The U.S. Racial Health Divide: The Role of Differences in Chronic Disease Onset

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Sociological and biomedical fields have established a marked disparity in health outcomes among Blacks and Whites in the U.S. Stress research suggests that differential exposure to stress is a key pathway to the production of racial health inequalities. This study extends research on racial health disparities by testing for racial differences in the onset of chronic disease and assessing whether timing differences are a function of differential stress accumulation by young adulthood. Data are from CARDIA, a prospective cohort study, which has psychosocial and biomarker measures. Continuous-time hazard models were used to analyze the antecedents of two chronic diseases, namely stress, and the timing of chronic disease onset. Results indicate that there are racial differences in the onset of hypertension and diabetes, but the timing difference was not found to be a function of stress, as measured here.

INTRODUCTION

Over the past century, the U.S. has experienced remarkable declines in mortality and morbidity and increases in life expectancy. Despite these *positive* population level health changes, sociological and biomedical researches continue to report disparities in the health outcomes among Blacks and Whites in the U.S. (Do, Frank, and Finch 2012; Engelaer, van Bodegom, and Westendorp 2013). Health disparities refer to differences in mortality, morbidity, and access to care according to socioeconomic status, gender, residence, but especially race (Dressler, Oths, and Gravlee 2005). In the U.S., Blacks consistently experience lower life expectancies (74.7 vs. 78.9 in 2010) (Murphy, Xu, and Kochanek 2013; Hayward et al. 2000) a higher health risk (Green and Darity 2010; Do, Frank, and Finch 2012; Hayward et al. 2000), and greater chronic disease burden (e.g., diabetes, hypertension, and cardiovascular diseases) (Kuzawa and Sweet 2009) compared to Whites. The 4 to 6 year disparity in life expectancy is largely associated with an earlier cumulative deterioration of health, which leads to a large disparity in future health (Shonkoff, Boyce, and McEwen 2009; Murphy, Xu, and Kochanek 2013). Research has also shown that disparities in health between Blacks and Whites to be large and persistent, which is the central enquiry here regarding the onset and cause of this gap.

Research on total life expectancy (TLE) and healthy life expectancy (HLE) has demonstrated that racial and gender gaps in healthy life expectancy seem to be mirroring trends in total life expectancy, such that Blacks continue to experience lower TLE, lower HLE, and a higher health risk, which is increasing over time (Chang, Nocetti, and Rubin 2005; Do, Frank, and Finch 2012; Green and Darity 2010). In essence, estimates of life expectancy has shown that Blacks are more likely to live shorter lives and in a disabled state (Crimmins and Saito 2001). Hypertension and diabetes are two chronic diseases associated with a decrease in live expectancy and more years lived with chronic diseases (Franco et al. 2005; Jagger and Robine 2011; Laditka and Laditka 2006). Therefore, the increasing prevalence of diabetes and hypertension in the U.S. is alarming due to their health effects (Engelaer, van Bodegom, and Westendorp 2013; Kuzawa and Sweet 2009). These chronic diseases are two major causes of cardiovascular diseases, a leading cause of death in the U.S. (Fryar et al. 2010). For instance, those with diabetes are more susceptible to a serious erosion of their quality of life due to microvascular, macrovascular, and neuropathic complications (Boyle et al. 2010). Additionally, being diagnosed with diabetes at middle ages has been associated with a 10 year decrease in life (Gregg, Cheng, and Saydah 2012). Equally, if the treatment of hypertension increased by 10% there could be an annual prevention of 14,000 deaths amongst those aged 25-79 (Yoon et al. 2012). Decreasing the prevalence and impact of these chronic diseases is vital for all groups, but is especially critical for Blacks who have a significantly higher prevalence of diabetes (14.6% vs. 9.9%, between 1999-2006) (Cowie et al. 2009; Fryar et al. 2010) and hypertension (40.4% vs. 27.4%, between 2009-2010) (Fryar et al. 2010; Yoon et al. 2012) than Whites.

Much research has been devoted to examining the effects of diabetes and hypertension on health, mortality and disability at older ages (i.e., risk of cardiovascular disease, TLE, and HLE), but this study aims to elucidate the implications of the timing of onset, especially at younger ages, for future quality of life and for racial disparities in health. This end is furthered by also prospectively analyzing the antecedents of these two chronic diseases, namely stress associated with negative life events, to investigate the origin of differences in the onset of these chronic diseases.

BACKGROUND

Hypertension & Diabetes

In order to understand racial disparities in health, one must first examine societies in which these disparities exist. With respect to the U.S., specific focus is given to the magnitude of the effect of Black and White disparities on overall health disparities (Dressler, Oths, and Gravlee 2005). Between 2009 and 2010 the CDC reported that the morbidity associated with hypertension was 31.9% among the non-institutionalized population over the age of 20 (CDC 2013b), while in 2011 diabetes affected 11.3% of the population was ranked as the 7th cause of death (CDC 2013a). Having either hypertension or diabetes is associated with being at a greater risk of other diseases. For instance, Hypertension is associated with a being at greater risk of having a stroke, kidney disease, and mortality attributable to cardiovascular disease (Thorpe, Brandon, and LaVeist 2008), while diabetes is a leading cause of non-traumatic amputations, kidney failure, and heart disease (CDC 2011). There is a well-documented racial disparity in hypertension and diabetes between Blacks and Whites, which corresponds to a higher prevalence and earlier onset that persists among Blacks (CDC 2011; Thorpe, Brandon, and LaVeist 2008). Therefore, this study examines hypertension and diabetes, two chronic diseases, which are among the greatest contributors to racial health disparities in the U.S. (Wong et al. 2002). These well-documented and persistent disparities have given rise to numerous frameworks to understating the mechanisms underlying this relationship.

