Developmental Origins of Cardiovascular Disease: Understanding High Mortality Rates in the American South

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Abstract

Many studies by social scientists view heart disease as the outcome of current or recent conditions such as poverty, smoking, and obesity. Here we apply an alternative approach to understand cardiovascular disease (CVD) death rates, which are elevated for southern whites relative to the rest of the county. In the developmental origins hypothesis CVD vulnerability follows from unbalanced physical development created by poor conditions *in utero* that underbuilds major organs such as the kidneys and the cardiovascular system relative to those needed to process lush nutrition later in life. The South underwent an economic transformation from generations of poverty to rapid economic growth in the post-WWII era, exposing many children born in the 1950s through the 1980s to unbalanced physical development. Here we use state-level data for whites on income growth, smoking, obesity and education to explain variation in CVD death rates in 2010-2011. Our proxy for unbalanced physical growth, the ratio of median household income in 1980 to that in 1950, has a large systematic influence on CVD mortality, an impact that increases dramatically with age. The income ratio combined with smoking, obesity, and education explains two thirds of the variance in CVD mortality across states. Metaphorically, persistent intergenerational poverty loads the gun and rapid income growth pulls the trigger on CVD.

Researchers recognize that the South has long had the nation's poorest health outcomes (Pickle 1996, Lynch, Harper et al. 2004). One can see from Table 1 that 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 the nation's leading cause of death, heart disease. Some may argue the region's poor showing follows from concurrent conditions such as low levels of education, lack of exercise, a poor diet, and so forth. This idea has merit, but past research on diabetes indicates that a neglected hypothesis, called the developmental origins of health and disease, can contribute to understanding the South's health predicament. 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 geographic disparity in cardiovascular disease has been exacerbated 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.

Cardiovascular disease (CVD) is a 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 3 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 2013). 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 this

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

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

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

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

Age differences in CVD are large while gender and racial differences are small (Heron 2013). Figure 1 shows the gradient of death rates by age in 2009. The death rate rises sharply beyond middle age, increasing from 12.3 per cent at ages 25-44, to 21.2 per cent at ages 45-64 and to 26.5 per cent at ages 65+. 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, although Hispanics are somewhat lower (20.8 percent) compared to non-Hispanic whites (24.6 percent) and non-Hispanic blacks (24.1 percent). Therefore we focus on mortality patterns of whites by age but in future research we plan to investigate racial differences in CVD deaths.

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 1995, Barker and Thornburg 2013). The hypothesis on developmental origins of health and disease (DOHaD)) connects non-harmonious growth trajectories in early-life with CVD and other chronic adult illnesses. A non-harmonious growth 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

then its body is maladapted to this unexpected 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 (Stöger 2008, Thayer and Kuzawa 2011). Numerous research projects have extended this work, including McEniry and Palloni (2010) who studied 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 beginning in the 1960s (see Figure 2). Our approach follows the efforts of Steckel (2013), who shows that the states with higher diabetes and hypertension prevalence in 2010 correlate well with the states that had the greatest median per capita income growth between 1950 and 1980. If the developmental origins hypothesis has merit then the ratio of median per capita income in 1980 to that in 1950 could be a useful proxy for non-harmonious 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

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

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 (counties in the U.S. vary widely in their economic conditions).

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 comes as no surprise 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. We expect that the penalties of unbalanced growth increase with age as the body has less ability to adapt to its new environment.

Data

We use detailed data of the underlying cause of death from the CDC's Wide-ranging Online Data for Epidemiologic Research (WONDER) to examine the developmental origins hypothesis. We examine the impact median household income changes have on the white CVD mortality rate in 2010-2011. The mortality rate is measured at the state-level 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 examine crude death rates per 100,000 people in 2010 and 2011 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.

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.

Median household incomes are 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 2 provides summary statistics of the variables used in this analysis.

We recognize that interstate migration can distort the empirical analysis, for which we do not have a fully satisfactory solution. 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.

[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.

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 the South. 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.

Methods

We evaluate the hypothesis that rapidly improving socioeconomic conditions preceded by intergenerational poverty causes a higher likelihood of CVD among offspring. The basis for the hypothesis is found in earlier epidemiological studies such as (Barker 2002). Families who lived in poverty for generations are more likely to have children with organs optimized for survival and reproduction in a life of deprivation. Under the developmental origins hypothesis, if these families suddenly experienced intergenerational prosperity and nutritional abundance, then these children would have greater propensity to CVD as adults.

The model we estimate is:

$$MR_i = \beta_0 + \beta_1 HI_i + \theta X$$

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 is a set of covariates that control for other 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

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

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 yield 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 could use turnover as an independent variable; however, there is no theoretically founded explanation for this type of relationship between migration and mortality. We believe that a more appealing option is to use a weighting scheme that reduces the importance of high-turnover states in the analysis.

Results

Figure 3 presents a scatter diagram of a statistically significant relationship of the mortality rate at ages 55-84 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 44.5 (std. error = 15.1), which implies that a two unit increase in the income ratio would explain about one standard deviation of the mortality rate across the states.

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.

Tables 3 through 5 display the results of regressions of mortality rates on the median income ratio, education, obesity, and smoking using the inverse of average turnover as weights.[7] In principle our concerns about turnover may have been well-founded, but the empirical results are affected little by weighting. Comparing results from the non-weighted regressions (Tables 3(a)-(c)), with those weighted using the inverse of average turnover from 1950 to 1980 (Tables 4(a)-(c)), and those using the same weighting scheme from 1950 to 2000 (Tables 5(a)-(c)), reveals similar patterns of coefficient sizes and statistical significance.

