests that individuals with organs (e.g., kidneys and liver) optimized in utero for survival in lean conditions are more likely to suffer from CVD if they consume a rich diet later in life.

Given the background studies behind the DOHaD hypothesis, intergenerational poverty followed by rapid socioeconomic improvement should elevate the risk of mortality from CVD among adults who experienced the improvement at an age beyond which further biological adaptation was not possible. Thus, we expect that the penalties from unbalanced growth should increase with age as the body has less ability to adapt to its changing environment. This prediction arises from the medical concept of developmental plasticity— a younger and developing individual responds to signals from its early environment. This developmental adjusting will actually heighten the risk of chronic adult diseases if the induced bodily changes

[4] Refer to http://apps.nccd.cdc.gov/brfss/index.asp

are suboptimal for the realized environment later in life (Bateson, Barker et al. 2004, Gluckman and Hanson 2007, Burdge and Lillycrop 2010).

II. Data

We use detailed data of the underlying cause of death from the CDC's Wide-ranging Online Data for Epidemiologic Research (WONDER) to calculate the state level mortality by age cohort. The mortality rate is measured for the years of 2010-2011 and includes deaths in which the recorded underlying cause falls within the ICD I00-I99 categories (diseases of the circulatory system which includes disorders like hypertensive diseases, cerebrovascular diseases, and pulmonary heart disease). We use crude rates of per 100,000 for individuals in the age groups of 55-64, 65-74, 75-84, and 55-84; the individuals in these age cohorts were born between 1926 and 1955. Table 2 displays the summary statistics for the CVD mortality rates in 2010 by age cohort.

[Insert Table 2]

We also use state level data on average levels of obesity, smoking, and education from the CDC's Behavioral Risk Factor Surveillance System (BRFSS) in 2010. The BRFSS is a cross-sectional telephone survey conducted by state health departments with technical assistance from the CDC. The respondents are asked to self-report basic demographics (race, gender, height, and weight) as well other health related conditions. The BRFSS staff then calculates each respondent's body mass index, which is coded as obese if the BMI is greater than or equal to 30. Respondents also report their highest grade or year of school completed.

In our analysis we use the share of the state population that has a high school diploma, GED, or less. The Smoking variable is the share of state's population who self-report being currently smokers who have smoked at least once in the previous week.

Median household income is taken directly from the U.S. Census of Population for 1950 and 1980. The nominal income was adjusted using the Federal Reserve Bank of Minneapolis' Chained-weighted CPI with 1982-1984 as the base years. The growth rate is found by dividing median household income in 1980 by median household income in 1950. Table 3 provides summary statistics of the independent variables used in this analysis.

[Insert Table 3]

We recognize that interstate migration can distort the empirical analysis, and we contend with the problem by using weighted regressions employing different measures of population turnover within the state. Conditions in 2010, the year mortality and other variables such as obesity are measured, create a chronological disconnect between the income ratio and measured effects. Data on domestic in-migration and out-migration are based on a subsample of the U.S. Census.[5] We define turnover as the sum of in-migration and out-migration for a state, which is used to measure chronological change in the state population. A larger turnover value implies the actual composition of individuals residing in the state have changed dramatically over time, which could potentially be masked by simply using net migration numbers. For example, if the entire state population departed, but was replaced by the exact same number of people arriving, the net migration for the state would be zero. Average turnover from 1950 to 1980 is simply the average of the turnover measure over the four census years.

This measure does not recognize the origins of the new residents; therefore, we also collect data from the U.S. Census on the proportion of adult residents in 2010 who were born in southern states. While this measure does not capture more general migration, it better represents the mixture of individuals with high probabilities of arriving from high growth income areas (i.e. the South) who were vulnerable to CVD. As a robustness check we estimate our model using the share of the population born in the South as a weight.

III. Methods

We evaluate the hypothesis that rapidly improving socioeconomic conditions preceded by intergenerational poverty causes a higher likelihood of CVD among offspring. The model we estimate is:

$$(1) \qquad MR_i = \beta_0 + \beta_1 HI_i + \theta X_i + \varepsilon_i$$

where MR_i is the CVD mortality rate of whites in state i in 2010-2011, HI_i is the ratio of median household income of whites in 1980 to that in 1950, and X_i is a set of covariates that control for

[5] The 1950 Census compared 1949 state of residence to 1950 state of residence, well every census year after that compares state of residence 5 years prior as to their current state.

other past and current conditions. Risk factors for mortality include low levels of education, smoking, and obesity.[6] These variables are measurable at the state level and are included as the controls in estimating the effect of income change. Under the proposed hypothesis the coefficient β_1 should be positive, large, and statistically significant.