Frameworks for Studying Stress

The need for understanding the source of racial health disparities is essential for facilitating accountability and encouraging action to reduce disparities (CDC 2013a). The frameworks researchers have used to study race-related health disparities will describe in brief, which include: the racial-genetic model, health-behavior model, socioeconomic model, psychosocial stress model, and the life-course development framework. First, the racial-genetic model emphasizes differences in the distribution of genetic variants and a propensity for poor health associated with this distribution as an explanation of disparities. For example, the "slavery hypothesis" suggests that Africans with a salt-sparing genetic variant were selected for such that under the conditions of slavery and the extreme sodium deprivation and high mortality during the Middle Passage, Africans who survived and made it to the New World were highly selected on the salt-sparing genetic variant. It is argued that the selection of the salt-sparing genetic variant among African Americans survivors, who now lived in a place where salt was plentiful, made them more susceptible to and at greater risk of developing hypertension (Grim and Robinson 1996).

Second, the health-behavior model emphasizes differences in individual health behaviors (physical activity, smoking, drinking) as the primary explanation for health differences between racial groups. Research in the medical sociology literature has found that health behaviors contribute to disease risk. For example, the lack of physical activity is inversely associated with health, alcohol consumption is associated with some cancers and liver cirrhosis, and smoking is associated with cardiovascular disease and cancer, therefore disparities are attributed to racial differences in health promoting behaviors (Adler, Boyce, and Chesney 1994). Although health behaviors contribute to disease risk, there is little evidence that health behaviors alone or in combination account racial health disparities (Dressler 1993).

Third, the socioeconomic model of health disparities holds that racial health disparities are confounded with SES disparities in health. Here, this premise is that the association between race and SES, such that Blacks are overrepresented in lower SES groups, controlling for SES will reveal the true effect of race or cause racial health disparities to dematerialize. Fourth, the psychosocial stress model highlights the stress experienced by minority groups, especially associated with the experience of racism and discrimination, as the dominant explanation of racial health disparities. The psychosocial stress model primarily focuses on stress associated with institutional racism, perceived racism, living in areas of high socioecologic stress (i.e., low-income communities), and negative affect (e.g., depression). Fifth and last, the Life-Course Health Development Framework (LCHD) contends that health and mortality are produced across the life-course and that health trajectories are reflective of developmental processes (biological, psychological, and social) happening across the lifespan through continuous interactions between biological, social, economic, genetic, environmental, and behavioral contexts to influence individual and population level health (Halfon and Hochstein 2002; Mishra, Cooper, and Kuh 2010). Similarly, different health trajectories are posited to be the result of the accumulation early experiences, protective and risky, on health that occurs through biological programming during critical and sensitive periods (Halfon and Hochstein 2002), where repeated psychologically and physically *stressful* experiences aggregate over the life-course, therefore diseases experienced in adulthood are hypothesized to be a result of acute events that took place during these critical and sensitive developmental periods (Taylor 2010).

The Life-Course Health Development Framework (LCHD) has helped to shape the discourse on the development of health across the lifespan (Halfon and Hochstein 2002). Research from the LCHD framework has shown that health trajectories are reflective of developmental processes (biological, psychological, and social) happening across the lifespan through continuous interactions between biological, social, economic, genetic, environmental, and behavioral contexts to influence individual and population level health (Halfon and Hochstein 2002; Mishra, Cooper, and Kuh 2010). Additionally, different health trajectories are the result of accumulation early experiences, protective and risky, on health. The accumulation of early experiences occurs through biological programming during critical and sensitive periods (Halfon and Hochstein 2002), where repeated psychologically and physically *stressful* experiences aggregate over the life-course, therefore diseases experienced in adulthood are hypothesized to be a result of acute events that took place during these critical and sensitive developmental periods (Taylor 2010).

Differential stress exposure across the life-course may in part explain racial differences in health (Biological Stress Perspective). Some studies argue that social forces such as stress and economic disadvantage are the driving factors in racial health disparities (Kuzawa and Sweet 2009). For example, the lived realities of Black mothers, who often times live in segregated communities, poverty stricken environments, and encountering racial discrimination, are expressed in the health of their children as we see Blacks having higher rates of low birth weight babies, pre-term delivery, and poorer birth outcomes (Kuzawa and Sweet 2009). It is argued that structural and cultural barriers impede Black's ability to live a healthy lifestyle, obtain quality medical care, and avoid chronic stressors associated with disease (Kuzawa and Sweet 2009).

Racial disparities in health may materialize due to differential exposure to stress. Stress, associated with major life events or environmental/social burdens, is associated with the activation of psychological and biological responses and repeated and prolonged activation of these responses can be deleterious to individual health (Lantz et al. 2005; Keller et al. 2012; McEwen 2001). Stress is a physiological response to social, economic, and environmental conditions that "tax or exceed the adaptive capacity of an organism resulting in psychological and biological changes that may place persons at risk of disease" (Cohen et al., 1997; see also Hayward et al., 2000; Schulz et al., 2000; Thoits, 2010). The stress response to these conditions can be adaptive or maladaptive, and the latter is associated with an increased risk of morbidity and mortality through what is termed allostatic load (Hayward et al. 2000; McEwen 2001). An adaptive stress response is one where the body perceives a threat and activates a flight/fight response. This may result in the release of adrenaline, noradrenaline and cortisol, which increases heart rate, blood sugar levels, breathing, diversion of blood flow from non-essential organs and a suppression of the immune system until the threat is removed—allowing the body to return to normal. Conversely, responses to stress may be maladaptive when stress is chronic (persistent)

and does not allow for the body to achieve homeostasis, therefore leading to dysfunctional stress responses, wear and tear on the body, and an inefficient management of stress, better known as allostatic load (McEwen and Seeman 1999; Keller et al. 2012), which is associated with poor physical health (McEwen 2001). The activation of the fight/flight response in reaction to stress supersedes the normal function and regulation of the organs and immune system, which are essential to the maintenance and regulation of biological systems. We propose that the racial health disparity is associated with differential exposure to stress which exposes one group to more frequent and prolonged activation of adaptive stress responses and therefore increased disease risk.