To further account for the effects of southern birth, tables 6 (a) – (c) display the results of the same regression specification except using percent of the population born in the South as a weight. If the DOHaD 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.

One might think that the initial level of income would affect the relationship; including median household income in 1950, however, 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 also has the expected sign. More people with less education implies that the average resident of the state is less informed about the importance of

[7] The definitions of the variables are as follows: rMedian8050 is the ratio of median income in 1980 to median income in 1950. ≤HS is the percentage of the state's population with a high school diploma or GED or less in 2010. Obesity is the percentage of the state's population with BMI greater than or equal to 30 in 2011 and Smoking is the percentage of state's population that are currently smokers who self-report as smoking at least once in the previous week in 2011.

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 (Lantz, House et al. 1998, Lleras-Muney 2005). 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 = 0.64), and so it is difficult to obtain precise estimates of their independent effects on mortality. When entered separately each variable is statistically significant (see Tables 6 (a) – (b)), but standard errors of the coefficients are somewhat higher when entered together as in Table 6 (c).

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 DOHaD hypothesis.

Second, 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, however, 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. We ran different specifications to test the effects of income inequality on our results but found little effect. [8]

[8] We calculated state-level Gini coefficients for income in 1980 and 1950. The ratio of the Gini coefficient 1980 to 1950 is negatively correlated with both CVD mortality and the ratio of median household income, but the relationship is rather weak and insignificant (see Figure 8). Another measure of inequality, other than the Gini coefficient, is the standard deviation of the income distribution. The ratio of standard deviation of income 1980 to

Third, 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 60 per cent 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.[9] We should ask, however, whether the ecological fallacy distorts the interpretation of relationships measured in Tables 5 and 6. 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

1950 is insignificant for all age cohorts and its inclusion doesn't appreciably affect the other coefficients. These results are available from the authors upon request.

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

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.

The coefficients on the obesity rate in 2011 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, Powers et al. 2013, Masters, Reither et al. 2013).

The coefficient on smoking in 2011 is positive and statistically significant for all age cohorts. The overall mortality among smokers in the United States is about three times higher than that among similar people who never smoked.[10] 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.[11] 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.

Possible Mechanisms. What processes might have translated rapid income growth in the South into adolescent weight gain? 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

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

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

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. As can be clearly seen in Figure 4, there is a strong negative correlation between CVD morality and health club membership. When the other control variables are included, however, health club membership becomes insignificant.

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 (see Table 7).[12] 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] http://www.cdc.gov/nutrition/downloads/State-Indicator-Report-Fruits-Vegetables-2013.pdf

Conclusions

Our paper makes two important contributions. First, the analysis confirms or is consistent with the developmental origins hypothesis as applied to CVD in explaining regional differences in mortality within the United States in 2010-2011. 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 body has less ability to adapt to a new environment.

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 Barker hypothesis has especially relevant implications for the developing world, where vast amounts of poor families are on the verge of experiencing significant increases in relative income. Chronic adult illness, like heart disease, are projected to increase dramatically in this locations in the future (Lopez, Mathers et al. 2006); Kinsella and He 2009).

References

Active Marketing Group (2007). Health Club Industry Review. San Diego**:** 1-17.

Anker, R. (2011). Engel's Law Around the World 150 Years Later. Amherst, MA, University of Massachusetts Amherst, Political Economy Research Institute.

Barker, D. (1995). "Fetal origins of coronary heart disease." British Medical Journal (International Edition) **311**(6998).

Barker, D. (2002). "Fetal Programming of Coronary Heart Disease." Trends in Endocrinology and Metabolism Trends in Endocrinology and Metabolism **13**(9): 364-368.

Barker, D. and C. Osmond (1986). "Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales." Lancet **1**(8489): 1077-1081.

Barker, D. and K. Thornburg (2013). "The obstetric origins of health for a lifetime." Clinical Obstetrics and Gynecology **56**(3): 511-519.

Basu, K. and P. H. Van (1998). "The Economics of Child Labor." American Economic Review **88**(3): 412.

Bateson, P., D. Barker, T. Clutton-Brock, D. Deb, B. D'Udine, R. A. Foley, P. Gluckman, K. Godfrey, T. Kirkwood, M. M. Lahr, J. McNamara, N. B. Metcalfe, P. Monaghan, H. G. Spencer and S. E. Sultan (2004). "Developmental Plasticity and Human Health." Nature **430**(6998): 419-421.

Dandona, S., A. F. R. Stewart and R. Roberts (2010). "Genomics in coronary artery disease: Past, present and future." anadian Journal of Cardiology **26**(SUPPLA): 56A-59A.

De Backer, G. (2008). "Risk Factors and Prevention of Cardiovascular Disease: A Review." Dialogues **13**(2): 83-90.

Devine, C. M., M. Jastran, J. Jabs, E. Wethington, T. J. Farell and C. A. Bisogni (2006). "A lot of sacrifices: Workfamily spillover and the food choice coping strategies of low-wage employed parents." Social Science & Medicine Social Science & Medicine **63**(10): 2591-2603.

Ellenberg, G. (2007). Mule South to tractor South mules, machines, agriculture, and culture in the cotton South, 1850-1950. Tuscaloosa, University of Alabama.

Faeh, D., J. Braun, S. Tarnutzer and M. Bopp (2011). "Obesity but not overweight is associated with increased mortality risk." European Journal of Epidemiology **26**(8): 647-655.

Fields, G. S. (2001). Distribution and development : a new look at the developing world. Cambridge, Mass., MIT Press.