Population turnover is relevant because current mortality rates are hypothesized to be a function of conditions that existed from 1950 to 1980. It would be ideal if there was no population turnover from 1950 to 2010, such that the population under study was constant, or at least undisturbed by people moving in or out of the state. Of course that is not true, and the amount of turnover varies across states and must be considered in using state-level data. The issues at hand are how to incorporate turnover into the analysis, and whether there was enough stability in state populations to create a systematic relationship between past conditions and current outcomes. If migration heavily contaminated the relationship and the error term was large, one would expect to find a low R^2 and coefficients that were statistically insignificant. Or worse, a statistical outcome that contradicted well-founded results, such as smoking and obesity were beneficial for CVD. Plausible outcomes for well-researched variables would lend credence to the measured impact of past income change on mortality rates. We use a weighting scheme that reduces the importance of high-turnover states in the analysis.

IV. Results

Figure 6 presents a scatter diagram of a statistically significant relationship of the mortality rate at ages 75-84 on the income ratio, in which a linear regression line is drawn as a point of reference. The scatter diagrams are similar using the other dependent variables. First, as expected, most of the states on the right hand side of the graph (largest median income growth) are located in the South. Second, the slope with respect to the income ratio is 117.33 (std. err. 44.63), which implies that a two unit increase in the income ratio would explain more than one standard deviation of the mortality rate across the states.

[Insert Figure 4]

[6] Refer to Grossman & Kaestner (1997) and http://www.cdc.gov/obesity/data/adult.html.

Several interesting outliers suggest that an expansion of the model would be useful. In particular the four largest outliers above the regression line (Arkansas, Oklahoma, West Virginia, and Mississippi) and the four largest below the line (Minnesota, Colorado, Arizona and Wyoming) all have above (or below) average characteristics linked to CVD in earlier studies. In particular, the four positive outliers have above average values of smoking, obesity, and years of education at high school or less, while all those below the line have below average values of these variables. Notably, Arkansas is third lowest and West Virginia the lowest among all states on the scale of education (57.1 and 64.5 per cent, respectively). Oklahoma, Louisiana, Arkansas, Alabama, and Mississippi have the second through sixth highest levels of smoking. It emerges that much of the scatter around the regression line may be explained by behaviors adversely linked to CVD.

[Insert table 4]

Controlling for current conditions, like smoking, obesity, and educational attainment, we regress mortality rates on the ratio of median household income Table 4 displays the results. To control for the concerns over population turnover we perform robustness checks by rerunning the model using the inverse of average turnover 1950 to 1980 and average turnover 1950 to 2000. In practice our concern about turnover may have been well founded, but the empirical results are affected little by the weighting, using the inverse of average turnover weighting scheme reveals similar patterns of coefficient sizes and statistical significance.[7] To further account for the effects of southern birth, Tables 5 displays the results of the same regression specification except using percent of the 2010 population born in the South as a weight. If the DOHD hypothesis is correct, then the southern-born should have been vulnerable to CVD regardless of where they lived as adults. As expected the income variable is both statistically and economically significant in the presence of the control variables. This is our preferred specification.

[Insert table 5]

[7] Regression results are available from the authors by request.

One might think that the initial level of income would affect the relationship; however, including median household income in 1950 does not qualitatively change the results, with the variable being statistically insignificant. Potentially the relationship of the income ratio to mortality is nonlinear but the coefficient of a squared term is insignificant, the other coefficients are less significant, and the adjusted R^2 is lower. This suggests that the variable is irrelevant to the equation. Further, the results are essentially unaffected by using a log functional form.

The coefficient on the education control has the expected sign. More people with less education implies that the average resident of the state is less informed about the importance of regular health maintenance, less knowledge of resources to assist in obtaining healthcare, and potentially less able to understand the medical advice received. This lack of information will generally be associated with an increase in the mortality rate. Studies show that obesity is also a factor contributing to higher levels of mortality from CVD (Faeh, Braun et al. 2011, Zheng, Tumin et al. 2013). It turns out that smoking and obesity are substantially correlated ($r = 0.638$), and so it is difficult to obtain precise estimates of their independent effects on mortality. However, the variables are jointly significant in this analysis.

V. Discussion

It is important to recognize limitations of the statistical analysis, first in linking cohorts born near the middle of the century with economic change at the state level from 1950 to 1980. An improvement would be to link the income growth of annual birth cohorts with CVD deaths in the same birth cohorts; however, this option is unavailable with the data at hand. The approximation employed here adds noise to the relationship, which diminishes the precision of the estimates. Nevertheless the coefficients of interest are both statistically and economically significant, adding credibility to the DOHD hypothesis.

States are heterogeneous in all the variables employed, and so it might be desirable to use smaller geographic units of analysis such as counties that are more homogeneous. Units as small as counties, however, often have very high rates of population turnover (above those of states) that complicate the links between income in the past with that of heart disease mortality in the present. States are not free of these problems, but they have fewer issues in this regard. Despite the shortcomings noted here, a state-level analysis explains over 70 percent of the variance in CVD mortality.

