Chapter 2
The Continuing Puzzle of Hypertension Among African Americans: Developmental Origins and the Mid-century Socioeconomic Transformation

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Abstract African Americans have an excessive prevalence of hypertension relative to whites, particularly in the South. We seek to understand this puzzle by applying the developmental origins hypothesis to the rapid socioeconomic improvement that occurred after World War II. The long experience of pre-World War II poverty prepared African Americans born around the 1950s for survival in a lean world of poor nutrition and hard work, but created vulnerabilities for chronic diseases when conditions improved later in life. We analyze individual-level evidence from the CDC's Behavioral Risk Factor Surveillance System with household income data, finding results consistent with the developmental origins hypothesis, that accelerated income growth from poverty strongly indicates an increased prevalence of hypertension. This strongly suggests that the collection of individual-level, intergenerational data is necessary to further evaluate this puzzle.

Keywords Developmental origins · Hypertension · Health · Behavioral Risk Factor Surveillance System · Cardiovascular disease

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2.1 Introduction

African Americans have a high prevalence of hypertension, an important precursor of cardiovascular disease and stroke, which is a major contributor to the racial disparities in health observed in America (Lackland and Keil 1996; Sowers et al. 2002; Wong et al. 2002; Hertz et al. 2005). In 2007–2014, the age-adjusted hypertension prevalence rates were 41.6% for African Americans and 29.0% for whites (Mozaffarian et al. 2016, chart 9.2). An extensive literature documents that the disparity in hypertension prevalence persists even after adjustment for a wide range of socioeconomic and behavioral factors (Redmond et al. 2011). Figure 2.1 shows that the prevalence of hypertension for African Americans in 2011 varied widely across the country. It is important to note that hypertension is particularly pronounced in the South. The highest rates exist in the swath of states from Texas to West Virginia. Notably, this region roughly coincides with the stroke and the diabetes belts identified by the CDC, conditions that are recognized as correlates or precursors of cardiovascular disease (Lackland et al. 2016).[1]

The prevalence of hypertension is also elevated in other states adjacent to this block. However, that elevated prevalence for African Americans living in the South contrasts with the lower rates among blacks living in Africa or in the Caribbean (Cooper et al. 1997). Given this evidence, unique aspects of the American environment likely contribute to the pattern of hypertension prevalence. The previous literature discusses numerous possible explanations for the high prevalence of hypertension among African Americans, but there is no scientific consensus on its

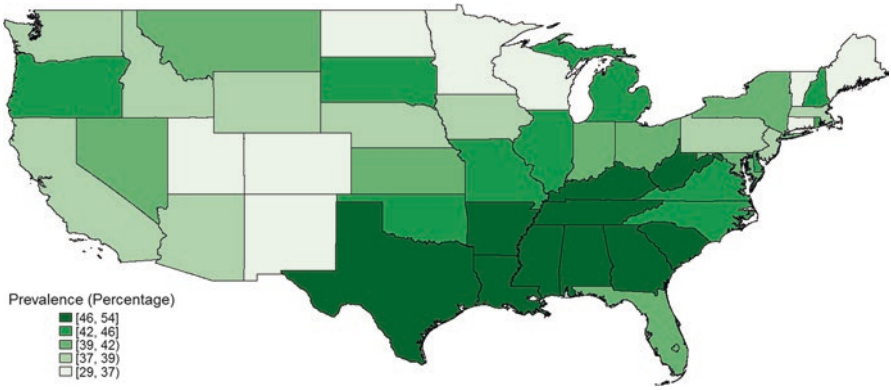

Fig. 2.1 Hypertension prevalence in 2011 of African Americans 18 years and older. (Source: BRFSS, CDC. Note: Prevalence denotes the share of individuals who are being treated for hypertension or who have been told by a physician, nurse, or healthcare professional that they have hypertension. Because some individuals have not been examined for hypertension, this measure underestimates the true rate)

[1] www.cdc.gov/dhdsp/maps/national_maps/stroke_all.htm and www.cdc.gov/diabetes/pdfs/data/diabetesbelt.pdf.

underlying cause. Therefore, the situation of higher prevalence of hypertension among African Americans in the South is a unique and interesting public health puzzle.

One approach worth further study, the developmental origins hypothesis, relates changes in the intergenerational socioeconomic conditions to increases in the prevalence of chronic adult diseases. Medical studies have shown that young children respond to poor nutrition and stress by compromising organ integrity and degrading biological processes that regulate physiological systems in later life (Gluckman et al. 2008; Barker and Thornburg 2013). If people rendered vulnerable then face an energy-rich diet or elevated stress in later life, pathophysiological processes are set into motion that might significantly increase their likelihood to have hypertension.

The developmental origins hypothesis views rapid socioeconomic improvement near the middle of the twentieth century as a possible latent factor that has elevated hypertension among African Americans who had an intergenerational history of poverty. Beginning in the 1950s, the South underwent an economic, social, technological, and political revolution that had profound effects for African Americans, creating a sudden improvement in living conditions broadly defined. Being guided by the literature on hypertension and the developmental origins hypothesis, we empirically examine the claim that this major socioeconomic change created unbalanced physical growth for cohorts born on the cusp of change, which made them particularly vulnerable to hypertension as adults.

We investigate the connection between socioeconomic change and hypertension using individual-level data gathered by the Behavioral Risk Factor Surveillance System (BRFSS) and by state-level, household income data for African Americans. As discussed below, two factors allow us to identify and measure the impact of the changing socioeconomic environment on hypertension among African Americans. First, conditions in utero are crucial for the development of major organs such as the cardiovascular system. At this age, the growth process adapts the design of these organs to the type of nutritional conditions the child is expected to inhabit in later life. Once fully formed, these organs have limited capacity to adapt to changing environmental conditions. Second, a sudden improvement in conditions between birth and adulthood places these cohorts at greater risk of hypertension. Successive generations of poverty and hard work, for example, led to low birth weight and limited the functional capacity of these organ systems. By dividing the BRFSS data into birth cohorts that experienced contrasting rates of socioeconomic change, we can measure the effects of rapid socioeconomic improvement on adult hypertension.

We consider how rapid socioeconomic change operated on biological processes known to affect hypertension. In this regard, income acts as a portmanteau variable that captures several effects associated with improving socioeconomic status. The others include diet, work effort or physical activity, leisure, and obesity, all of which accompanied the mid-century industrialization and modernization of the South. Specifically, we consider how the diffusion of new technology, especially the tractor and the cotton picker after 1950, eased work effort and calories expended in agriculture. Higher incomes enabled the purchases of automobiles that reduced the need for walking as a primary source of transportation. Other activities that would expend

calories, such as recreational exercise, made limited headway in the South. With industrialization women increasingly worked outside the home, and while beneficial for income, led to unsupervised eating habits of children who consumed more snack foods, perhaps while watching television after school, and to greater purchase of less-nutritious prepared foods for family meals. In the context of the southern diet, which featured starch, fat, and salt, these behavior patterns contributed to obesity, which is a major risk factor for hypertension (Hall et al. 2015; Jiang et al. 2016; Leggio et al. 2017).

Because our data and methods lead to conclusions that are only suggestive, an important goal is to motivate the collection of intergenerational household-level data for African Americans that could provide a rigorous evaluation of this methodology. Most useful would be intergenerational evidence on household income and socioeconomic status combined with measures of lifestyle behaviors and hypertension.

2.2 Background

The literature discusses numerous possible explanations for the disparity in hypertension prevalence, including obesity, diet, quality of medical care, stress related to socioeconomic change, poor access to health insurance, socioeconomic status, salt retention, and substance abuse (Centers for Disease Control and Prevention 2010). Several studies report, however, that the disparity in hypertension prevalence persists even after adjustment for a wide range of socioeconomic, behavioral, and biomedical risk factors (Redmond et al. 2011). Despite large interventions to eliminate hypertension disparities, evidence such as that shown in Table 2.2 indicates that these differences have actually grown over the past few decades (Geronimus et al. 2007), suggesting that unrecognized factors are important in driving inequalities in hypertension (Fuchs 2011).