Futterman, L. G. and L. Lemberg (1998). "Fifty percent of patients with coronary artery disease do not have any of the conventional risk factors." American Journal of Critical Care **7**(3): 240-244.

Graham, G. (2014). "Population-based approaches to understanding disparities in cardiovascular disease risk in the United States." International Journal of General Medicine **7**: 393-400.

Grundy, S. M., I. J. Benjamin, G. L. Burke, A. Chait, R. H. Eckel, B. V. Howard, W. Mitch, S. C. J. Smith and J. R. Sowers (1999). "Diabetes and cardiovascular disease: a statement for healthcare professionals from the American Heart Association." Circulation **100**(10): 1134-1146.

Hanson, M. A. and P. D. Gluckman (2008). "Developmental Origins of Health and Disease: New Insights." Basic & Clinical Pharmacology & Toxicology **102**(2): 90-93.

Hareven, T. K. (1982). Family time and industrial time : the relationship between the family and work in a New England industrial community. Cambridge; New York, Cambridge University Press.

Heinicke, C. and W. A. Grove (2008). ""Machinery Has Completely Taken Over": The Diffusion of the Mechanical Cotton Picker, 19491964." Journal of Interdisciplinary History **39**(1): 65-96.

Heron, M. (2013). "Deaths: leading causes for 2010." National Vital Statistics Reports **62**(6): 1-96.

Hurt, R. D. (1989). Mechanization. Encyclopedia of Southern Culture. C. Wilson and W. Ferris. Chapel Hill, University of North Carolina Press**:** 26-27.

Kim, S. and R. A. Margo (2003). Historical Perspectives on U.S. Economic Geography. Cambridge, Mass., National Bureau of Economic Research.

Kuzawa, C. and E. Sweet (2009). "Epigenetics and the embodiment of race: Developmental origins of US racial disparities in cardiovascular health." American Journal of Human Biology **21**(1): 2-15.

Kuzawa, C. W. (2005). "Fetal origins of developmental plasticity: Are fetal cues reliable predictors of future nutritional environments?" American Journal of Human Biology **17**(1): 5-21.

Lantz, P. M., J. S. House, J. M. Lepkowski, D. R. Williams, R. P. Mero and J. Chen (1998). "Socioeconomic factors, health behaviors, and mortality: Results from a nationally representative prospective study of us adults." JAMA **279**(21): 1703-1708.

Lleras-Muney, A. (2005). "The Relationship Between Education and Adult Mortality in the United States." Review of Economic Studies **72**(1): 189-221.

Logan, T. D. (2012). Freedom Passed Us By: The Social and Economic World of a Cotton Picking Family 1950-1970. Columbus, The Ohio State University

Lopez, A. D., C. D. Mathers, M. Ezzati, D. T. Jamison and C. J. L. Murray (2006). "Global and regional burden of disease and risk factors, 2001: Systematic analysis of population health data." Lancet **367**(9524).

Lynch, J., S. Harper, G. Davey Smith, N. Ross, M. Wolfson and J. Dunn (2004). "US regional and national cause-specific mortality and trends in income inequality: Descriptive Findings." Demographic Research **Special 2**: 183-228.

Masters, R. K., D. A. Powers and B. G. Link (2013). "Obesity and US mortality risk over the adult life course." American Journal of Epidemiology **177**(5): 431-442.

Masters, R. K., E. N. Reither, D. A. Powers, Y. C. Yang, A. E. Burger and B. G. Link (2013). "The Impact of Obesity on US Mortality Levels: The Importance of Age and Cohort Factors in Population Estimates." American Journal of Public Health **103**(10): 1895-1901.

McEniry, M. and A. Palloni (2010). "Early Life Exposures and the Occurrence and Timing of Heart Disease Among the Older Adult Puerto Rican Population." Demography **47**(1): 23-43.

McMillen, S. (1989). No Easy Time: Rural Southern Women, 1940-1990. The Rural South since World War II. . R. D. Hurt. Baton Rouge, Louisiana State University Press**:** 59-94.

Pickle, L. W. (1996). Atlas of United States Mortality. Hyattsville, Md., National Center for Health Statistics, Centers for Disease Control and Prevention, U.S. Dept. of Health and Human Services.

Prins, B. P., V. Lagou, F. W. Asselbergs, H. Snieder and J. Fu (2012). "Genetics of coronary artery disease: Genome-wide association studies and beyond." Atherosclerosis **225**(1): 1-10.

Roberts, R. and A. F. R. Stewart (2012). "Genes and Coronary Artery Disease:Where Are We?" Journal of the American College of Cardiology **60**(18): 1715-1721.

Steckel, R. H. (2013). "The hidden cost of moving up: Type 2 diabetes and the escape from persistent poverty in the American South." American Journal of Human Biology **25**(4): 508-515.

Stöger, R. (2008). "The thrifty epigenotype: an acquired and heritable predisposition for obesity and diabetes?" BioEssays **30**(2): 156-166.

Swerdlow, D., M. Holmes, S. Harrison and S. Humphries (2012). "The genetics of coronary heart disease." British Medical Bulletin **102**(1): 59-77.

Taylor, J. G. (1989). Foodways. Encyclopedia of Southern Culture. C. Wilson and W. Ferris. Chapel Hill, University of North Carolina**:** 613-616.

Thayer, Z. M. and C. W. Kuzawa (2011). "Biological memories of past environments: Epigenetic pathways to health disparities." Epigenetics **6**(7): 798-803.

Tilly, L. and J. W. Scott (1978). Women, work, and family. New York, Holt, Rinehart and Winston.