Measurement of Stress

Previous research on stress and health gives support to the argument that stress proliferates across the life-course and facilitates the widening of health gaps between privileged and disadvantaged groups (Thoits 2010). Research measuring the proliferation of stressful life experiences on health has a long history of measuring the association between stress and health using stress checklists or indices. Thomas Holmes and Richard Rahe (1967) helped to accelerate the measurement of stress using checklists after creating the Social Readjustment Rating Scale, a life event checklist, which measured social stressors to test the hypothesis that multiple adjustments for *major life events* in a short period of time could over tax a person's ability to cope or adjust (T. Holmes and Rahe 1967). This checklist was found to have a modest to weak association between increased number of stressful events and health; the major criticism was that a large proportion of the events were either symptoms or consequences of illness. Researchers studying stress and health have subsequently developed more comprehensive measures of stressors that better explain illness. In 1994, Wheaton developed stress checklist which included 51 chronic stressors that also included an additional category, which had been previously neglected in prior stress scales, traumas due to psychological and physical threats to a person's well-being. Thereafter, Dohrenwend (1974) designed the Psychiatric Epidemiology Rating Interview (PERI) Life Events Scale. This measure improved upon previous scales by asking about the recent occurrence of 102 events that tapped multiple life domains including school, work, love and marriage, having children, family, residence, crime and legal matters, finances, social activities, miscellaneous, and health(Dohrenwend and Dohrenwend 1974; Dohrenwend et al. 1978). By and large, a substantial weight of evidence indicates that exposure to stressful life events, as measured through the use of checklists, is associated with a greater risk of poor physical health such as poorer overall health, greater incidence of hypertension and diabetes, arthritis, and cancer (Jemmott and Locke 1984; T. H. Holmes and Masuda 1973).

Research Aims

The aim of this study is to extend research on the racial health divide by testing for racial differences in the onset of chronic disease, demonstrating how timing differences in onset contributes to health disparities in adult prevalence rates, and assessing whether timing differences are a function of differential stress accumulation by young adulthood. This study examined the onset of early adulthood chronic diseases and its implications for future quality of life by answering the following two questions: 1) Are there racial differences in the onset (timing) of two early adult chronic conditions, hypertension and diabetes, and if so, what are the implications for racial differences in health?; 2) Is the origin of this timing difference in part a function of differential stress accumulation? This study follows respondents prior to middle ages, where most studies examine these conditions, thus being able to follow respondents prior to the life stage where most people have already experienced the onset of chronic conditions, which excludes fewer respondents.

METHODS

Model estimates are based on data from the Coronary Artery Risk Development in (Young) Adults (CARDIA) study, a prospective cohort study of more than 5,000 participants from Alabama, Illinois, California, and Minnesota. At the time of recruitment, in 1985 to 1986, respondents were aged 18-30 years of age and were recruited to achieve a balance by race (Black & White), sex (men & women), age (18-24 & 25-30), and

education (less than high school education & more than high school education). A more comprehensive description of the CARDIA study has been described fully elsewhere (Friedman and Cutter 1988; Hughes et al. 1988).

Respondents were reexamined from baseline at years 2, 5, 7, 10, 15, 20 and 25 with a response rate of the surviving cohort of 91%, 86%, 81%, 79%, 74%, 72%, and x% (Matthews et al. 2004; Holvoet et al. 2008). The analytic sample was based on 4,366 of the 5,112 baseline participants, with respondents excluded if they had incomplete data on stressful life events, hypertension, diabetes, age, education, race, gender, and BMI (N = 512). Respondents who reported a pregnancy were also excluded (N = 237).

Study Variables

The CARDIA study has data on sociodemographic measures (education, age, sex) and health indicators (blood pressure, physical activity, medical histories, and stressful life events); the longitudinal nature of the data allows for the measure of change across time. Moreover, self-reports of chronic diseases are associated with an underreporting of the prevalence (Fryar et al. 2010), but CARDIA allows for improved measurement of disparities because of the data on reported and measured biomarkers.

Resting blood pressure (BP) was measured 3 times on the right arm with respondents in a seated position at each follow-up using a random zero sphygmomanometer, after an initial resting period of 5 minutes. Each measurement was taken 1 minute apart with final levels reflective of an average of the 2^{nd} and 3^{rd} readings (Matthews et al. 2004). Respondents were considered hypertensive if their systolic blood pressure (SBP) was greater than or equal to 140 mmHg, if their diastolic blood pressure (DBP) was greater than or equal to 90 mmHg, reported being on hypertensive medications, or if they self-reported having hypertension. Respondents had their fasting glucose levels checked at each survey year and were considered diabetic if their fasting glucose level was greater than or equal to 126 mg/DL, or self-reported having diabetes (Lee 2003).

Standardized questionnaire were administered to assess age (years), race (Black & White), sex (male & female), and years of education at each examination year, which was recoded as less than high school graduate, high school graduate, some college, and college graduate and beyond. These measures were verified at year 2 and are used in all analysis. Height was measured without shoes to the nearest 0.50cm and body weight was measured in light clothing to the nearest 0.2 pounds. BMI was then computed as weight in kilograms divided by height in meters squared (kg/m^2). The experience of external stressful life Events was measured at baseline and year 2 by 102 events on the Psychiatric Epidemiology Research Interview (PERI) Life Events Scale and used to create a Stressful Life Events Score (SLES) where the 102 events were assigned a weight representing its stress magnitude. A sub-scale of 64 items was chosen by the CARDIA Psychosocial Working group and further reduced to 50 stressful live events linked to educational attainment, family formation, career development, etc. (e.g., respondent fired, respondent divorced, and death of respondent's close friend). These items were weighted and summed to create an interval level measure of stressful life events. The measure was then recoded and measured in quartiles (Baseline quartile ranges: 232-1416 (Q1), 1417-2175 (Q2), 2176-3150 (Q3), 3151-11414 (Q4); Year 2 quartile ranges 232-1134 (Q1), 1135-1828 (Q2), 1829-2368 (Q4), and 2639-7349 (Q4)).