The coefficients on the ratio of median household income in 1980 to that in 1950 are all positive, and economically and statistically significant. Considering the three mutually exclusive groups (55-64, 65-74, 75-84), the magnitude of the effect of a rise in household income increases as the groups climb in age. The 55-64 year old cohort would have been under 4 years old in 1950 and between 25 and 34 years old in 1980. The 75-84 year old cohort would have been between 15 and 24 years old in 1950 and between 45 and 54 years old in 1980. This result suggests that if the income growth occurs early enough in life, while the individual is still developing, the body is better able to adjust to the changing nutritional state. The individuals who were older when the income growth occurred were less able to physiological adapt and thus they were the most adversely affected by the income growth (Bateson, Barker et al. 2004, Kuzawa 2005) .

Our analysis agrees with many studies that show smoking, obesity and low levels of education are risk factors for CVD. This is the case despite known problems of inference about individual behavior from regressions based on aggregate data.[8] We should ask, however, whether the ecological fallacy distorts the interpretation of relationships behind past conditions on current outcomes. First, we are not trying to infer individual behavior from aggregate data on smoking, obesity and education. These relationships are already well-established at the individual level. We include these variables in the regressions to control for distortions they may impose on the income-mortality relationship. Second, examples of the fallacy contemplate distortions created by contemporaneous feed-back loops, such as immigrants who often have low levels of education, deciding where to settle based on the quality of educational opportunities. In our case, there is no such feed-back loop, or if there was it would operate with a very long lag. We argue that income change from 1950 to 1980 affected mortality rates a half a century or more later, and it is hard to imagine how mortality rates could affect past income change. Individual death from CVD is not subject to decision in the same way as immigrants choosing a destination. A devil's advocate might argue that older individuals with CVD chose to live in states that had high levels of past income change, but the mechanism is unclear. This might mean that CVD sufferers who lived in low-mortality states migrated to the South, perhaps for the availability of good medical care boosted by past income growth; we find that implausible.

[8] The ecological fallacy is illustrated by a famous study on immigration and illiteracy by Robinson (1950).

The coefficients on the obesity rate in 2010 are positive for all groups and statistically significant for all age groups except for the 65-74 cohort. Other research has shown that carrying elevated amounts of body fat is associated with higher levels of mortality(Faeh, Braun et al. 2011, Zheng, Tumin et al. 2013). Furthermore, we find evidence that the obesity-mortality relationship becomes stronger with age. This result coincides with recent advancements in the medical literature (Masters, Reither et al. 2013a, Masters, Powers et al. 2013b)

The coefficient on smoking in 2011 is positive for all age cohorts, but only statistically significant for the younger two cohorts. The overall mortality among smokers in the United States is about three times higher than that among similar people who never smoked.[9] Our results are well in line with this established literature. As a robustness check, we collected data on the percentage of people in the state who smoked at least 100 cigarettes lifetime and self-reports as currently smoking.[10] The reported regressions were all rerun using this new measure of smoking and the results are qualitatively the same. The coefficients and goodness of fit hardly change with this adjustment.

VI. Robustness Checks

Inequality within states might affect the measured income-mortality relationship to the extent that income growth generated rising inequality, a pattern called the Kuznets curve, found within many countries during industrialization (Fields 2001). The expected net effect of changes in inequality is unclear. If the poor benefited relatively more from growth, then average income growth would understate their improving conditions and therefore their susceptibility to CVD. To examine the effect of inequality of income growth, we calculate the standard deviation of the income distribution in 1950 and 1980. The ratio of standard deviation of income 1980 to 1950 measures how much more varied income is in 1980 as compared to in 1950. The coefficients are positives, but insignificant for all age cohorts and inclusion does not qualitatively affect the other coefficients or goodness of fit of the regression.[11]

[9] CDC's "The Health Consequences of Smoking—50 Years of Progress. A Report of the Surgeon General" (2014)

[10] The data comes from the 2014 America's Health Rankings published by the United Health Foundation, American Public Health Association, and Partnership for Prevention.

[11] Results from these regressions are available from the author upon request.

One of the factors behind the rapid household income growth from 1950 to 1980 is the increase in female participation in the labor force. The state with the smallest increase in the female labor force participation rate still had over 44% more women in the force labor, while the average growth is 75%. It is possible that the income growth variable is also capturing some of the effect from mothers spending more time working outside the home. To explore this factor, we add the ratio of female labor force participation rate 1980 to 1950 to the main specification. The coefficient is positive but insignificant for all age cohorts. The inclusion of this variable only slightly reduces the coefficient on ratio median income and the adjusted R^2 are about the same.

Another factor to consider is the compositional shift that occurred in the sectors of employment from the 1950 to 1980. Individuals were moving away from employment in the agricultural sector to the manufacturing and service sectors. We rerun the main regression including the ratio of the share of non-agriculture employment 1980 to 1950. The coefficients are positive but insignificant. The inclusion of this variable only slightly reduces the coefficient on ratio median income and the adjusted R^2 are about the same.