Torres, S. J. and C. A. Nowson (2007). "Relationship between stress, eating behavior, and obesity." Nutrition **23**(11-12): 887-894.

Watson, R. R. and V. R. Preedy (2013). Bioactive food as dietary interventions for cardiovascular disease. San Diego, Academic Press.

Zheng, H., D. Tumin and Z. Qian (2013). "Obesity and Mortality Risk: New Findings From Body Mass Index Trajectories." American Journal of Epidemiology American Journal of Epidemiology **178**(11): 1591-1599.

Rank	State	All Cause	Heart Disease	Rank	State	All Cause	Heart Disease
1	West Virginia	970.2	332.2	25	Virginia	778.9	257.5
2	Kentucky	944.1	322.1	26	Illinois	777.2	273.3
3	Alabama	940.9	330.9	27	Montana	774.6	236.4
4	Mississippi	940.1	345.9	28	Maryland	766.8	266
5	Oklahoma	939.3	346.8	29	Idaho	766	247.3
6	Tennessee	909.8	316.7	30	Rhode Island	765.7	263.6
7	Arkansas	905	320	31	Washington	752.3	245.4
8	Louisiana	904.3	307.4	32	Vermont	751.6	242.2
9	Nevada	875.2	293.2	33	New Hampshire	751.1	247.2
10	Georgia	862.3	289.5	34	Nebraska	751	246.7
11	Indiana	853	295.1	35	New Jersey	749.2	267.8
12	South Carolina	849.8	275.4	36	Iowa	749	264
13	Missouri	848.6	303.6	37	Massachusetts	746.9	238.9
14	Ohio	846.7	293.7	38	Wisconsin	744.2	254
15	North Carolina	823.7	270	39	Arizona	743.3	236.6
16	Texas	810.4	279	40	Utah	741.8	228.9
17	Pennsylvania	807	283.1	41	New York	737.3	293.8
18	Wyoming	805.5	251.3	42	Colorado	735.7	222.8
19	Michigan	801.4	293.7	43	California	735.6	271.2
20	Delaware	797.8	267.4	44	Florida	729.8	243.5
21	Maine	796.6	246.1	45	South Dakota	715.4	246
22	Kansas	793.5	264.9	46	North Dakota	709.2	245.2
23	New Mexico	785.3	236.5	47	Connecticut	706.4	238.9
24	Oregon	779.7	241.3	48	Minnesota	691	204.9

Table 1: Age-Adjusted All Cause and Heart Disease Mortality Rates per 100,000 for Whites by State

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

http://wonder.cdc.gov/ucd-icd10.html

	Obesity	Smoking	rMedian8050	≤HS
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	2.964	3.298	0.585	6.381
N	48	48	48	48

Table 2(a): Descriptive Statistics of Independent Variables

	55-64	65-74	75-84	55-84	% Southern	Turnover 8050	Turnover 0050
Max	330.1	696	1,989.2	762.9	0.6960	2,426,276	2,459,675
Mean	212.5	499.99	1,538.1	545.94	0.3457	525,168	561,592
Median	201.9	472.1	1,499.5	482.05	0.3220	409,600	434,780
Min	130.2	341.4	1,134.9	378.3	0.1202	80,457	90,096
Std Dev	51.7	91.35	193.1	92.64	0.1614	453,378	471,839
N	48	48	48	48	48	48	48

Table 2(b): Descriptive Statistics of Death Rates per 100,000 by Age Cohort and Population Turnover

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	27.53***	33.29**	57.52*	30.57**
	[3.47]	[2.62]	[1.78]	[2.32]
≤HS	4.145***	8.964***	18.40***	9.187***
	[4.98]	[6.72]	[5.43]	[6.66]
Smoking	4.451***	7.106***	11.47*	7.003**
	[2.70]	[2.69]	[1.71]	[2.56]
Constant	-143.29***	144.28**	303.95*	-98.54
	[-3.66]	[-2.30]	[1.90]	[-1.52]
N	48	48	48	48
Adj R2	0.6416	0.7051	0.5734	0.6925

Table 3(a) Regressing mortality rate on income ratio, education, and smoking

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	32.88***	42.01***	72.75**	39.26***
	[4.23]	[3.46]	[2.45]	[3.15]
≤HS	3.974***	8.39***	15.646***	8.496***
	[4.57]	[6.18]	[4.71]	[6.09]
Obesity	5.00***	9.074***	21.54***	9.44***
	[2.68]	[3.11]	[3.02]	[3.16]
Constant	-205.18***	-258.87***	18.17	-218.80***
	[-4.32]	[-3.48]	[0.10]	[-2.87]
N	48	48	48	48
Adj R2	0.6408	0.7185	0.6233	0.7120

Table 3(b) Regressing mortality rate on income ratio, education, and obesity

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	29.57***	37.45***	69.96**	35.10***
	[3.74]	[3.00]	[2.25]	[2.73]
≤HS	3.647***	7.947***	15.37***	8.085***
	[4.15]	[5.74]	[4.45]	[5.67]
Obesity	3.304	6.736**	20.11**	7.311**
	[1.56]	[2.02]	[2.41]	[2.13]
Smoking	2.978	4.104	2.510	3.745
	[1.59]	[1.39]	[0.34]	[1.23]
Constant	-187.94***	-235.10***	32.70	-197.12***
	[-3.11]	[-3.11]	[0.17]	[-2.53]
N	48	48	48	48
Adj R2	0.6528	0.7243	0.6156	0.7153

Table 3(c) Regressing mortality rate on income ratio, education, smoking, and obesity