The developmental origins hypothesis provides a mechanism for understanding the origins of hypertension and other noncommunicable diseases found in later adult life. Some 30 years ago, David Barker and colleagues proposed the developmental origins approach (Barker and Osmond 1986; Barker 1990), after Barker noticed that older adult deaths from heart disease in England were correlated with infant mortality rates and birth sizes in their cohorts and geographic locations of birth. Although there were many skeptics, discovery of the relationship stimulated a search for possible mechanisms built around the idea that early life conditions influence adult susceptibility to cardiovascular disease (Lackland 2004). Subsequently, medical researchers proposed and refined ideas (see, e.g., discussions in Kuzawa and Pike (2005) and Kuzawa and Sweet (2009)), and a strong following developed among economists, demographers, and numerous medical researchers (Lackland et al. 1999, 2003; Hanson and Gluckman 2008; Skogen and Overland 2012; Barker and Thornburg 2013; Steckel 2013; Lackland 2014). This type of work also engages economic history (Fogel and Costa 1997; Bleakley 2007), environmental economics (Deschenes et al. 2009), and family decision-making (Del Bono and Ermisch

2009; Del Bono et al. 2012). The approach also has the advantage of providing a mechanism for understanding the interconnection with stroke and diabetes.

Mounting evidence suggests that fetuses and infants respond to poor nutrition and stress by compromising organ integrity and degrading biological processes that regulate physiological systems in later life (Gluckman et al. 2008; Barker and Thornburg 2013). Evidence shows that individuals are predisposed to hypertension 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 face an energy-rich diet or elevated stress in later life, pathophysiological processes are set into motion that might significantly increase their likelihood of hypertension.

During their developmental stages, humans are able to accommodate stresses and environmental changes by pleiotropic gene expression patterns that promote survival. The adaptations made to the stressful environment change the structure and function of organs before birth and might be passed on to future generations through epigenetic mechanisms (Aiken and Ozanne 2014). When the fetus is optimized for a lean world, but instead must process a lush load of net nutrition as an adult, there is a mismatch between expectations and reality. This unexpectedly rich nutrition actually proves harmful in certain ways to the individual in the longer term (De Boo and Harding 2006; Swanson et al. 2009). Given this mounting evidence, there is a need for determining the mechanisms that underlie the observation and the generality of the finding for other noncommunicable diseases (Jasienska 2009; Kuzawa and Sweet 2009).

Study of the Helsinki birth cohort, a longitudinal study of 13,517 men and women who were born in Helsinki University Hospital from 1924 to 1944, shows that low birth weight, especially when followed by obesity in early adolescence, is associated with later life hypertension (Barker et al. 2002). Figure 2.2 shows the excess burden of hypertension as a function of these factors. All births of low weight (<3000 g) had elevated odds ratios of adult hypertension, but the risks were greatest (odds = 2.5) for individuals born with weights under 3000 g and a BMI at 11 that exceeded 17.6.

Our goal is not to test or evaluate all of the suggested explanations for elevated hypertension among African Americans, which would be a considerable task, but rather to integrate social science research with medical knowledge to advance the understanding of this puzzle. Our efforts are warranted by the persistence of the puzzle and the lack of generally accepted explanations. With our data and methods, we cannot "prove" that the mechanism of the hypothesis operated through rapidly changing socioeconomic conditions, but we can achieve the important goal of making a plausible case for additional study.

2.3 Creating a Vulnerable Population

The Socioeconomic Transformation Figure 2.3 shows that the American South was relatively poor for several decades following the Civil War. Regional income per capita in New England was roughly three times that of the South (Kim and Margo 2003). Conditions drastically improved in the middle of the twentieth cen-

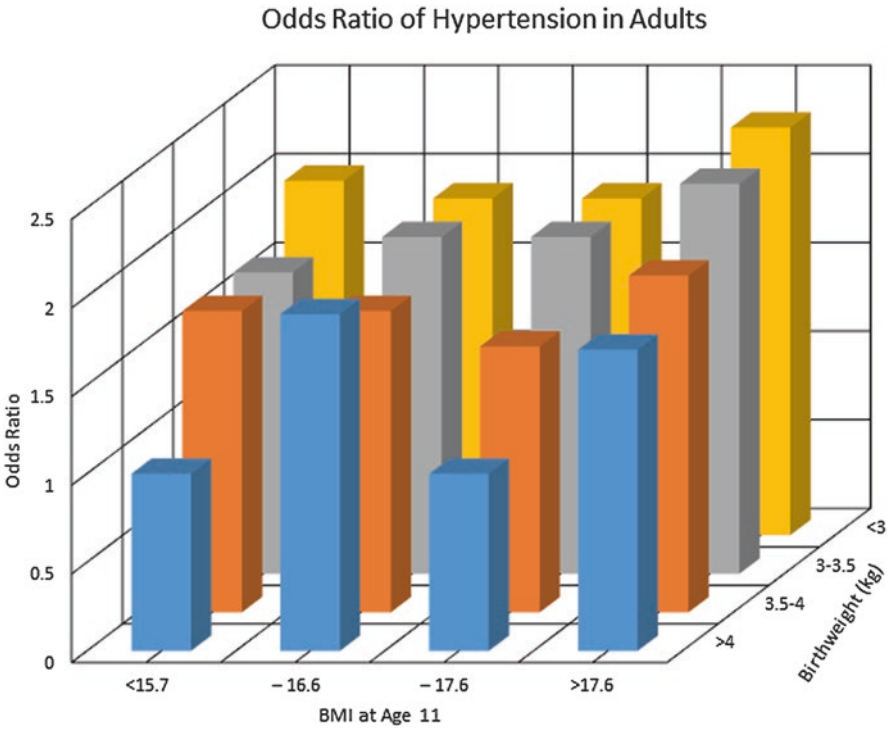

Fig. 2.2 Odds ratios of hypertension in adults as a function of birth weight and BMI at age 11. (Source: Barker et al. 2002)

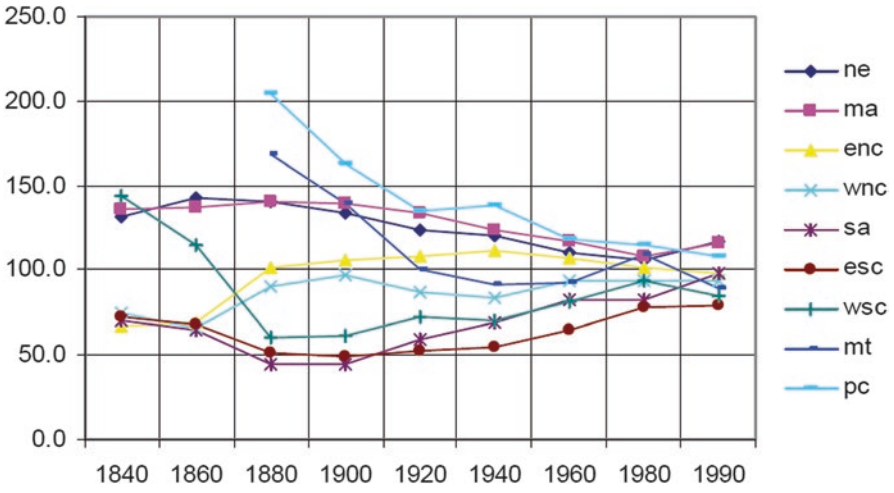

Fig. 2.3 Relative regional income per capita, 1840–1990 (USA = 100). Legend: *ne* New England, *ma* Middle Atlantic, *enc* East North Central, *wnc* West North Central, *sa* South Atlantic, *esc* East South Central, *wsc* West South Central, *mt* Mountain, *pc* Pacific. (Source: Kim and Margo 2003)

tury, as regional industrial structures as well as income per capita converged dramatically. Southern per capita incomes grew significantly faster than the national average (Kim and Margo 2003). This was a remarkable achievement because the quarter century following 1950 was the strongest period for economic growth in the twentieth century. Not only did the South gain relative to the rest of the country in mid-century, but African Americans gained relative to whites. Between 1940 and 1980, the real incomes of white men grew 2.5 times, while that for African Americans grew fourfold (Smith and Welch 1989). As a percentage of white male wages, those for African Americans averaged 43.3 in 1940 and 72.6 in 1980. Opinions differ on the sources of this progress, but schooling, civil rights legislation, and south-north migration were all part of the mix (Heckman 1990; Donohue and Heckman 1991; Margo 1993).