Table 6 shows the regression results from rerunning the specifications from Table 5, but this time adding the variables for the ratio of standard deviation of income, the ratio of female labor force participation, and the ratio of the share of non-agriculture employment. The inclusion of all three of these variables reduces the magnitude of the effect of the income ratio and slightly increases the goodness of fit of the regression for the younger and older cohort. This further supports that the ratio of median household income is a proxy for a whole bundle of changes that occur and were allow and supported by income growth. The following section will briefly discuss the possible mechanisms.

[Insert table 6]

VII. Possible Mechanisms

What processes might have translated rapid income growth in the South into adolescent weight gain, and eventually CVD? What distinctive features of the South led to this outcome? One suspects that the reality could be quite complex but it is worth speculating to guide future research initiatives. Of course, higher incomes alone enabled families to purchase more food, an item that would have been high on the list of priorities for southern families, which were

especially poor. This relationship is enshrined in economics as Engle's Law, named after a nineteenth century statistician who observed that the poorer the family the greater the outlay of income on food. He claimed that the proportion of income spent on food is a good measure of the standard of living of a population, and numerous modern studies substantiate this conclusion (Anker 2011).

Economic historians know that rapid economic change creates many new opportunities but also disrupts family life, as studies of industrialization make clear (Tilly and Scott 1978, Hareven 1982). As southern agriculture mechanized and food became cheaper, farm women joined the labor force, often taking jobs in food processing plants, the service sector, and government installations (McMillen 1989). To realize these opportunities families may have relocated and members may have acquired new skills, adopted new commuting patterns and so forth, all of which were stressful. Some people find that food allows them to cope with stress, and they eat more and gain weight (Torres and Nowson 2007).

Mothers who were not employed outside the home often prepared meals for their families. By joining the labor force they had less time for home production of meals and less opportunity to supervise the eating habits of their children. They may have used their earnings to purchase more processed food, which often has lower nutritional quality (Devine, Jastran et al. 2006). Another outcome is that children who once had to work at manual jobs to help support the family were released by higher incomes from this work, adding to net nutrition and thereby contributing to weight gain (Basu and Van 1998).

Future research should consider the possible role of southern culture and the interaction of diet and traditional attitudes towards rest and physical exercise. Unlike other regions, agriculture was the dominant employer in the South prior to the beginning of industrialization after the middle of the 20th century. Relative to other regions, southern farmers were slow to adopt the tractor, and mules lingered on small farms operated by older farmers until the 1950s (Ellenberg 2007). Mechanization of the harvest was difficult to accomplish in its most important crops of cotton and tobacco, and relief from field labor came late relative to other regions (Hurt 1989).

Mechanical cotton pickers largely replaced hand labor between the late 1940s and the 1960s but hand methods persisted on small farms for a decade or more (Heinicke and Grove 2008, Logan 2012). Southern customs were fashioned by a long history of physical labor in the fields that welcomed rest at the end of the work day and that traditionally discouraged work on Sunday.

The South was not a region where habits of recreational exercise and health club memberships readily replaced a decline in caloric expenditure associated with a reduction in physical labor. In 2007 the share of the population belonging to health clubs ranged from a low of 6.3 per cent in West Virginia to a high of 21.8 per cent in Colorado (Active Marketing Group 2007). In every state in the high CVD risk region of the South, the share of the population belonging to a health club was below the national average of 15.5 per cent. There is a strong negative correlation between CVD morality and health club membership (-0.381 to -0.485); however, when the other control variables are included health club membership is insignificant and does not materially affect the results.[12]

Persistence of long-established dietary habits probably contributes to CVD in the South. The food ways of southerners had roots in the nineteenth century, when pioneer farmers planted corn and created swine herds (Taylor 1989). For most of the year the hogs foraged on acorns and other products of the forest and then early in the fall farmers assembled them for fattening on corn. Meat processing occurred after the first cold spell, and an orgy of pork eating followed. Fat was rendered into lard and the hams and shoulders were salted, smoked and stored. As long as pork was available these farmers ate it daily, accompanied by various forms of corn processed into bread, grits or hominy. When available, vegetables were usually fried or boiled with a piece of lard or pork.

According to southern tradition, a boiling vegetable pot was good only if it had enough grease to "wink back" after lifting the lid. Sweet potatoes were also common fare in the diet because they required minimal cultivation and they could be stored for months in underground cellars. By the twentieth century the price of wheat began to decline and new methods of milling and distribution enabled even poor southern farmers to buy flour in bulk to make into biscuits that were eaten with syrup or red-eye gravy. Furthermore, southern states have the highest proportions of adults who self-report consuming one or less fruit and one or less vegetable in any form per day.[13] In this environment, income growth, the decline of food prices, the reduction of work, and changing roles within the family created the perfect storm generating chronic adult diseases.

[12] Results from these regressions are available from the author upon request.