Data Analysis

First, descriptive statistics were calculated on CARDIA Study data (Table 1). Incidence rates (densities) were calculated as the incident number of disease onsets divided by the sum of the person time at risk, measured as the time from baseline to the exam at which the chronic disease occurrence was first reported or the last follow-up exam. Hypertension and diabetes were assumed to be present at the first exam it was recorded; therefore events could only occur at 2, 5, 7, 10, 15, and 20 years of follow-up (Lee 2003; Matthews et al. 2004). Respondents were censored at pregnancy or last observation.

Parametric regression survival-time models, with time modeled continuously and duration of survival as the dependent variable, were used to assess the association of stressful life events in relation to the risk of two chronic diseases, hypertension and diabetes, adjusting for the covariates that may affect survival time age, sex, race, education, and BMI. Chronic disease risk was modeled progressively using more complex regression models and in each successive model covariates were grouped as follows: Model 1—measure of race only, Model 2—measure of BMI only, Model 3—measures of both race and BMI, Model 4—demographic variables, Model 5— BMI and demographic variables, Model 6—race, BMI, and demographic variables, Model 7—race, BMI, demographic variables, and a measure of stress, and Model 8—race, BMI, demographic variables, a measure of stress, and an interaction term between race and stress. The follow-up period was 20 years, the earliest age at which respondents could be considered at risk was 18 years, and survival analyses were performed on each year at risk for each respondent. This analysis gives an opportunity to prospectively analyze the antecedents of two chronic diseases, namely stress, in order to expose the implications of the timing of the onset of chronic diseases for future quality of life and for racial disparities in health. Moreover, investigating the association between disease onset and race will provide further direction as to where to focus programs and policies to help ameliorate racial health disparities.

Hazard models were used to calculate multivariate-adjusted hazard ratios in separate models for hypertension and diabetes. In line with previous research, a Weibull survival distribution, with a hazard that increased with time, was chosen, and values of $P<0.05$ were considered statistically significant (Matthews et al. 2004). Finally, the association between age, race, sex, education, BMI and the outcomes were assessed to examine if the results were modified by baseline and year 2 stressful life event scores. All analysis were conducted using Stata13/MP for Windows (StataCorp 2013).

RESULTS

Table 1 describes the means and standard deviations, at baseline, for the covariates used in the hazard models. All of the covariates are time-varying except race and gender, which are based on information collected at baseline and verified at the year 2 follow-up. On average, Black respondents were slightly younger (24.3 vs. 25.5), had lower levels of educational attainment, and higher BMI's (25.3 vs. 23.6) than Whites. Additionally, Blacks' had lower fasting glucose's, higher systolic blood pressure (SBP), and diastolic blood pressure (DBP) comparable to Whites. Over the course of 20 years of follow-up, 1,046 Blacks became hypertensive and 470 became diabetic, compared to 598 and 297 Whites, respectively; the overall incidence densities of hypertension and diabetes were 19.2 (1,644 cases) and 12.8 (767 cases) per 1000 person-years, correspondingly.

The first question to address is whether the time to onset of hypertension and diabetes typically vary in duration by race. Results will first be reported for hypertension. The overall median survival time across 20 years of follow-up (i.e. the time when half of the respondents are expected to be hypertensive) was 32 years, but the median survival time for Black respondents was 29 years; since only 42 percent of White (Black = 66%) respondents reported hypertension a median survival time could not be calculated. Figure 1a highlights the difference in the survival curves for Blacks and Whites that is indicative of Blacks having a greater risk of hypertension that persists and increases across time. In fact, the Log-rank test for equality of Black and White survivor functions confirms the visual evidence that there is indeed a difference in survivorship (i.e. the time without hypertension). Looking at the results for diabetes yields a slightly different story. During the 20 years of follow-up only 20% of Whites and 29% of Blacks reported being diabetic and the Log-rank test confirmed a difference in survivorship between the two groups (Figure 2a). To examine whether differences in timing or duration vary by race, an event history analysis was conducted in which risk of chronic disease was predicted from stress, controlling for age, education, sex, and BMI. In this context, a hazard ratio above one indicates an increased risk of chronic disease or that the measure caused the respondent to progress more quickly. In contrast, a hazard ratio below one indicates a decreased probability of chronic disease risk and hence a longer duration free of disease.

Results from this analysis for hypertension are presented in Table 2. Respondents, who were older, male, more educated, and had a BMI greater than 30 were at greater risk of hypertension. Black respondents had a 63% greater probability of developing hypertension (Model 7), but in order to test the hypothesis that the relationship between race and the survival time without hypertension differed by the amount of reported stress, an interaction term between race and stress was included in Model 8. The interaction term lacked significance, which indicated that the effect of racial group categorization on hypertension did not differ by levels of stress. Results for diabetes are presented in Table 3. Differences in timing duration of diabetes are reported in Table 3. Respondents who were older female, had a college degree or more, or who had a BMI greater than 25 were at a higher risk of developing diabetes sooner. Additionally, Black respondents had a 27% greater probability of developing diabetes earlier. Once more, the interaction term between race and stress failed to reach significance, which indicated that the effect of racial group categorization on progression to diabetes did not differ by level of stress. The measures of hypertension and diabetes used for the results in Table 2 and Table 3 are more inclusive estimates in that they account for biological markers (SBP and DBP), self-reports of hypertension and diabetes, and self-reported use of medication for chronic disease control, as opposed to relying solely on self-reports of hypertension and diabetes. Figures 1b and 2b illustrate what the survivorship curve would have looked like in the absence of biomarker data. The sole use of self-reports of the outcome measure are not expected to not fully capture the pervasiveness of the condition and the disparity in prevalence between racial groups. In fact, Figures 1b and 2b reflect greater survivorship, a lower hazard, and under estimate of disease prevalence than an outcome that includes biological markers of disease (Table 2s & Table 3s).