Table 3: Regression Results by Age Cohort, t-statistics in brackets, * p<0.10 ** p<0.05 *** p<0.01, unweighted

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	26.59***	37.88***	79.61**	29.98**
	[3.32]	[3.14]	[2.67]	[2.23]
≤HS	4.47***	9.072***	18.82***	9.54***
	[4.95]	[6.66]	[5.6]	[6.29]
Smoking	4.68***	8.086***	11.69*	7.701***
	[2.79]	[3.2]	[1.87]	[2.74]
Constant	-166.37***	-188.39***	188.57	-138.23*
	[-3.83]	[-2.88]	[1.17]	[-1.89]
N	48	48	48	48
Adj R2	0.6097	0.6997	0.5817	0.6478

Table 4(a) Regressing mortality rate on income ratio, education, and smoking

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	29.47***	43.68***	80.71**	34.02**
	[3.73]	[3.56]	[3.01]	[2.63]
≤HS	4.32***	9.15***	15.96***	9.02***
	[4.54]	[6.19]	[4.94]	[5.78]
Obesity	5.29**	7.67**	23.80***	9.88***
	[2.6]	[2.43]	[3.45]	[2.96]
Constant	-223.69***	-267.49***	-97.81	-248.48***
	[-4.34]	[-3.34]	[-0.56]	[-2.94]
N	48	48	48	48
Adj R2	0.6017	0.6734	0.6443	0.6563

Table 4(b) Regressing mortality rate on income ratio, education, and obesity

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	25.83***	36.90***	75.24***	28.44**
	[3.30]	[3.10]	[2.73]	[2.20]
≤HS	3.845***	8.266***	15.25***	8.288***
	[4.06]	[5.73]	[4.56]	[5.29]
Obesity	3.780*	4.852	21.53***	7.567**
	[1.80]	[1.52]	[2.91]	[2.18]
Smoking	3.577**	6.663**	5.373	5.481*
	[2.05]	[2.50]	[0.87]	[1.90]
Constant	-214.1***	-249.7***	-83.48	-233.9***
	[-4.28]	[-3.28]	[-0.47]	[-2.83]
N	48	48	48	48
Adj R2	0.629	0.708	0.642	0.676

Table 4(c) Regressing mortality rate on income ratio, education, smoking, and obesity

Table 4: Regression Results by Age Cohort, t-statistics in brackets, * p<0.10 ** p<0.05 *** p<0.01, weighted by average turnover 1950-1980

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	25.87***	37.52***	80.32***	29.23**
	[3.28]	[3.18]	[2.72]	[2.19]
≤HS	4.542***	9.201***	19.16***	9.649***
	[5.18]	[7.01]	[5.83]	[6.50]
Smoking	4.702***	8.083***	11.90*	7.839***
	[2.87]	[3.30]	[1.94]	[2.83]
Constant	-168.3***	-193.9***	165.8	-143.6*
	[-3.93]	[-3.03]	[1.03]	[-1.98]
N	48	48	48	48
Adj R2	0.618	0.712	0.594	0.655

Table 5(a) Regressing mortality rate on income ratio, education, and smoking

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	28.31***	42.70***	80.04***	32.65**
	[3.65]	[3.55]	[3.01]	[2.54]
≤HS	4.325***	9.190***	16.26***	9.056***
	[4.67]	[6.41]	[5.13]	[5.90]
Obesity	5.442***	7.792**	23.91***	10.07***
	[2.74]	[2.54]	[3.52]	[3.06]
Constant	-224.7***	-269.9***	-113.5	-251.1***
	[-4.46]	[-3.46]	[-0.66]	[-3.01]
N	48	48	48	48
Adj R^2	0.613	0.687	0.656	0.664

Table 5(b) Regressing mortality rate on income ratio, education, and obesity

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	24.83***	36.20***	74.59***	27.18**
	[3.24]	[3.12]	[2.73]	[2.13]
≤HS	3.888***	8.374***	15.58***	8.370***
	[4.24]	[6.04]	[4.78]	[5.48]
Obesity	3.935*	4.976	21.55***	7.699**
	[1.92]	[1.61]	[2.96]	[2.26]
Smoking	3.540**	6.614**	5.543	5.566*
	[2.08]	[2.57]	[0.92]	[1.96]
Constant	-217.1***	-255.7***	-101.7	-239.1***
	[-4.46]	[-3.47]	[-0.59]	[-2.95]
N	48	48	48	48
Adj R^2	0.640	0.722	0.655	0.685

Table 5(c) Regressing mortality rate on income ratio, education, smoking, and obesity

Table 5: Regression Results by Age Cohort, t-statistics in brackets, * p<0.10 ** p<0.05 *** p<0.01, weighted by average turnover 1950-2000

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	26.19***	36.64***	75.27***	34.44***
	[3.54]	[3.08]	[2.48]	[2.75]
≤HS	3.99***	8.85***	17.39***	8.60***
	[4.98]	[6.87]	[5.29]	[6.34]
Smoking	5.97***	9.18***	17.15**	9.93***
	[3.73]	[3.57]	[2.61]	[3.66]
Constant	-152.64***	-179.92***	212.86	-126.84*
	[-4.16]	[-3.05]	[1.41]	[-2.04]
N	48	48	48	48
Adj R2	0.7127	0.7666	0.6537	0.7469