[13] http://www.cdc.gov/nutrition/downloads/State-Indicator-Report-Fruits-Vegetables-2013.pdf

VIII. Conclusions

According to the mechanism of developmental programming, the placenta acts as if it contains a small "biological econometrician" who forecasts the future environment for the developing fetus and accordingly tailors the organs such as the heart, kidneys, and pancreas to promote survival at least through the age of reproduction. The forecasting model draws upon not only current nutritional experiences of the mother but also those of earlier generations going back to the great grandmother. Although the precise biochemical mechanisms and strength of intergenerational linkages are subject to debate, medical research has established a useful empirical pattern that economists have applied to understand the long-term outcomes of stressful events such as the 1918 flu pandemic and the Great Chinese Famine of 1958-1962.

Our paper makes two important contributions. First, the analysis confirms or is at least consistent with the developmental origins hypothesis as applied to CVD in explaining regional differences in mortality within the United States in 2010. Second, the impact on mortality of rapidly improving intergenerational conditions during the middle of the twentieth century increases with age across the groups 55-64 to 75-84. This suggests that the penalties of unbalanced physical growth increase when the developing body has less ability to adapt to a new environment. Individuals aged 75-84 in 2010 were born from 1926-1935 and were young adults with fixed biological structures, when dramatic social and economic change transformed the South after 1950.

The traditional southern diet was a disaster for heart disease when accompanied by a decline in physical labor and habits that eschewed recreational exercise. The southern diet is gradually changing but fried foods such as chicken, catfish and hushpuppies remain popular to this day. Pockets of strong dietary tradition linger in many rural regions, a pattern that offers an opportunity to study CVD at the county level. A topic untouched by the evidence analyzed here is the consequence of duration of relative poverty and affluence on CVD mortality. One might reasonably hypothesize that for a given increase in income, children of those women having had longer intergenerational experiences of poverty may have had greater susceptibility. Similarly, for a given duration of poverty, children of women having had greater increases in income would also be more susceptible. Individual-level intergenerational evidence is needed to investigate these interesting questions.

The fetal origins hypothesis has especially relevant implications for the developing world, where vast numbers of poor families are on the verge of experiencing significant increases in income. Chronic adult illness such as heart disease are likely to increase dramatically in the once-poor but now rapidly growing countries (Lopez, Mathers et al. 2006, Kinsella and He 2009).

More generally, disasters, important new technologies, and profound changes in government policy create laboratories that present opportunities for study by economic historians using developmental origins concepts. Any dramatic change that unfolds over several months or years has cohort effects on human physical development, and therefore cognitive function (and ultimately human capital) and adult noninfectious disease. To the traditional horsemen of the apocalypse--war, pestilence and famine--we can add other exogenous influences on human function of extreme weather events (droughts and floods), new health technologies (e.g. germ theory of disease and public health infrastructure), deep business cycles, and new varieties of disease. In this light one may ponder, for example, the continuing cost of the American Civil War. Cohorts, especially those born in the South, experienced physiological stress *in utero* and in early childhood. One may ask how these effects unfolded over time and across regions that experienced variable levels of stress. Therefore our contribution is not limited to explaining CVD mortality in the South but extends to a general approach to understanding shocks and history.

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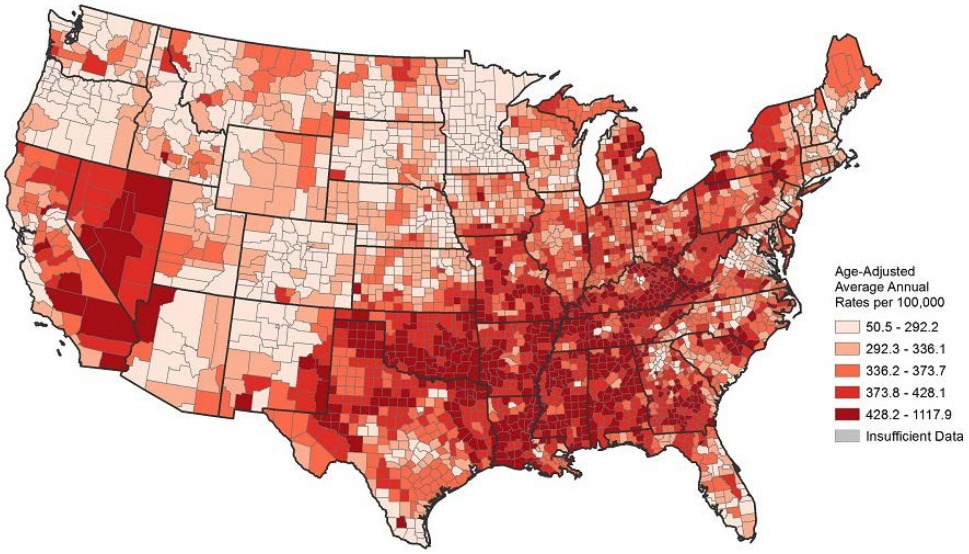

Figure 1: Age Adjusted Heart Disease Death Rate by County, White, All Genders, 35+ for 2011-2013
Note: Source is the CDC/NCHS, National Vital Statistics System.