DISCUSSION/CONCLUSION

Racial disparities in health persist across all health domains. Increasing our understanding of the relationship between race and chronic disease is amplified by the fact that a good long life is indeed a reality for some racial groups but a hypothetical construct for others (Hayward et al. 2000). By middle-ages (51-61) Blacks already have higher prevalence of hypertension and diabetes along with a myriad of other diseases—this points toward higher incidences of these diseases at younger ages (Hayward et al. 2000). The aim of this study was to test for racial differences in the onset of two chronic diseases, hypertension and diabetes, in order to demonstrate how differences in chronic disease onset contribute to adult health disparities, while also assessing whether timing differences were a function of differential stress accumulation. Results pointed to two key findings: (1) support was found for racial differences in the onset (timing) of hypertension and diabetes, and (2) there was no support found for the origin of this timing difference being in part a function of differential stress accumulation. The use of a prospective cohort was important for investigating the antecedents of these two chronic conditions and the origin of differences in disease onset through the use of survival modes, a method that speaks to the larger literature on health disparities. Furthermore, the use of biomarker data allowed for a more inclusive versus restrictive approach to measuring hypertension and diabetes. This approach more accurately captures the prevalence of these chronic diseases, by gaining those who have undiagnosed hypertension or diabetes, versus relying solely on self-reported health status.

Hypertension, commonly referred to as high blood pressure, varies by age, gender, and race. In the U.S., Blacks are more likely to develop high blood pressure and at younger ages, and likewise Black women are more likely to have high blood pressure than Black men. Findings from the CARDIA Study support previous research in that they reproduce these common associations between race and chronic disease risk, where respondents who were Black, male, older, had higher levels of BMI, and lower levels of education had a higher risk of an earlier development of hypertension. It is important to highlight that high blood pressure increases your chances of a stroke, first heart attack, chronic heart failure and kidney disease (Fryar et al. 2010; CDC 2013b). We have shown that Blacks experience hypertension sooner than Whites so it would stand to reason that other studies have demonstrated that Blacks have a higher risk of these poor health outcomes, that are associated with hypertension and diabetes, and that racial gaps in health mirror disparities in TLE and HLE trends, such that Blacks have a higher health risk that is increasing over time (CDC 2013a).

Analysis for diabetes yielded similar results. Respondents, who were Black, less educated, female, older, and had a BMI greater than 25 were at greater risk of developing diabetes quicker. Diabetes is a serious disease that is a major cause of heart disease and stroke and diabetics have a 2 to 4 times higher mortality rate associated with heart disease and stroke than non-diabetics. Diabetics have an increased rate of death that is 2 times higher than non-diabetics, and while diabetes is the 7th leading cause of death it is also the leading cause of adult blindness, kidney failure, and non-traumatic amputation. Moreover, diabetics also have a greater susceptibility to other illness such as the pneumonia and the flu (CDC 2011). Furthermore, diabetes and the resultant complications from the condition are more susceptible to a serious erosion of their quality of life and to more years lived in a disabled state.

The literature on stress and health suggests that differential exposure to stress is a key pathway to the production of gender, racial, and SES health inequalities (Thoits 2010). In the CARDIA Study, stress was measured using the PERI Stressful Life Events Scale, which was not associated with the earlier onset of chronic illness. Although this scale is an improved measurement of stressful life events, over prior scales, an observed association could be lacking for a number of reasons. First, the stress theory focuses on how the accumulation of stress impacts health due to the nature of the stress response. Over time the stress response negatively affects health through the repeated activation of biological and physiological systems as a result of chronic stress. The continual activation of these systems does not allow the body to achieve homeostasis, which leads to a wear and tear and an inefficient management of stress. The expectation was that higher stressful life event scores would have been associated with an increased chronic disease risk, but stressful life events were only measured at baseline and year two, which does not efficiently asses the cumulative nature and effect of stress across the 20 years of follow-up. The stress scale also included both positive and negative stressful events. Although change itself and the associated readjustment that takes place can be stressful, negative events are reasoned to have a greater impact on health outcomes. Consequently, the effect of stress could have been mitigated by the inclusion of clearly positive events (Sheldon Cohen, Ronald C. Kessler, and Lynn Underwood Gordon 1997). The CARDIA Study also included measures of discrimination and C- reactive protein in later years of follow-up; it would be worthwhile in future studies to pursue the creation of a composite stress measure to better tap into the cumulative nature of stress.

To conclude, research examining the relationship between race and health abound and consistently finds that Blacks in the U.S. have a shorter expectation of life (Murphy, Xu, and Kochanek 2013; Hayward et al. 2000) and greater morbidity (Green and Darity 2010; Do, Frank, and Finch 2012; Kuzawa and Sweet 2009; Hayward et al. 2000) compared to Whites. Health disparities research helps facilitate the understanding of the mechanisms that produce inequalities in an effort to promote accountability and encourage action to reduce these modifiable disparities. The life-course approach taken here is an effort to understand racial health disparities in terms of social inequalities or stressors that chronically affect Blacks in the U.S. across the life-course and the role their role in facilitating an earlier onset of chronic diseases.

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Table 1. Descriptive Statistics at Baseline