Pointing toward the importance of civil rights legislation in creating new labor market opportunities, the median income of African American men in the South relative to the 25th percentile of southern white men grew by 34% points between 1960 and 1990 (Card and Krueger 1993). Therefore, we argue that southern African American adolescents of the 1960s and 1970s were particularly vulnerable to hypertension as adults because their parents and older ancestors were poor, and this generation realized dramatic improvements in net nutrition beyond an age when biological adaptation to rapidly improving circumstances was limited or impossible. Income growth per se created vulnerabilities, but as discussed below, this variable is also a proxy for many changes affecting the diet, work effort, and lifestyles of the African American population, especially those living in the South. Among these factors was desegregation of hospitals and fair housing laws, which may have had independent, beneficial effects on hypertension.

In our analysis, the developmental origins hypothesis predicts that the children of generationally poor parents who were born under rapidly improving conditions would have higher rates of hypertension as adults. This paper considers how these changes translated into greater prevalence of hypertension for African Americans by relying on the timing of socioeconomic change and its differential impact across states to identify forces that influence this disease. The empirical strategy has acknowledged limitations, but if the hypothesis is powerful, one would expect to find elevated prevalence rates in states and among ethnic groups with this dynamic environmental history.

Earlier work using state-level data has found that long-term poverty followed by rapid economic improvement increased the risk for type 2 diabetes at the state level (Steckel 2013). Given the aforementioned related study and following the developmental origins hypothesis, we suspect that African American families, especially in the South, who were persistently and severely poor until undergoing significant income growth after the middle of the twentieth century will have suffered high rates of hypertension.

Circumstantial evidence suggests an association between income growth and prevalence. Figures 2.4 and 2.5 provide data on the socioeconomic transformation in the South and its relationship to the geographic prevalence of hypertension.

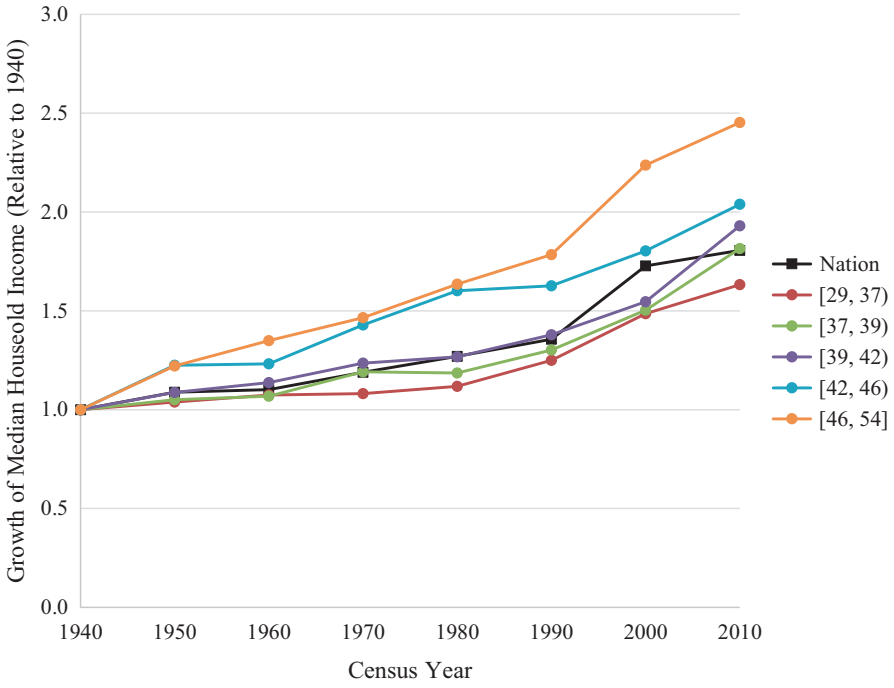

Fig. 2.4 Growth of black median household income by hypertension region (population weighted). (Sources: Census 1940, Census 1950 V2 Detailed Characteristics Table 87 and 56 for AL and HI, Census 1960 V1 Chapter D: Detailed Characteristics Table 133, Census 1970 V1 Chapter D: Detailed Characteristics Table 192, Census 1980 V1 Chapter D: Detailed Characteristics Table 243 and 244 for AL, Census 1990 V1 CP-2 Table 53, Census 2000 Summary File 4, Census 2010 ACS)

Figure 2.4 shows the growth of median African American household income from 1940 to 2010 ranked by the prevalence of hypertension in 2011 and organized by clusters of states having similar levels of prevalence. The group of states having the highest prevalence of hypertension in 2011 were also the states in which income growth was most rapid. For example, in the cluster of states where prevalence was in the range of 46%–54%, median household income grew the fastest, by a factor of 2.45 from 1940 to 2010. Figure 2.5 shows that the states with the highest prevalence rates in 2011 were also the poorest in 1940. These results indicate that rapid growth out of poverty may have triggered the rise in the prevalence of hypertension.

2.4 Controls

We recognize that many variables other than those associated with fetal origins are linked to hypertension, and they must be recognized in the empirical analysis. Among these are smoking, educational attainment, current income, and exercise.

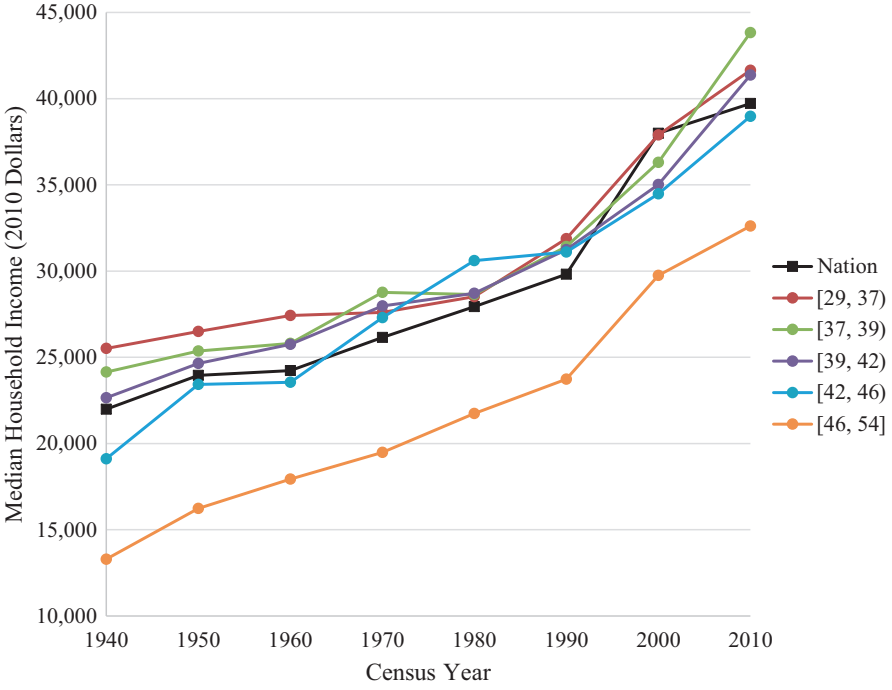

Fig. 2.5 Black median household income by hypertension region (population weighted). (Sources: Census 1940, Census 1950 V2 Detailed Characteristics Table 87 and 56 for AL and HI, Census 1960 V1 Chapter D: Detailed Characteristics Table 133, Census 1970 V1 Chapter D: Detailed Characteristics Table 192, Census 1980 V1 Chapter D: Detailed Characteristics Table 243 and 244 for AL, Census 1990 V1 CP-2 Table 53, Census 2000 Summary File 4, Census 2010 ACS)

Numerous studies identify smoking as a risk factor in cardiovascular disease, although there is some disagreement on the biological pathways (Rhee et al. 2007; Virdis et al. 2010; Gao et al. 2017). Likely suspects are impaired endothelial function, arterial stiffness, inflammation, and lipid modification.