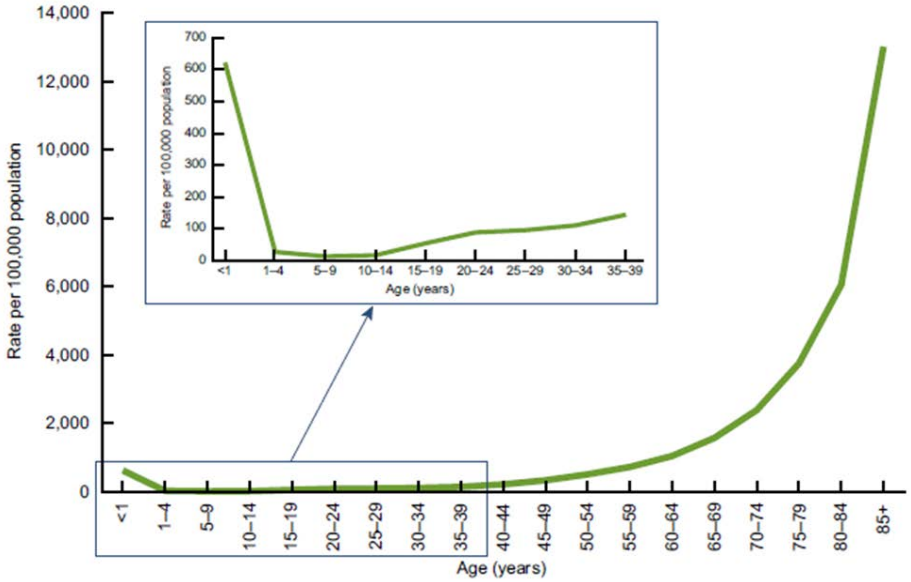

Figure 2: Age-Specific Death Rates from CVD in 2009
Note: Source is the CDC/NCHS, National Vital Statistics System.

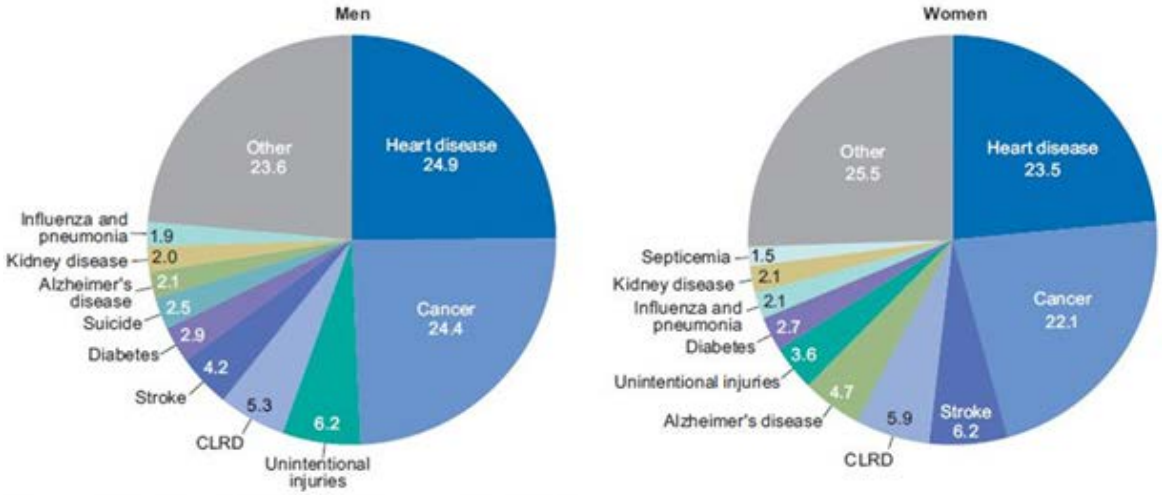

Figure 3: Percent Distribution of the 10 Leading Causes of Death, by Sex: United States 2010
Note: Source is the CDC/NCHS, National Vital Statistics System.