Characteristics	Black (n=2,137)	White (n= 2,229)	Total (n=4,366)
Demographic Variables			
Age, y	24.3 ±3.7	25.5 ±3.3	24.9 ±3.6
Education			
Less Than High School, n (%)	271 (68.1)	127 (31.9)	509
High School Graduate, n (%)	785 (63.0)	462 (37.0)	1,522
Some College, n (%)	790 (54.6)	657 (45.4)	1,690
College Graduate & Beyond Graduate, n (%)	291 (22.8)	983 (77.2)	1,391
Male, n (%)	932 (47.0)	1,049 (53.0)	1,981
Female, n (%)	1,205 (50.5)	1,180 (49.5)	2,385
Health Behaviors			
BMI	25.2 ±5.4	23.6 ±3.8	24.4 ±4.7
Clinical Variables			
SBP, mm Hg	111.3 ±11.0	109.2 ±10.8	110.2 ±10.9
DBP, mm Hg	68.8 ±9.9	68.3 ±9.1	68.5 ±9.5
Fasting Glucose (mmol/l)	82.0 ±18.0	83.0 ±12.3	82.5 ±15.5
Psychosocial Measures of Stress			
Stressful Life Events Score, Baseline	2,619.1 ±1534.1	2,209.7 ±1195.8	2,410.1 ±1386.9
Stressful Life Events Score, Year 2	2,103.5 ±1205.2	1,924.0 ±1096.5	2,011.7 ±1154.2
Outcomes			
Incident Hypertension, n (%)	1,046 (63.6)	598 (36.4)	1,644
Incident Diabetes, n (%)	470 (61.3)	297 (38.7)	767
Hypertension Cumulative incidence (per 1,000 person-years)	24.8	12.8	19.2
Diabetes Cumulative incidence (per 1,000 person-years)	9.6	6.4	8
Total Number of Observations	11,023	12,096	23,119
Total Person Years at Risk of Hypertension	42,244	43,300	85,544
Total Person Years at Risk of Diabetes	48,885	46,793	95,678

Table 2. Adjusted Hazard Ratios (95% CIs) for Cumulative 20-Year Hypertension Incidence: CARDIA Study 1985-2005

	Model 1	**Model 2**	**Model 3**	**Model 4**	**Model 5**	**Model 6**	**Model 7**	**Model 8**
Black	2.174**		1.832**			1.631**	1.631**	2.053**
	(13.67)		(10.34)			(8.03)	(7.99)	(4.87)
BMI: 17-18.5		0.581	0.660		0.619	0.678	0.672	0.664
		(1.40)	(1.09)		(1.25)	(1.02)	(1.04)	(1.08)
BMI: 18.5-25		1.868	1.629		1.775	1.610	1.627	1.644
		(1.58)	(1.25)		(1.47)	(1.22)	(1.25)	(1.28)
BMI: 25-30		1.053	1.043		1.038	1.035	1.035	1.035
		(1.41)	(1.17)		(1.01)	(0.94)	(0.94)	(0.93)
BMI: >30		0.898**	0.907**		0.906**	0.909**	0.909**	0.910**
		(4.44)	(4.05)		(4.05)	(3.94)	(3.93)	(3.89)
Age				0.000**	0.000**	0.000**	0.000**	0.000**
				(40.67)	(40.73)	(40.67)	(40.66)	(40.65)
Age Squared				2.165**	2.166**	2.162**	2.162**	2.162**
				(38.83)	(38.89)	(38.81)	(38.80)	(38.79)
Age Cubed				0.994**	0.994**	0.994**	0.994**	0.994**
				(36.80)	(36.85)	(36.77)	(36.76)	(36.74)
Sex				0.955	0.903	0.875*	0.876*	0.869*
				(0.84)	(1.78)	(2.34)	(2.31)	(2.44)
Less Than High School				1.000	1.000	1.000	1.000	1.000
High School Graduate				0.708**	0.694**	0.701**	0.701**	0.701**
				(3.24)	(3.43)	(3.34)	(3.31)	(3.31)
Some College				0.624**	0.602**	0.618**	0.619**	0.619**
				(4.48)	(4.81)	(4.55)	(4.52)	(4.51)
College Grad & Beyond				0.396**	0.451**	0.529**	0.531**	0.534**
				(8.59)	(7.35)	(5.78)	(5.72)	(5.66)
Baseline Life Event Score-Quartile 1							1.000	1.000
Baseline Life Event Score-Quartile 2							0.975	1.156
							(0.32)	(1.17)
Baseline Life Event Score-Quartile 3							1.033	1.092
							(0.41)	(0.68)
Baseline Life Event Score-Quartile 4							1.046	1.210

							(0.55)	(1.42)
Year 2 Life Event Score-Quartile 1							1.000	1.000
Year 2 Life Event Score-Quartile 2							1.031	0.998
							(0.39)	(0.01)
Year 2 Life Event Score-Quartile 3							1.077	1.193
							(0.94)	(1.41)
Year 2 Life Event Score-Quartile 4							0.968	1.088
							(0.40)	(0.62)
Black* Baseline Life Event Score-Quartile 1								1.000
Black* Baseline Life Event Score-Quartile 2								0.748
								(1.79)
Black* Baseline Life Event Score-Quartile 3								0.909
								(0.59)
Black* Baseline Life Event Score-Quartile 4								0.799
								(1.33)
Black* Year 2 Life Event Score-Quartile 1								1.000
Black* Year 2 Life Event Score-Quartile 2								1.051
								(0.31)
Black* Year 2 Life Event Score-Quartile 3								0.847
								(1.03)
Black* Year 2 Life Event Score-Quartile 4								0.836
								(1.06)
	19,600	19,600	19,600	19,600	19,600	19,600	19,600	19,600

* $p<0.05$; ** $p<0.01$

Table 3. Adjusted Hazard Ratios (95% CIs) for Cumulative 20-Year Diabetes Incidence: CARDIA Study 1985-2005