Table 6(a) Regressing mortality rate on income ratio, education, and smoking

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	33.09***	47.26***	95.14***	45.93***
	[4.55]	[4.31]	[3.49]	[3.98]
≤HS	3.80***	7.99***	14.49***	7.67***
	[4.45]	[6.2]	[4.53]	[5.66]
Obesity	6.53***	12.07***	27.19***	13.07***
	[3.52]	[4.31]	[3.91]	[4.44]
Constant	-232.08***	-330.18***	-131.87	-289.53***
	[-5.15]	[-4.85]	[-0.78]	[-4.05]
N	48	48	48	48
Adj R^2	0.7051	0.7885	0.7033	0.772

Table 6(b) Regressing mortality rate on income ratio, education, and obesity

	55-64	**65-74**	**75-84**	**55-84**
rMedian8050	28.40***	41.63***	88.23***	39.84***
	[3.89]	[3.7]	[3.09]	[3.38]
≤HS	3.39***	7.50***	13.88***	7.13***
	[4.03]	[5.79]	[4.21]	[5.25]
Obesity	4.00*	9.04***	23.47***	9.80***
	[1.89]	[2.77]	[2.83]	[2.86]
Smoking	4.06**	4.88*	5.99	5.28*
	[2.19]	[1.71]	[0.82]	[1.76]
Constant	-204.56***	-297.14***	-91.33	-253.80***
	[-4.54]	[-4.28]	[-0.52]	[-3.49]
N	48	48	48	48
Adj R^2	0.7286	0.7973	0.7011	0.7824

Table 6(c) Regressing mortality rate on income ratio, education, smoking, and obesity

Table 6: Regression Results by Age Cohort, t-statistics in brackets, * p<0.10 ** p<0.05 *** p<0.01, weighted by percentage of 2010 population that was born in the South

State	Fruit	Vegetable	State	Fruit	Vegetable
Mississippi	50.8	32.3	Wyoming	38.2	22.4
Oklahoma	50.2	26.8	Idaho	38.1	20.1
Arkansas	47.5	28.6	New Mexico	38	21.9
West Virginia	47.2	26.2	Arizona	38	20.6
Louisiana	46.7	32.5	Florida	37.7	22.6
Tennessee	46.3	25.4	Michigan	37.3	23.2
Kentucky	45.9	25.2	Nevada	36.9	24.4
South Carolina	44.4	27.3	Maryland	36.4	22.8
Missouri	43.9	25.2	Minnesota	36.2	23.6
Alabama	43.8	24.3	Pennsylvania	36.1	23.9
Georgia	41.9	23.2	Illinois	36	25.2
Indiana	41.6	27.3	Colorado	35.7	19.1
Kansas	41.4	22.2	Wisconsin	35.6	26
North Carolina	40.8	21.9	Washington	35	18.8
Ohio	40.5	26	Utah	34.9	19.8
Texas	40.3	21.8	New York	33.9	23
Nebraska	40.1	26.2	New Jersey	33.9	22.2
Iowa	39.8	26.9	Maine	33.2	18.9
South Dakota	39.6	26.3	Rhode Island	32.9	20.7
Hawaii	39.5	22.6	Connecticut	32	20.6
Montana	39.2	21.7	Oregon	32	15.3
Delaware	39.2	23.8	Massachusetts	31.6	20.7
North Dakota	39.1	27.1	Vermont	31.4	18.1
Alaska	38.7	19.7	California	30.4	16.5
Virginia	38.4	22.2	New Hampshire	30.3	17.6

Table 7: Percentage of 18+ Population, All Genders who self-report eating one or less fruit (of any form) per day over the last month. CDC: Division of Nutrition, Physical Activity, and Obesity.

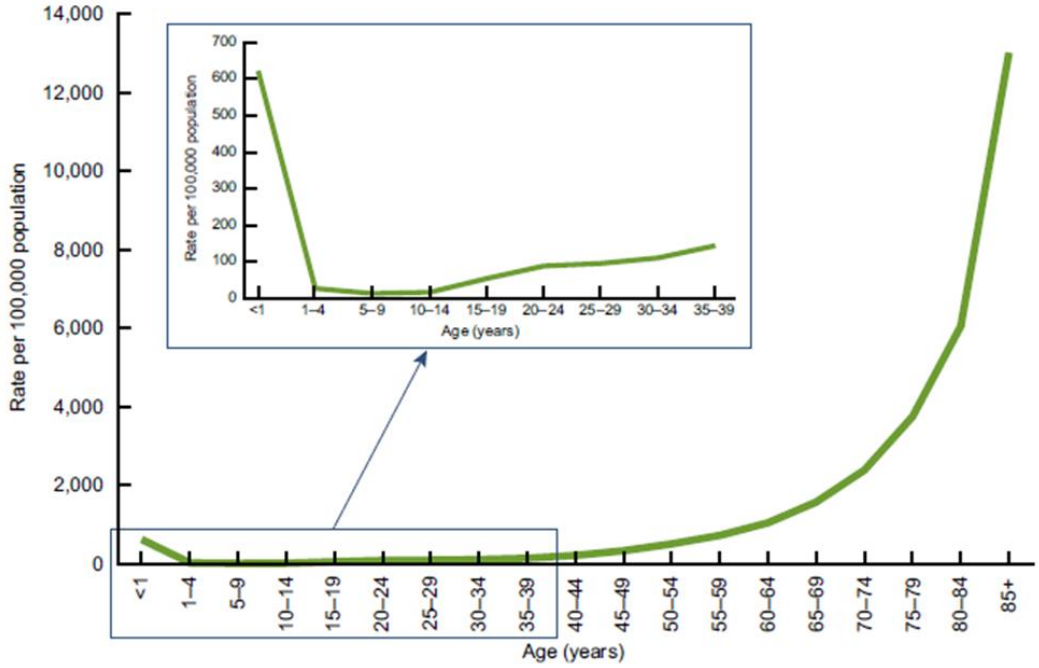

Figure 1: Age-specific death rates from CVD in 2009.