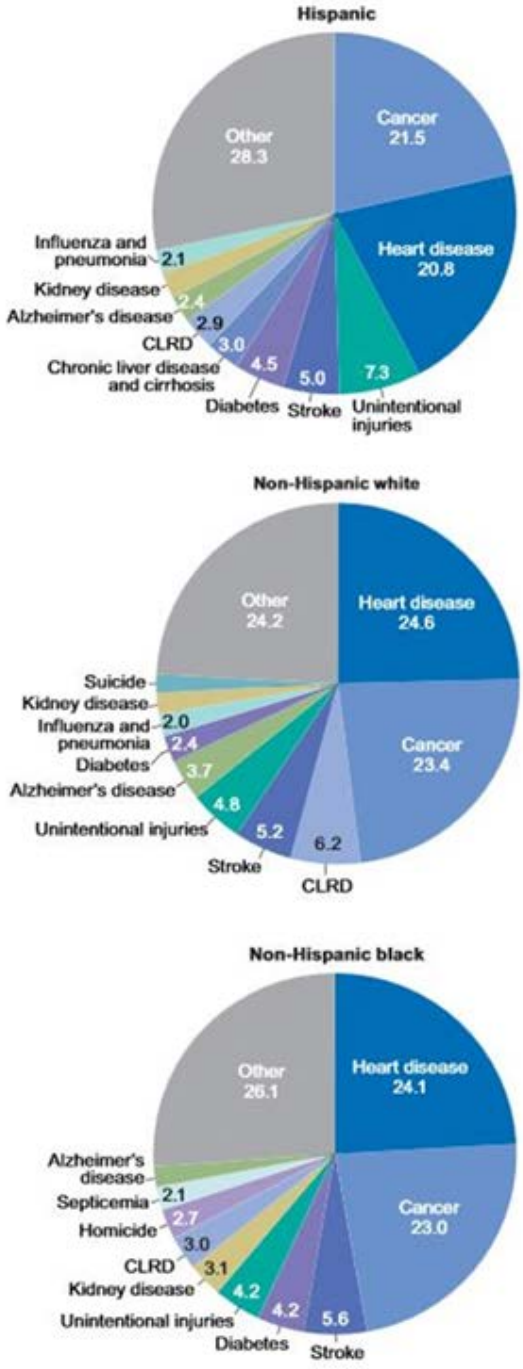

Figure 4: Percent Distribution of the 10 Leading Causes of Death, by Race: United States 2010
Note: Source is the CDC/NCHS, National Vital Statistics System

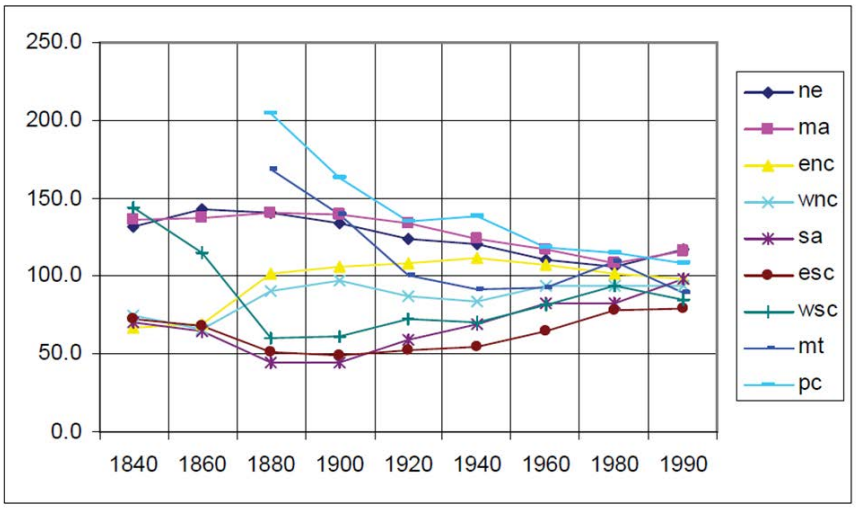

Figure 5: Regional Income per Capita, 1840-1900
Note: Legend: ne = New England; ma = Middle Atlantic; enc = East North Central; wnc = West North Central; sa = South Atlantic; esc = East South Central; wsc = West South Central; mt = Mountain; pc = Pacific. Source: Kim and Margo (2003).

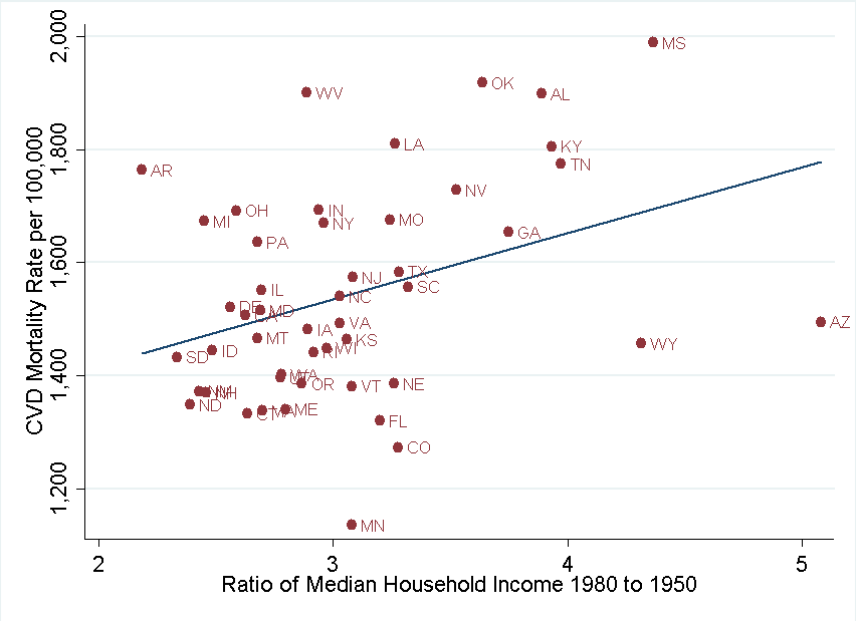

Figure 6: Scatter Plot of Median Household Income and Mortality Rate for the 75-84 Age Cohort
Note: Source is the CDC/NCHS, National Vital Statistics System.