Education and current income may operate through several pathways to lower hypertension (Leng et al. 2015). First, high-income, well-educated people were better informed about the risks and causes of hypertension, and therefore more likely to pursue a healthy lifestyle. Second, the poor and less educated have less knowledge of healthcare facilities, and a greater feeling of helplessness or lack of control over their health situation (Xu et al. 2013).

Exercise obviously affects obesity, but studies show that it has an independent beneficial effect on hypertension (Dimeo et al. 2012; Pescatello et al. 2015; Naci et al. 2018). A meta-analysis of major exercise and drug trials showed that exercise and drug interventions were similarly effective in reducing mortality outcomes for coronary heart disease, and physical activity interventions were actually more effective for the secondary prevention of stroke mortality (Naci and Ioannidis 2013).

2.5 Testing the Hypothesis: Data and Methods

We investigate the strength of the developmental origins hypothesis by analyzing data on individuals collected by the 2011 BRFSS, a cross-sectional telephone survey conducted by state health departments with technical assistance from the CDC. In addition to age and race, this source provides data on education, poverty, smoking, obesity, and patterns of exercise. These variables are defined by answers to the following questions:

- Hypertension: Have you ever been told by a doctor, nurse, or other health professional that you have high blood pressure?
- Smoking: Do you now smoke cigarettes every day, some days, or not at all?
- Education: What is the highest grade or year of school you completed?
- Exercise: During the past month, other than your regular job, did you participate in any physical activities or exercises such as running, calisthenics, golf, gardening, or walking for exercise?
- Obesity: About how much do you weigh without shoes? About how tall are you without shoes? Data used to calculate BMI.

According to the developmental origins hypothesis, one would expect rapid socioeconomic change to have had its greatest impact on adult disease for African Americans when the individuals were children or young adults. Development in utero and early childhood created a thrifty phenotype for them, and rich net nutrition would have challenged their cardiovascular system as adults. To match periods of vulnerability to chronological patterns of economic growth, we divide the BRFSS sample into five cohorts defined by these age groups: 18–34, 35–44, 45–54, 55–64, and 65+. The group aged 65+ in 2011 would have been children or young adults between roughly 1940 and 1970, so for this cohort, we measure economic growth between these 2 years.[2] Similarly, for the group aged 55–64 in 2011, the corresponding years are 1950–1980, and so forth. There is nothing compelling about a 30-year interval, and so we also conduct sensitivity tests for other windows. For a 20-year interval, the oldest age group would have spent birth to early adulthood from roughly 1940 to 1960.

The BRFSS questionnaire provides information on income and poverty, but unfortunately, the data cannot be linked longitudinally because the survey participants differ over time. As an alternative, we use the median income of African American households at the state level, which has limitations because there is heterogeneity within states. Competitive labor markets within states ameliorate the problem but migration complicates it. We adjust nominal income using the Federal Reserve Bank of Minneapolis' Chained-weighted CPI with 1982–1984 as the base year. The variable we use is the ratio of median household income in period 2 to that in period 1.

[2] We would consider measuring growth from an earlier period, say 1930, but unfortunately the median household income data are unavailable prior to 1940.

Our interpretation of the developmental origins hypothesis accepts that rapidly improving socioeconomic conditions that follow persistent poverty create non-harmonious growth and elevate the risk of adult hypertension. Individuals whose families lived in poverty for generations would have been prone to have children whose cardiovascular system would be stressed by rich net nutrition later in life. Under this theory, sudden prosperity would have created nutritional abundance and weight gain, exposing these cohorts of children to greater risk of hypertension. Maternal income has been shown to have a significant impact on birth weight for those infants who are already at high risk hereditarily. However, it is not clear whether income acts as a developmental buffer for low-birth-weight infants as their lives progress. These findings suggest the existence of biosocial interactions between hereditary predisposition and socioeconomic environment matter (Conley and Bennett 2001). The developmental origins concept connects non-harmonious growth trajectories in early life with chronic illnesses of adulthood. As noted in Barker (2002), per capita income can be a proxy for nutritional conditions as discussed below.

We employ a probit regression to model the presence or absence of hypertension of individuals in 2011. In this statistical formulation, the dependent variable takes on the value of 0 or 1 depending upon the hypertensive condition of the individual, with 1 (present) and 0 (absent). The model estimates the probability that an observation with particular characteristics will fall into either the present or the absent category.

Among the explanatory variables, economic change is measured by the ratio of median household income at the state level in period 2 compared to period 1. Period 1 is defined by the approximate year of birth and period 2 by the approximate year in which the individual reached early adulthood. The times can be only approximate because income is measured every decade.

Under the proposed hypothesis, the coefficient on the ratio of income should be positive, large, and statistically significant, but the size of the coefficient would depend upon the timing of the birth cohorts relative to the rate of economic growth. Specifically, the impact would have been greater on individuals who were older when rapid growth occurred because they had less opportunity to adjust.

2.6 Results

The coefficients in the table of results denote the marginal effect of each independent variable on the dependent variable, holding other variables constant. As indicated earlier, the value of the variable rMedian income used in the regression depends upon the age group in which the individual is located. To capture the effect of changing socioeconomic circumstances on hypertension, we measure income growth from the time period of birth to young adulthood.

It is well-known that hypertension increases with age, and for this reason, we include age dummies as regressors that identify birth cohorts. As the age of the

groups declined relative to the period of rapid growth, individuals had greater opportunity to adapt to change, and consistent with this observation, the coefficients on the dummy variables for age declined. Notably the coefficient for the age group 65+ (1.536) was 4.1 times greater than that for the age group 35–44. This result is consistent with our expectation, derived from the developmental origins hypothesis, that individuals who were younger during a time of great change had more opportunity to adapt.

The results for the other coefficients are as expected. Higher median household income in 2010 lowers the prevalence of hypertension because richer households are better able to afford medical care. A related income measure, living in poverty, raises it. Many studies have noted that health improves with the level of education and in our specification people with lower education (high school or less) have a greater prevalence. Several studies also report that the prevalence of hypertension increases with smoking, obesity, and lack of exercise, all of which are confirmed in Table 2.2.

It is reasonable to ask whether this number is large or small. In making this determination, we note that reported hypertension underestimates actual hypertension prevalence, especially among minority groups. A recent study found that fewer than 50% of adults with hypertension controlled their blood pressure in 2007–2008, which approximately doubles the impact on health of the income coefficient in Table 2.2. The coefficient on median income in 2010 is negative and significant in two specifications and is marginally significant in a third one. The direction of the effect (negative) is intuitive because larger current income enables households to better provide healthcare for their children.