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
Black	1.812**		1.407**			1.279**	1.269**	1.132
	(7.17)		(3.93)			(2.72)	(2.62)	(0.61)
BMI: 17-18.5		0.693	0.740		0.926	0.962	0.973	0.965
		(0.65)	(0.54)		(0.14)	(0.07)	(0.05)	(0.06)
BMI: 18.5-25		1.331	1.241		1.037	0.994	0.981	0.991
		(0.50)	(0.38)		(0.06)	(0.01)	(0.03)	(0.02)
BMI: 25-30		1.313**	1.304**		1.273**	1.270**	1.274**	1.272**
		(4.78)	(4.66)		(4.19)	(4.16)	(4.21)	(4.18)
BMI: >30		0.876**	0.881**		0.862**	0.865**	0.863**	0.863**
		(3.75)	(3.60)		(4.17)	(4.09)	(4.16)	(4.16)
Age				0.000**	0.000**	0.000**	0.000**	0.000**
				(26.97)	(26.97)	(26.97)	(26.96)	(26.96)
Age Squared				3.916**	3.920**	3.918**	3.917**	3.917**
				(26.07)	(26.05)	(26.05)	(26.04)	(26.04)
Age Cubed				0.990**	0.990**	0.990**	0.990**	0.990**
				(25.04)	(25.02)	(25.01)	(25.00)	(25.00)
Sex				1.787**	1.529**	1.510**	1.517**	1.517**
				(6.72)	(4.72)	(4.58)	(4.61)	(4.60)
Less Than High School				1.000	1.000	1.000	1.000	1.000
High School Graduate				0.791	0.857	0.866	0.870	0.875
				(1.37)	(0.90)	(0.84)	(0.81)	(0.77)
Some College				0.689*	0.712*	0.722	0.723	0.728
				(2.20)	(2.00)	(1.92)	(1.90)	(1.86)
College Grad & Beyond				0.497**	0.649*	0.704*	0.710*	0.714
				(4.12)	(2.54)	(2.03)	(1.97)	(1.93)
Baseline Life Event Score-Quartile 1							1.000	1.000
Baseline Life Event Score-Quartile 2							1.019	0.950
							(0.16)	(0.30)
Baseline Life Event Score-Quartile 3							0.950	0.850
							(0.44)	(0.90)
Baseline Life Event Score-							0.975	0.844

Quartile 4								
							(0.21)	(0.87)
Year 2 Life Event Score-Quartile 1							1.000	1.000
Year 2 Life Event Score-Quartile 2							0.968	0.848
							(0.28)	(0.91)
Year 2 Life Event Score-Quartile 3							0.842	0.922
							(1.40)	(0.45)
Year 2 Life Event Score-Quartile 4							1.159	1.292
							(1.27)	(1.40)
Black* Baseline Life Event Score-Quartile 1								1.000
Black* Baseline Life Event Score-Quartile 2								1.147
								(0.59)
Black* Baseline Life Event Score-Quartile 3								1.219
								(0.83)
Black* Baseline Life Event Score-Quartile 4								1.295
								(1.03)
Black* Year 2 Life Event Score-Quartile 1								1.000
Black* Year 2 Life Event Score-Quartile 2								1.251
								(0.96)
Black* Year 2 Life Event Score-Quartile 3								0.836
								(0.73)
Black* Year 2 Life Event Score-Quartile 4								0.834
								(0.77)
	21,810	21,810	21,810	21,810	21,810	21,810	21,810	21,810

* $p<0.05$; ** $p<0.01$

Table 2s. Adjusted Hazard Ratios (95% CIs) for Cumulative 20-Year Hypertension Incidence: CARDIA Study 1985-2005

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
Black	1.816**		1.508**			1.338**	1.330**	1.435*
	(9.29)		(6.20)			(4.24)	(4.13)	(2.20)
BMI: 17-18.5		0.385**	0.424**		0.405**	0.431**	0.423**	0.420**
		(2.97)	(2.70)		(2.81)	(2.63)	(2.68)	(2.70)
BMI: 18.5-25		2.717**	2.452**		2.627**	2.458**	2.501**	2.522**
		(3.02)	(2.73)		(2.91)	(2.72)	(2.77)	(2.79)
BMI: 25-30		1.112*	1.105*		1.099*	1.097*	1.097*	1.097*
		(2.55)	(2.42)		(2.24)	(2.21)	(2.20)	(2.19)
BMI: >30		0.881**	0.887**		0.885**	0.887**	0.887**	0.887**
		(4.58)	(4.36)		(4.42)	(4.35)	(4.36)	(4.36)
Age				0.000**	0.000**	0.000**	0.000**	0.000**
				(34.48)	(34.50)	(34.46)	(34.45)	(34.46)
Age Squared				2.035**	2.033**	2.031**	2.030**	2.030**
				(32.51)	(32.52)	(32.48)	(32.47)	(32.47)
Age Cubed				0.995**	0.995**	0.995**	0.995**	0.995**
				(30.48)	(30.49)	(30.44)	(30.43)	(30.43)
Sex				1.001	0.924	0.909	0.914	0.912
				(0.02)	(1.22)	(1.47)	(1.36)	(1.39)
Less Than High School				1.000	1.000	1.000	1.000	1.000
High School Graduate				0.758*	0.754*	0.756*	0.763*	0.765*
				(2.31)	(2.36)	(2.33)	(2.24)	(2.22)
Some College				0.657**	0.642**	0.652**	0.660**	0.662**
				(3.53)	(3.72)	(3.59)	(3.46)	(3.44)
College Grad & Beyond				0.439**	0.514**	0.563**	0.573**	0.575**
				(6.74)	(5.44)	(4.63)	(4.45)	(4.40)
Baseline Life Event Score-Quartile 1							1.000	1.000
Baseline Life Event Score-Quartile 2							0.937	1.022
							(0.71)	(0.16)
Baseline Life Event Score-Quartile 3							0.988	0.934
							(0.13)	(0.47)
Baseline Life Event							1.090	1.068

Score-Quartile 4								
							(0.93)	(0.45)
Year 2 Life Event Score-Quartile 1							1.000	1.000
Year 2 Life Event Score-Quartile 2							0.954	0.929
							(0.52)	(0.52)
Year 2 Life Event Score-Quartile 3							1.040	1.145
							(0.44)	(0.98)
Year 2 Life Event Score-Quartile 4							0.958	1.043
							(0.47)	(0.28)
Black* Baseline Life Event Score-Quartile 1								1.000
Black* Baseline Life Event Score-Quartile 2								0.856
								(0.85)
Black* Baseline Life Event Score-Quartile 3								1.099
								(0.51)
Black* Baseline Life Event Score-Quartile 4								1.042
								(0.22)
Black* Year 2 Life Event Score-Quartile 1								1.000
Black* Year 2 Life Event Score-Quartile 2								1.041
								(0.22)
Black* Year 2 Life Event Score-Quartile 3								0.842
								(0.94)
Black* Year 2 Life Event Score-Quartile 4								0.865
								(0.76)
	20,253	20,253	20,253	20,253	20,253	20,253	20,253	20,253