TABLE 1- Mortality Rates for All Causes and Heart Disease

State	All Cause	State	Heart Disease
West Virginia	970.2	Oklahoma	346.8
Kentucky	944.1	Mississippi	345.9
Alabama	940.9	West Virginia	332.2
Mississippi	940.1	Alabama	330.9
Oklahoma	939.3	Kentucky	322.1
Tennessee	909.8	Arkansas	320.0
Arkansas	905.0	Tennessee	316.7
Louisiana	904.3	Louisiana	307.4
Nevada	875.2	Missouri	303.6
Georgia	862.3	Indiana	295.1

Notes: Age-Adjusted All Cause and Heart Disease Mortality Rates per 100,000 in 2010 for Whites.
Source: Centers for Disease Control and Prevention and National Center for Health Statistic.

TABLE 2- Descriptive Statistics of Dependent Variables

	55-64	65-74	75-84
Max	330.1	696	1,989.20
Mean	212.5	499.99	1,538.10
Median	201.9	472.1	1,499.50
Min	130.2	341.4	1,134.90
Std Dev	51.7	91.35	193.1
N	48	48	48

Source: Centers for Disease Control and Prevention and National Center for Health Statistic.

TABLE 3- Descriptive Statistics of Independent Variables

	Obesity	Smoking	rMedian	HS or Less
Max	32.4	26.3	5.08	64.5
Mean	26.52	17.81	3.06	46.36
Median	26.75	17	2.95	45.95
Min	18.9	9.2	2.18	33.4
Std Dev	2.96	3.29	0.58	6.38
N	48	48	48	48

Source: Centers for Disease Control and Prevention and Behavior Risk Factor Surveillance System

TABLE 4- Impact of Income Growth on CVD Mortality

	55-64	**65-74**	**75-84**
rMedian	29.58***	37.46***	95.99***
	(7.92)	(12.47)	(30.24)
HS or Less	3.65***	7.95***	15.12***
	(0.88)	(1.38)	(3.35)
Smoking	2.98	4.10	2.21
	(1.88)	(2.95)	(7.16)
Obesity	3.30	6.74**	17.63**
	(2.12)	(3.34)	(8.09)
Constant	-187.94***	-235.11***	39.48
	(47.99)	(75.56)	(183.29)
N	48	48	48
Adj R^2	0.6528	0.7243	0.6246

Notes: Regression results by age cohort, standard errors in parentheses, using OLS.
Source: CDC WONDER and CDC BRFSS.
*** Significant at the 1% level. ** Significant at the 5% level. * Significant at the 10% level.

TABLE 5- Weighted Regression of the Impact of Income Growth on CVD Mortality

	55-64	**65-74**	**75-84**
rMedian	28.40***	41.63***	105.24***
	(7.30)	(11.24)	(27.77)
HS or Less	3.39***	7.50***	13.70***
	(0.84)	(1.30)	(3.20)
Smoking	4.06**	4.88*	5.78
	(1.86)	(2.86)	(7.06)
Obesity	4.00*	9.04***	21.34**
	(2.12)	(3.27)	(8.07)
Constant	-204.56***	-297.14***	-72.35
	(45.05)	(69.39)	171.50
N	48	48	48
Adj R^2	0.7286	0.7973	0.6815

Notes Regression results by age cohort, standard errors in parentheses, weighted OLS using percentage of 2010 population that was born in the South.
Source: CDC WONDER and CDC BRFSS.
*** Significant at the 1% level. ** Significant at the 5% level. * Significant at the 10% level

TABLE 6- Weighted Regression of the Impact of Income Growth on CVD Mortality

	55-64	**65-74**	**75-84**
rMedian	21.68***	36.85***	100.42***
	(7.50)	(12.19)	(30.97)
HS or Less	3.48**	7.03	13.91***
	(1.08)	(1.76)	(4.47)
Smoking	4.12**	4.82	5.98
	(1.89)	(3.08)	(7.81)
Obesity	2.76	8.15**	20.48**
	(2.12)	(3.45)	(8.77)
HCMember	-1.17	-0.59	-1.31
	(1.80)	(2.93)	(7.45)
rStdDev	31.25	16.10	36.13
	(26.55)	(43.18)	(109.69)
rShareNonAgr	54.05	23.72	40.90
	(40.54)	(65.93)	(167.49)
rFemale LFPR	22.74	41.89	-1.26061
	(27.71)	(45.08)	(114.51)
Cons	-347.84***	-366.58**	-193.60
	(85.19)	(138.55)	(351.99)
N	48	48	48
Adj R^2	0.7675	0.7897	0.7341

Notes Regression results by age cohort, standard errors in parentheses, weighted OLS using percentage of 2010 population that was born in the South.
Source: CDC WONDER and CDC BRFSS.
*** Significant at the 1 percent level. ** Significant at the 5 percent level. * Significant at the 10 percent level