The regression results suggest that intergenerational poverty followed by rapid socioeconomic improvement elevates the risk of hypertension, which describes the experience of African American adults born after World War II. This analysis is consistent with developmental origins hypothesis as states with the larger income growth, controlling for other factors, tended to have larger prevalence of hypertension. Below we offer interpretations of the variables that control for current conditions.

Lower education suggests that the individual is less informed about the importance of regular health maintenance or less able to locate resources to assist in obtaining healthcare. In line with established research, we find that the coefficient is positive and statistically significant. Stress associated with poverty can cause hypertension. It is well documented that potential stresses include job, financial, and family distress (Kulkarni et al. 1998). The coefficient is positive and significant; our result is well in line with the documented fact that low-income families tend to have generally poorer health than wealthier families (Marmot 2002).

Medical research has shown that excess body fat is associated with higher levels of hypertension and mortality (Faeh et al. 2011; Zheng et al. 2013). Consistent with this pattern, obese individuals were significantly more likely to be hypertensive in all cohorts. Similarly, exercise reduced the chances of hypertension, although the variable is marginally significant in only one specification.

2.7 Discussion

In recent decades, social scientists and medical researchers have studied the upward trend in obesity rates, and collectively they have put forward several explanations. All begin, however, with some type of energy accounting, i.e., that the growth of calorie consumption outpaces the growth of physical activity. Among the ideas put forward are a rise in the cost of time-intensive, home-prepared meals associated with women working outside the home (Cutler et al. 2003; Hamrick and Okrent 2015), technological change that made work less demanding (Philipson and Posner 2003; Lakdawalla and Philipson 2009), changes in diet featuring processed foods that replaced home-prepared meals (Devine et al. 2006), stress-induced eating created by managing the challenges of socioeconomic change (Torres and Nowson 2007), and the proliferation of fast-food restaurants that conveniently provided calories at low cost (Chou et al. 2004; Schlosser 2012). Many of these arguments apply to African Americans, especially those who lived in the South.

Table 2.1 shows the trend in obesity by race from 1959–1962 to 2015–2016. In all years, obesity rates of blacks exceeded those of whites, and on average were 39% higher. Here we discuss pathways by which the transformation of African American socioeconomic conditions, particularly in the South, ultimately promoted obesity, which in turn contributed to hypertension in a vulnerable population by reducing the physical activity of daily life in an environment of a rich diet and little recreational exercise. The major components of our analysis are the mechanization of agriculture, lack of recreational exercise, the spread of automobiles, women's employment outside the home, and the continuation of a rich diet.

The Mechanization of Agriculture Based on the 1950 Census, agriculture was the dominant industry in the South (U.S. Bureau of the Census 1952). In the swath of states that extend from Texas and Oklahoma to North Carolina, the average share of African Americans employed in agriculture was 31.8%. It exceeded 35% in North Carolina, South Carolina, Arkansas, and Georgia. By 1980, however, the average share in the 11 states had declined to 3.4% and slightly exceeded 5% in only three states—Florida, Arkansas, and Mississippi (U.S. Bureau of the Census 1983).

Relief from field labor came late to the South relative to other regions (Hurt 1989). Mechanization of the harvest was difficult to accomplish in the region's most important crops of cotton and tobacco. Even today, the latter requires extensive hand labor and thus mechanization contributed little to productivity in this crop. Therefore, we focus on the predominant crop, cotton.

A 1939 study of man-hours per acre in cotton production in the Mississippi Delta showed that that vast majority of time (62.9%) was devoted to the harvest, while cultivating, thinning, and weeding the crop absorbed an additional 30.9% (Holley 2000, p.134). Picking cotton by traditional methods required long hours of stoop labor, and unlike grain for which mechanical harvesters had existed for over a century, cotton harvesting faced two challenges: irregularly spaced bolls and bolls that ripened at different times on the same plant (Holley 2000). Development of new

Table 2.1 Trend in obesity rates, white and African Americans

Year	Freq.	% obese AA	% obese white
1959–1962	6672	20.82	13.21
1971–1974	16,730	22.28	13.73
1976–1980	12,520	30.91	20.62
1988–1994	17,752	28.57	21.41
1999–2000	5448	37.77	27.60
2005–2006	5563	43.60	32.04
2009–2010	6527	48.63	33.87
2015–2016	5992	44.77	37.21

Source: CDC. National Health and Nutrition Examination Survey

Table 2.2 Explaining the prevalence of hypertension across individuals within age cohorts with 30-year income gap after birth

	80/50	30 Year Gap
rMedian Income	0.015***	0.022***
	(0.007)	(0.007)
Income 2010	-0.004***	-0.004***
	(0.001)	(0.001)
HS or Less	0.085***	0.086***
	(0.019)	(0.019)
Poverty	0.163***	0.163***
	(0.023)	(0.023)
Smoking	0.072***	0.072***
	(0.019)	(0.019)
Obesity	0.483***	0.483***
	(0.001)	(0.002)
Exercise	-0.004***	-0.004***
	(0.001)	(0.002)
Age 35-44	0.396***	0.374***
	(0.037)	(0.039)
Age 45-54	0.893***	0.862***
	(0.032)	(0.036)
Age 55-64	1.340***	1.302***
	(0.032)	(0.038)
Age 65+	1.589***	1.536***
	(0.034)	(0.043)
N	21,718	21,718
Pseudo R^2	0.2472	0.2473

Notes: Age group 18–34 is the omitted group. rMedian income for the 30-year gap column: 65+ (70/40), 55–64 (80/50), 45–54 (90/60), 35–44 (00/70), and 18–34 (10/80)

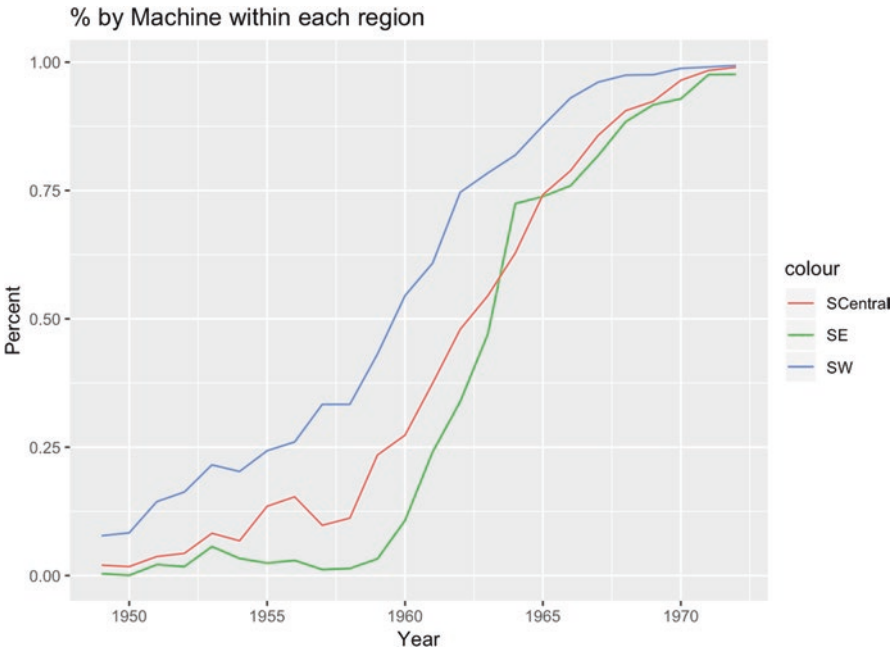

Fig. 2.6 Diffusion of the cotton picker: percent of the crop picked by machine within regions. (Source: US Department of Agriculture (1974), Table 185)

varieties that ripened bolls at about the same time solved the latter, but it took some engineering to build a machine that was effective at removing bolls with little destruction of fibers while also eliminating plant debris.