Source: CDC/NCHS, National Vital Statistics System.

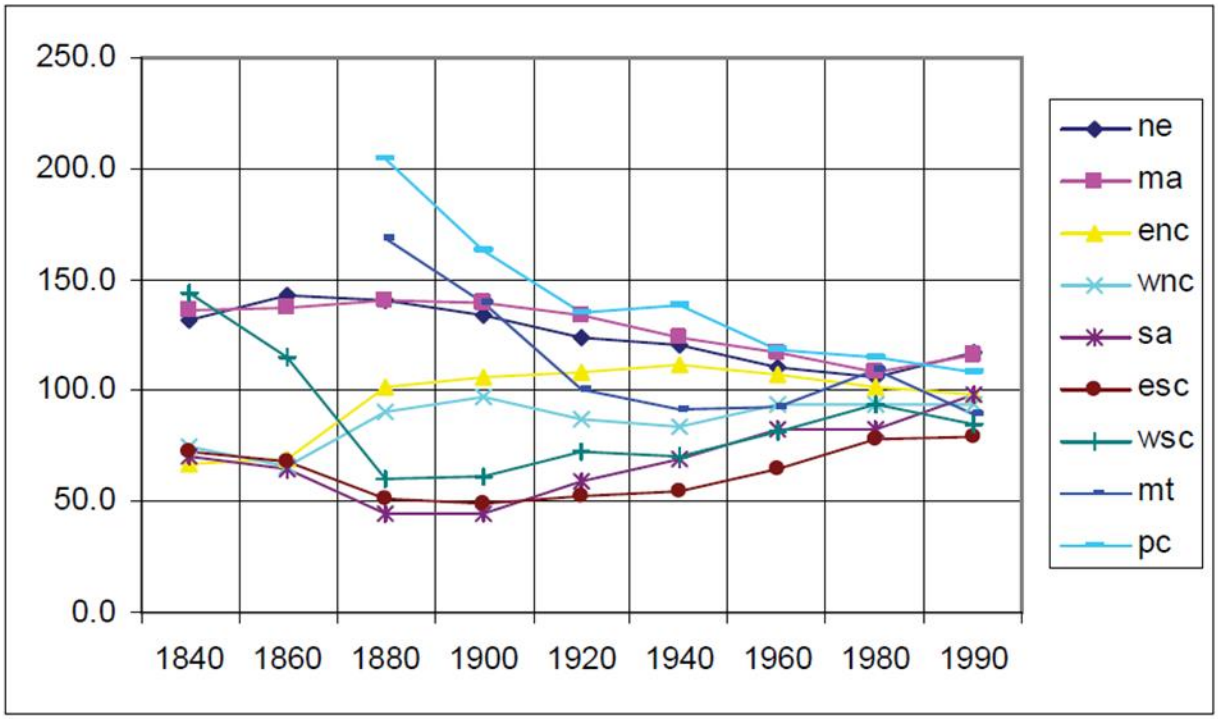

Figure 2: Regional Income Per Capita, 1840-1990; Kim and Margo, 2003. Legend: ne = New England; ma = Middle Atlantic; enc = East North Central; wnc = West North Central; sa = South Atlantic; esc = East South Central; wsc = West South Central; mt = Mountain; pc = Pacific.

Source: (Kim and Margo 2003)