The diffusion of the cotton-picking machine during the 1950s and 1960s nearly eliminated hand labor in picking by the early 1970s. Figure 2.6 demonstrates the extent of change. In 1950, approximately 5% of the crop was picked by machine and by 1970 the figure had risen to virtually 100%. Although mechanical cotton pickers largely replaced hand labor between the late 1940s and the 1960s, hand methods persisted on small farms for a decade or more (Heinicke and Grove 2008; Logan 2015).

The diffusion of the tractor was a second important change that eased the burden of physical labor in the South (Fig. 2.7). Relative to other regions, farmers were slow to adopt this machine, and mules lingered on small farms operated by older farmers until the 1960s (Ellenberg 2007). Southern customs were fashioned by a long history of physical labor in the fields that welcomed rest at the end of the workday and discouraged work on Sunday. These habits persisted after diffusion of the tractor and the mechanical cotton harvester, thereby adding to weight gain in an environment where people maintained a rich diet and eschewed recreational exercise (Church et al. 2011)

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

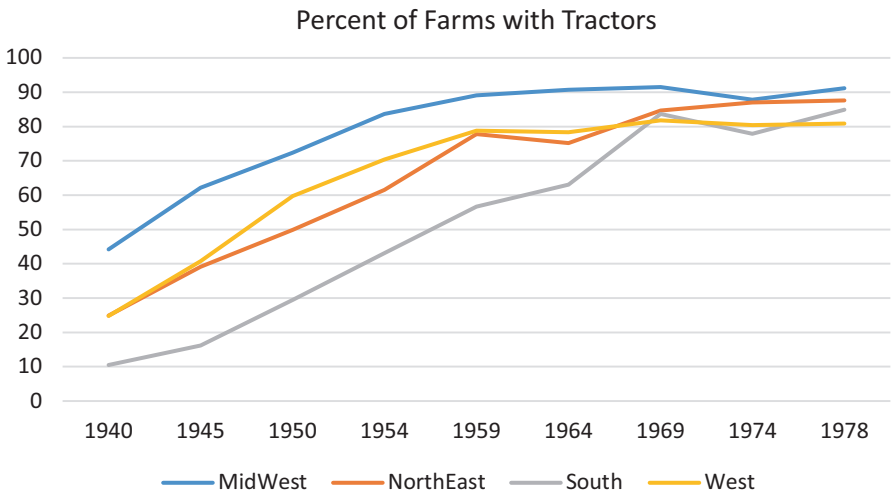

Fig. 2.7 Percent of farms with tractors by region, 1940–1978. (Source: Agriculture censuses of 1945, 1959, 1964, 1974, and 1979)

reduction in physical labor. In 2007, the share of the population belonging to health clubs ranged from a low of 6.3% in West Virginia to a high of 21.8% in Colorado (Active Marketing Group 2007). In every state in the high hypertension region of the South, the share of the population belonging to a health club was below the national average of 15.5%.

Diffusion of Automobiles Rising incomes enabled families to replace walking with a less taxing form of transportation and hauling, the automobile. According to the Federal Highway Administration's National Household Travel Survey, from 1950 to 1980, the number of miles traveled in a personal vehicle per capita increased by more than 2.3 times, while the number of vehicle registrations per capita increased by about 2.5 times for the South. That was by far the biggest increase of any region in the country. Table 2.3 shows that in the mid-1930s, whites were nearly four times more likely than African Americans to own automobiles (59% vs. 15%). With the relatively larger income growth for African Americans, they experienced significantly more growth in car ownership rates as well. By 1970 the ratio was 1.57 and by 1989 it had fallen to 1.25 as nearly 70% of African Americans owned automobiles (Lebergott 2014).

Women's employment outside the home Table 2.4 shows the growing labor force employment of black women with children from 1950 to 1980. The first column gives the ratio of the percent in 1980 to that in 1950 for all industries in each region. In the South, for example, the share employed grew by 43%. All regions registered gains in women's employment from 1950 to 1980.

All regions but the West showed declines in women's employment in agriculture, a sector that featured great strides in mechanization. Important crops in the Pacific

Table 2.3 Percent of US families with automobiles by race

	Race	
Year	White	Black
1935–1936	59	15
1970	83	53
1989	86	69

Source: Lebergott (2014), p. 130

Table 2.4 Trend in employment of black women with children, by region and industry

	Ratio of female labor force participation rate, 1980/1950		
Region	Total 8050	Agriculture 8050	Manufacturing 8050
Northeast	1.32	0.64	4.25
Midwest	1.72	0.83	8.98
South	1.43	0.38	17.66
Southwest	1.80	0.26	22.33
West	1.52	1.13	17.44

Source: US Census

states such as grapes, fruit, and vegetables mechanized somewhat but continued to employ considerable hand labor. Notably, the South in 1980 employed only 38% of the black female labor it did in 1950.

Economic historians know that rapid economic change created many new opportunities but also disrupted family life, as studies of industrialization make clear (Hareven 1982; Tilly and Scott 1989). As southern agriculture mechanized and food became cheaper, farm women joined the labor force, often taking jobs in manufacturing, 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 communing patterns, and so forth, all of which were stressful. By far the largest gains in the employment of women with children occurred in manufacturing. The Southwest led the pack with a gain of over 2,000%, but the South was second at 1,766%. The great shift of women's employment to manufacturing had profound implications for the home economy.

First, consider the growth in the value of employed women's time, which is a key ingredient in the explanation for substitution of restaurant or premade meals from home cooking. The latter was very time-intensive and the switch created a less nutritious diet but was significantly less time demanding. Second, families that had both parents employed outside the home had less opportunity to supervise the eating and leisure habits of their children. These children, whose after-school time was once occupied by field labor or chores around the home, were now free to enjoy more leisure time. Ownership rates of TVs increased over 30-fold for southerners from 1950 to 1970 (Census 1950 and 1970).

The Rich Southern Diet The traditional southern diet was rich in starch, fat, and salt. 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 they were assembled for fattening on corn. Meat processing occurred after the first cold spell, and a massive increase of pork consumption followed. According to USDA Nationwide Food Surveys from 1955, 1965, and 1977, southerners on average consumed 4% more meat, 5% more fats and oils, and 18% more sugar and sweets (by weight) weekly than the national average.

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 pork lard. 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. According to USDA Nationwide Food Surveys in 1977, southerners consumed about 12% less fresh fruits by weight than the national average. This lower consumption pattern still holds. Southern states currently have the highest proportions of adults who self-report consuming one or less fruit and one or less vegetable in any form per day (Centers for Disease Control and Prevention 2013).

The traditional southern diet was a disaster for heart disease when accompanied by a decline in physical labor combined with 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 hypertension prevalence at the county level.

2.8 Conclusions

Heart disease is a major public health problem that causes disabilities and premature death. In an effort to design remedies, numerous studies have investigated causes, leading to recommendations on diet, exercise, cessation of smoking, weight control, careful monitoring of blood pressure and so forth (De Backer 2008). Our goal has not been to displace but to supplement such studies with investigating an early warning to future potential severe cardiovascular problems. 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 the disease is well-advanced, but this problem could be lessened by knowledge of a person's proclivity based on socioeconomic information that many patients could readily provide, such as occupations of the parents and grandparents

and their counties of birth (counties in the USA vary widely in their economic prosperity). Individuals thought to be vulnerable, for example, could be encouraged to engage in healthy behaviors at younger ages and to have regular medical exams.

Our analysis confirms or is at least consistent with the developmental origins hypothesis as applied to heart disease in explaining regional differences in hypertension among African Americans within the USA in 2011. Income growth substantially increased the prevalence of actual (as opposed to undiagnosed or untreated) hypertension. 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 pockets of strong dietary tradition linger in many rural regions, a pattern that offers an opportunity to study hypertension at the county level.

A topic untouched by the evidence analyzed here is the consequence of duration of relative poverty and affluence on hypertension. 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 developmental origins hypothesis has especially relevant implications for the developing world, where vast numbers of poor families are on the verge of experiencing rapid income growth. Chronic adult illnesses, like heart disease, are likely to increase dramatically in the once-poor but now rapidly growing countries (Lopez et al. 2006; Kinsella and He 2009).

We acknowledge that our evidence cannot "prove" that fetal origins mechanisms, working through socioeconomic channels, explain high rates of hypertension among African Americans in the American South. Preferably, we would have panel data on birth cohorts that link family socioeconomic histories with adult health outcomes, but to our knowledge, this evidence is currently unavailable. One of our goals is to stimulate research to acquire such evidence by showing that, despite its flaws, our state-level data are consistent with the hypothesis, and therefore deserves more study.

Acknowledgments The views in this paper are those of the authors and do not reflect those of the Office of the Comptroller of the Currency or the Department of the Treasury.

Appendix: Murray Reflections by Richard Steckel

John Murray entered my graduate economic history class in 1989 and we soon became friends. We had a common educational ancestor in Oberlin College and like me, he came late to the field of economic history as an older student. It was greatly rewarding to see him flourish under the freedom economic history offered for research topics. Virtually, every subfield of economics has a history, and soon he was exploring interdisciplinary topics as well, writing a term paper on the Shakers,

which later turned into a dissertation on the relationship of their living standards to their rise and decline in the century after 1780. John had an important qualification for a good economic historian, a nose for data and delight in discovering useful evidence in the archives. In fact, he and his wife, the epidemiologist Lynn Wellage, spent part of their honeymoon at the restored Pleasant Hill Shaker site. His intellectual curiosity propelled him into research on many topics from anthropometric history, literacy, fertility, the origins of American health insurance, and the Charleston Orphan House. Beyond his substantial research output, John was truly a good citizen, winning teaching awards, serving on editorial boards, helping as referee and book review editor, being trustee of the Cliometric Society, and performing various tasks for the Social Science History Association. As his dissertation advisor, I sometimes wondered if I pushed him too hard as a graduate student, urging him to gather more evidence, conduct additional analyses, and rewrite his work. One day, frustrated with the lack of light at the end of the tunnel, he came into my office and said he had enough. I told him to finish writing and he would be ready to graduate in a few weeks. Several years later, he thanked me for pressing him so hard because it expedited publishing and tenure.

John Murray was one of the good guys in the economic history profession. Friendly, gregarious, approachable, and eager to offer comments and suggestions on research in progress, he had many friends and admirers. I am proud to be among them.

References

Active Marketing Group (2007) Health club industry review, San Diego

Aiken CE, Ozanne SE (2014) Transgenerational developmental programming. Hum Reprod Update 20(1):63–75

Barker D (1990) The fetal and infant origins of adult disease: the womb may be more important than the home. BMJ Brit Med J 301(6761):1111

Barker D (2002) Fetal programming of coronary heart disease. Trends Endocrin Met 13(9):364–368

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

Barker D, Thornburg K (2013) The obstetric origins of health for a lifetime. Clin Obstet Gynecol 56(3):511–519

Barker D, Eriksson JG, Forsen T, Osmond C (2002) Fetal origins of adult disease: strength of effects and biological basis. Int J Epidemiol 31(6):1235–1239

Bleakley H (2007) Disease and development: evidence from Hookworm eradication in the South. Q J Econ 122(1):73–117

Card D, Krueger AB (1993) Trends in relative black-white earnings revisited. Amer Econ Rev 83(2):85–91

Centers for Disease Control and Prevention (2010) A closer look at African American men and high blood pressure control: a review of psychosocial factors and systems-level interventions. US Dept of Health and Human Services, USGPO

Centers for Disease Control and Prevention (2013) State indicator report on fruits and vegetables. US Dept of Health and Human Services, USGPO

Chou SY, Grossman M, Saffer M (2004) An economic analysis of adult obesity: results from the behavioral risk factor surveillance system. J Health Econ 23(3):565–587

Church TS, Thomas DM, Tudor-Locke C, Katzmarzyk PT, Earnest CP, Rodarte RQ, Martin CK, Blair SN, Bouchard C (2011) Trends over 5 decades in US occupation-related physical activity and their associations with obesity. PloS One 6(5):e19657–e19657

Conley D, Bennett NG (2001) Birth weight and income: interactions across generations. J Health Soc Behavior 42(4):450–465

Cooper R, Rotimi C, Ataman S, McGee D, Osotimehin B, Kadiri S, Muna W, Kingue S, Fraser H, Forrester T, Bennett F, Wilks R (1997) The prevalence of hypertension in seven populations of west African origin. Am J Public Health 87(2):160–168

Cutler DM, Glaeser EL, Shapiro JM (2003) Why have Americans become more obese? J Econ Perspect 17(3):93–118

De Backer G (2008) Risk factors and prevention of cardiovascular disease: a review. Dialogues 13(2):83–90

De Boo HA, Harding JE (2006) The developmental origins of adult disease (Barker) hypothesis. Aust NZ J Obstet Gynecol 46(1):4–14

Del Bono E, Ermisch J (2009) Birth weight and the dynamics of early cognitive and behavioural development. IZA Discussion Papers. Bonn, Institute for the Study of Labor, pp 1–30

Del Bono E, Ermisch J, Francesconi M (2012) Intrafamily resource allocations: a dynamic structural model of birth weight. J Labor Econ 30(3):657–706

Deschenes O, Greenstone M, Guryan J (2009) Climate change and birth weight. Am Econ Rev 99(2):211–217

Devine CM, Jastran M, Jabs J, Wethington E, Farell TJ, Bisogni CA (2006) A lot of sacrifices: workfamily spillover and the food choice coping strategies of low-wage employed parents. Soc Sci Med 63(10):2591–2603

Dimeo F, Pagonas N, Seibert F, Arndt R, Zidek W, Westhoff Timm H (2012) Aerobic exercise reduces blood pressure in resistant hypertension. Hypertension 60(3):653–658

Donohue JJ, Heckman J (1991) Continuous versus episodic change: the impact of civil rights policy on the economic status of blacks. J Econ Lit 29(4):1603–1643

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

Faeh D, Braun J, Tarnutzer S, Bopp M (2011) Obesity but not overweight is associated with increased mortality risk. Eur J Epidemiol 26(8):647–655

Fogel R, Costa D (1997) A theory of technophysio evolution, with some implications for forecasting population, health care costs, and pension costs. Demography 34(1):49–66

Fuchs FD (2011) Why do Black Americans have higher prevalence of hypertension? An enigma still unsolved. Hypertension 57(3):379–380

Gao K, Shi X, Wang W (2017) The life-course impact of smoking on hypertension, myocardial infarction and respiratory diseases. Sci Rep 7(1):4330

Geronimus AT, Bound J, Keene D, Hicken M (2007) Black-white differences in age trajectories of hypertension prevalence among adult women and men, 1999-2002. Ethn Dis 17(1):40–48

Gluckman PD, Hanson MA, Cooper C, Thornburg KL (2008) Effect of in utero and early-life conditions on adult health and disease. New Engl J Med 359(1):61–73

Hall JE, do Carmo JM, da Silva AA, Wang Z, Hall ME (2015) Obesity-induced hypertension. Circ Res 116(6):991–1006

Hamrick KS, Okrent AM (2015) The role of time in fast-food purchasing behavior in the United States. In: Evans R (ed) Purchasing food away from home: demand patterns for fast food and full-service. Nova Science Publishers, New York