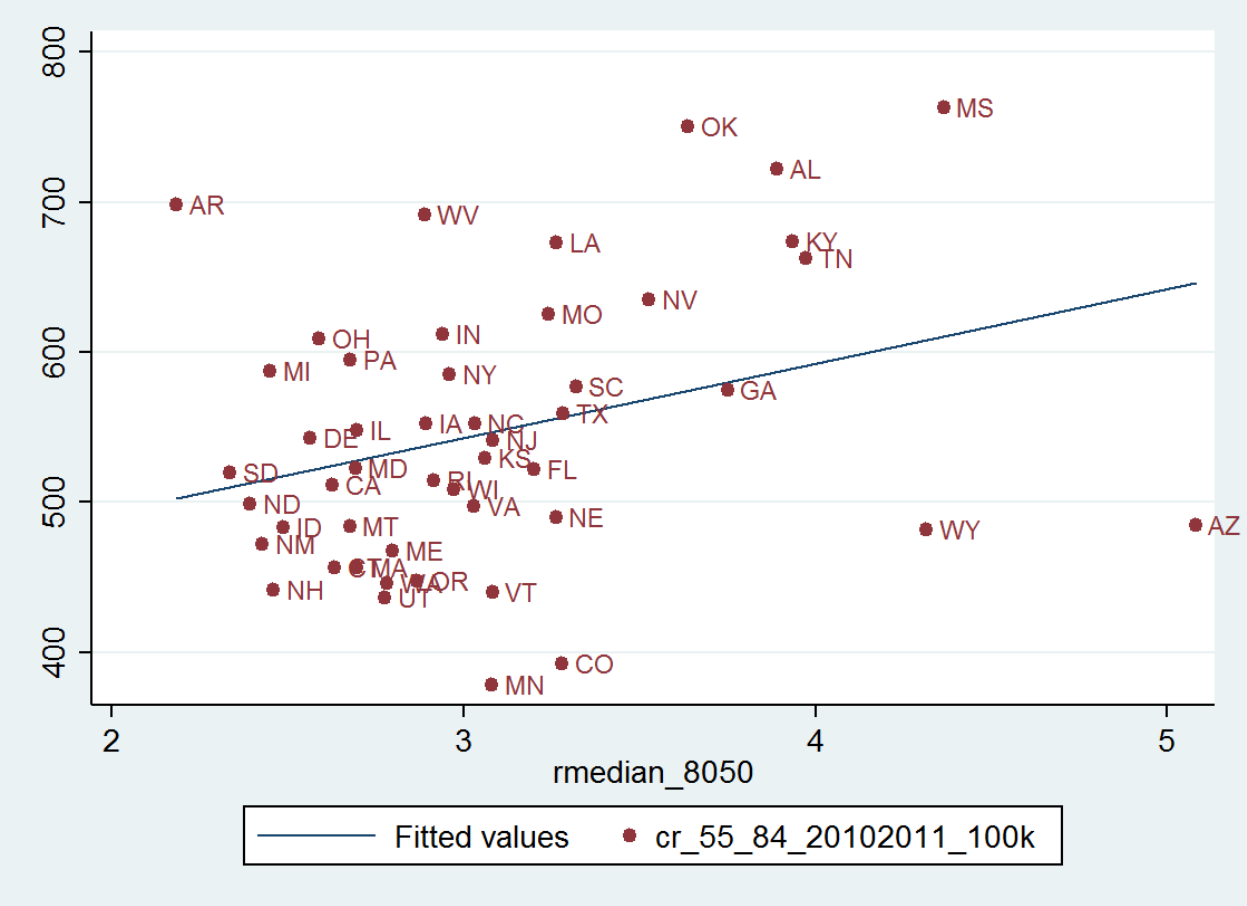

Figure 3: Scatter plot of Median Household Income vs Crude Death Rate for the 55-84 Age Cohort

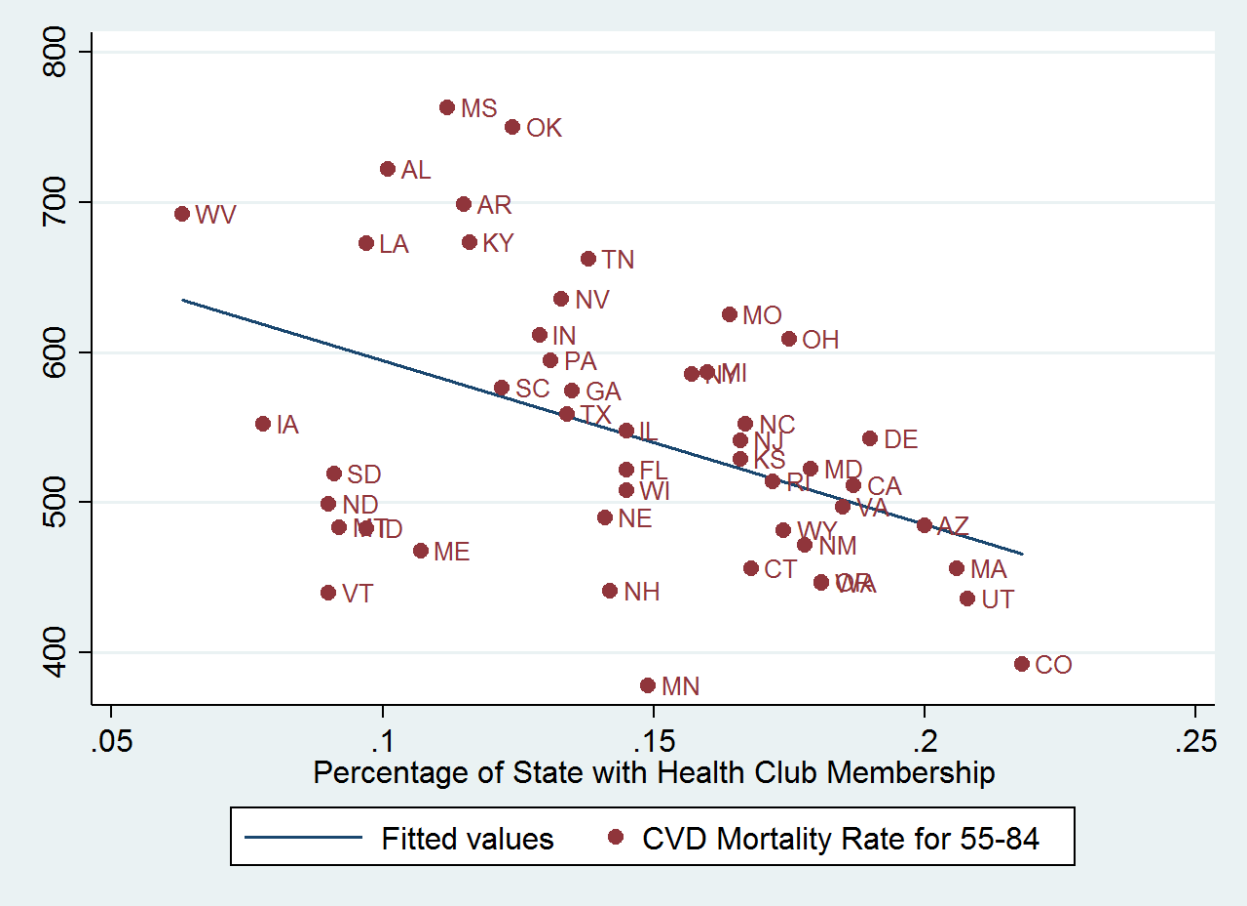

Figure 4: Scatter plot of Percentage of State with Health Club Membership vs CVD Crude Death Rate for the 55-84 Age Cohort