Hanson MA, Gluckman PD (2008) Developmental origins of health and disease: new insights. Basic Clin Pharmacol 102(2):90–93

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

Heckman JJ (1990) The central role of the South in accounting for the economic progress of black Americans. Am Econ Rev 80(2):242–246

Heinicke C, Grove WA (2008) Machinery has completely taken over: the diffusion of the mechanical cotton picker, 1949 1964. J Interdiscipl Hist 39(1):65–96
Hertz RP, Unger AN, Cornell JA, Saunders E (2005) Racial disparities in hypertension prevalence, awareness, and management. Arch Intern Med 165(18):2098–2104
Holley D (2000) The second great emancipation: the mechanical cotton picker, Black migration, and how they shaped the modern South. University of Arkansas, Fayetteville
Hurt RD (1989) Mechanization. In: Wilson C, Ferris W (eds) Encyclopedia of southern culture. University of North Carolina Press, Chapel Hill, pp 26–27
Jasienska G (2009) Low birth weight of contemporary African Americans: an intergenerational effect of slavery? Am J Hum Bio 21(1):16–24
Jiang SZ, Lu W, Zong XF, Ruan HY, Liu Y (2016) Obesity and hypertension. Exp Ther Med 12(4):2395–2399
Kim S, Margo R (2003) Historical perspectives on US economic geography. National Bureau of Economic Research, Cambridge
Kinsella K, He W (2009) An aging world: 2008. National Institute of Aging, Washington DC
Kulkarni S, O'Farrell I, Erasi M, Kochar MS (1998) Stress and hypertension. Wisc Med J 97(11):34–38
Kuzawa C, Pike IL (2005) Introduction. Am J Hum Bio 17(1):1–4
Kuzawa C, Sweet E (2009) Epigenetics and the embodiment of race: developmental origins of US racial disparities in cardiovascular health. Am J Hum Bio 21(1):2–15
Lackland DT (2004) Fetal and early life determinants of hypertension in adults: implications for study. Hypertension 44(6):811–812
Lackland DT (2014) Racial differences in hypertension: implications for high blood pressure Management. Am J Med Sci 348(2):135–138
Lackland DT, Keil JE (1996) Epidemiology of hypertension in African Americans. Semin Nephrol 16(2):63–70
Lackland DT, Egan BM, Jones PJ (1999) Impact of nativity and race on 'Stroke Belt' mortality. Hypertension 34(1):57–62
Lackland DT, Egan BM, Ferguson PL (2003) Low birth weight as a risk factor for hypertension. J Clin Hypertens 5(2):133–136
Lackland DT, Voeks JH, Boan AD (2016) Hypertension and stroke: an appraisal of the evidence and implications for clinical management. Exp Rev Cardio Ther 14(5):609–616
Lakdawalla D, Philipson T (2009) The growth of obesity and technological change. Econ Hum Bio 7(3):283–293
Lebergott S (2014) Pursuing happiness: American consumers in the twentieth century. Princeton University Press, Princeton
Leggio M, Lombardi M, Caldarone E, Severi P, D'Emidio S, Armeni M, Bravi V, Bendini MG, Mazza A (2017) The relationship between obesity and hypertension: an updated comprehensive overview on vicious twins. Hypertens Res 40:947
Leng B, Jin Y, Li G, Chen L, Jin N (2015) Socioeconomic status and hypertension: a meta-analysis. J Hypertens 33(2):221–229
Logan TD (2015) A time (not) apart: a lesson in economic history from cotton picking books. Rev Black Pol Econ 42(4):301–322
Lopez AD, Mathers CD, Ezzati M, Jamison DT, Murray CJL (2006) Global and regional burden of disease and risk factors, 2001: systematic analysis of population health data. Lancet 367(9524)
Margo RA (1993) What is the key to black progress? second thoughts. In: McCloskey DN (ed) Myths and morals of US economic history. Oxford University Press, New York, pp 65–69
Marmot M (2002) The influence of income on health: views of an epidemiologist. Health Aff 21(2):31–46
McMillen S (1989) No easy time: rural southern women, 1940-1990. In: Hurt RD (ed) The rural South since World War II. Louisiana State University Press, Baton Rouge, pp 59–94
Mozaffarian D, Benjamin EJ, Go AS et al (2016) Executive summary: heart disease and stroke statistics—2016 update. Circulation 133(4):447

Naci H, Ioannidis JPA (2013) Comparative effectiveness of exercise and drug interventions on mortality outcomes: metaepidemiological study. Brit Med J 347:f5577–f5577
Naci H, Salcher-Konrad M, Dias S et al (2018) How does exercise treatment compare with antihypertensive medications? a network meta-analysis of 391 randomised controlled trials assessing exercise and medication effects on systolic blood pressure. British J Sport Med 53(13):859–869
Pescatello LS, MacDonald HV, Lamberti L, Johnson BT (2015) Exercise for hypertension: a prescription update integrating existing recommendations with emerging research. Curr Hypertens Rep 17(11):87
Philipson TJ, Posner RA (2003) The long-run growth in obesity as a function of technological change. Perspect Biol Med 46(3 Suppl):S87–107
Redmond N, Baer HJ, Hicks LS (2011) Health behaviors and racial disparity in blood pressure control in the National Health and Nutrition Examination Survey. Hypertension 57(3):383–389
Rhee MY, Na SH, Kim YK et al (2007) Acute effects of cigarette smoking on arterial stiffness and blood pressure in male smokers with hypertension. Am J Hypertens 20(6):637–641
Schlosser E (2012) Fast food nation: the dark side of the all-American meal. Mariner Books/ Houghton Mifflin Harcourt, Boston
Skogen JC, Overland S (2012) The fetal origins of adult disease: a narrative review of the epidemiological literature. JRSM Short Rep 3(8):59
Smith JP, Welch FR (1989) Black economic progress after Myrdal. J Econ Lit 27(2):519–564
Sowers J, Ferdinand KC, Bakris GL, Douglas JG (2002) Hypertension-related disease in African Americans: factors underlying disparities in illness and its outcome. Postgrad Med 112(4):24–26, 29–30, 33–24 passim
Steckel RH (2013) The hidden cost of moving up: type 2 diabetes and the escape from persistent poverty in the American South. Am J Hum Bio 25(4):508–515
Swanson JM, Entringer S, Buss C, Wadhwa PD (2009) Developmental origins of health and disease: environmental exposures. Semin Reprod Med 27(5):391–402
Taylor JG (1989) Foodways. In: Wilson CR, Ferris W (eds) Encyclopedia of southern culture. University of North Carolina Press, Chapel Hill, pp 613–616
Tilly L, Scott JW (1989) Women, work, and family. Routledge, New York
Torres SJ, Nowson CA (2007) Relationship between stress, eating behavior, and obesity. Nutrition 23(11-12):887–894
U.S. Bureau of the Census (1952) Characteristics of the population, number of inhabitants, general and detailed characteristics of the population. US Government Printing Office, Washington DC
U.S. Bureau of the Census (1983) Characteristics of the population. US Government Printing Office, Washington DC
US Department of Agriculture (1974) Statistics on cotton and related data 1920-1972. US Department of Agriculture, Washington DC
Virdis A, Giannarelli C, Neves MF et al (2010) Cigarette smoking and hypertension. Curr Pharm Des 16(23):2518–2525
Wong MD, Shapiro MF, Boscardin WJ, Ettner SL (2002) Contribution of major diseases to disparities in mortality. New Eng J Med 347(20):1585–1592
Xu LJ, Meng Q, He SW et al (2013) The effects of health education on patients with hypertension in China: a meta-analysis. Health Educ J 73(2):137–149
Zheng H, Tumin D, Qian Z (2013) Obesity and mortality risk: new findings from body mass index trajectories. Am J of Epidemiol 178(11):1591–1599