* $p<0.05$; ** $p<0.01$

Table 3s. Adjusted Hazard Ratios (95% CIs) for Cumulative 20-Year Diabetes Incidence: CARDIA Study 1985-2005

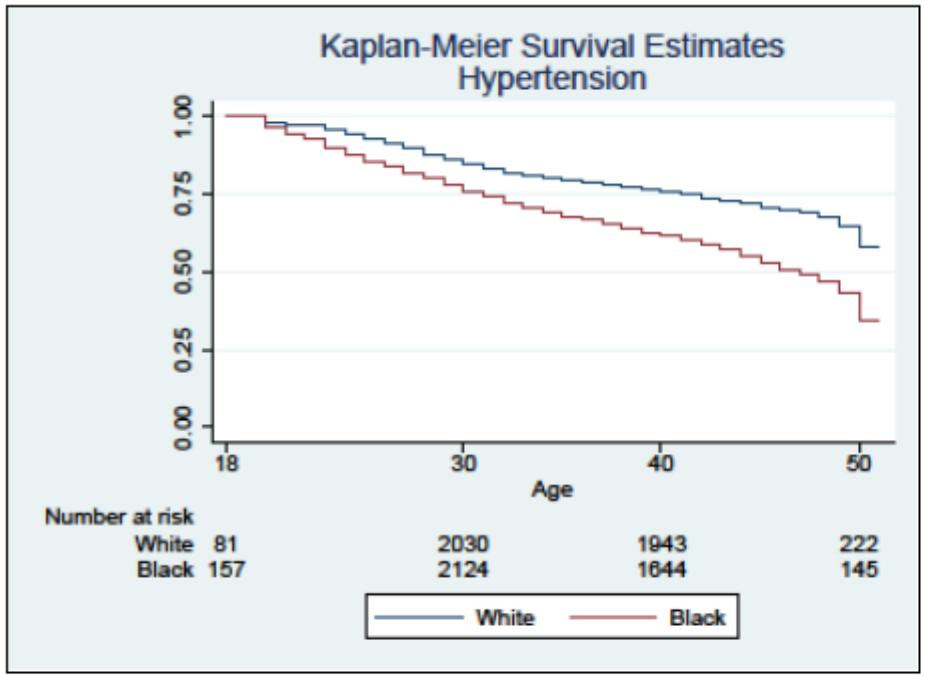
	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7	Model 8
Black	1.629**		1.271*			1.121	1.119	0.782
	(4.30)		(2.02)			(0.92)	(0.90)	(0.87)
BMI: 17-18.5		0.383	0.403		0.554	0.567	0.568	0.562
		(1.40)	(1.34)		(0.87)	(0.84)	(0.84)	(0.85)
BMI: 18.5-25		2.470	2.336		1.822	1.777	1.773	1.797
		(1.29)	(1.22)		(0.86)	(0.83)	(0.82)	(0.84)
BMI: 25-30		1.254**	1.249**		1.175*	1.174*	1.176*	1.172*
		(2.85)	(2.80)		(2.02)	(2.01)	(2.03)	(1.98)
BMI: >30		0.891*	0.895*		0.874**	0.876**	0.875**	0.876**
		(2.37)	(2.29)		(2.76)	(2.72)	(2.73)	(2.71)
Age				0.000**	0.000**	0.000**	0.000**	0.000**
				(19.91)	(19.93)	(19.93)	(19.92)	(19.93)
Age Squared				3.184**	3.185**	3.184**	3.183**	3.186**
				(19.37)	(19.37)	(19.37)	(19.37)	(19.37)
Age Cubed				0.992**	0.992**	0.992**	0.992**	0.992**
				(18.69)	(18.69)	(18.69)	(18.69)	(18.69)
Sex				2.804**	2.534**	2.514**	2.541**	2.553**
				(7.80)	(6.83)	(6.77)	(6.83)	(6.86)
Less Than High School				1.000	1.000	1.000	1.000	1.000
High School Graduate				1.034	1.097	1.103	1.108	1.114
				(0.12)	(0.33)	(0.35)	(0.36)	(0.38)
Some College				1.003	1.029	1.036	1.038	1.045
				(0.01)	(0.10)	(0.13)	(0.14)	(0.16)
College Grad & Beyond				0.676	0.878	0.911	0.917	0.916
				(1.41)	(0.47)	(0.33)	(0.30)	(0.31)
Baseline Life Event Score-Quartile 1							1.000	1.000
Baseline Life Event Score-Quartile 2							1.124	0.968
							(0.74)	(0.14)
Baseline Life Event Score-Quartile 3							0.925	0.807
							(0.48)	(0.87)
Baseline Life Event Score-Quartile 4							1.094	0.933

							(0.53)	(0.27)
Year 2 Life Event Score-Quartile 1							1.000	1.000
Year 2 Life Event Score-Quartile 2							1.021	0.771
							(0.13)	(1.07)
Year 2 Life Event Score-Quartile 3							0.921	0.834
							(0.49)	(0.75)
Year 2 Life Event Score-Quartile 4							1.100	1.170
							(0.58)	(0.65)
Black* Baseline Life Event Score-Quartile 1								1.000
Black* Baseline Life Event Score-Quartile 2								1.347
								(0.94)
Black* Baseline Life Event Score-Quartile 3								1.280
								(0.74)
Black* Baseline Life Event Score-Quartile 4								1.355
								(0.88)
Black* Year 2 Life Event Score-Quartile 1								1.000
Black* Year 2 Life Event Score-Quartile 2								1.647
								(1.55)
Black* Year 2 Life Event Score-Quartile 3								1.186
								(0.51)
Black* Year 2 Life Event Score-Quartile 4								0.912
								(0.28)
	22,467	22,467	22,467	22,467	22,467	22,467	22,467	22,467

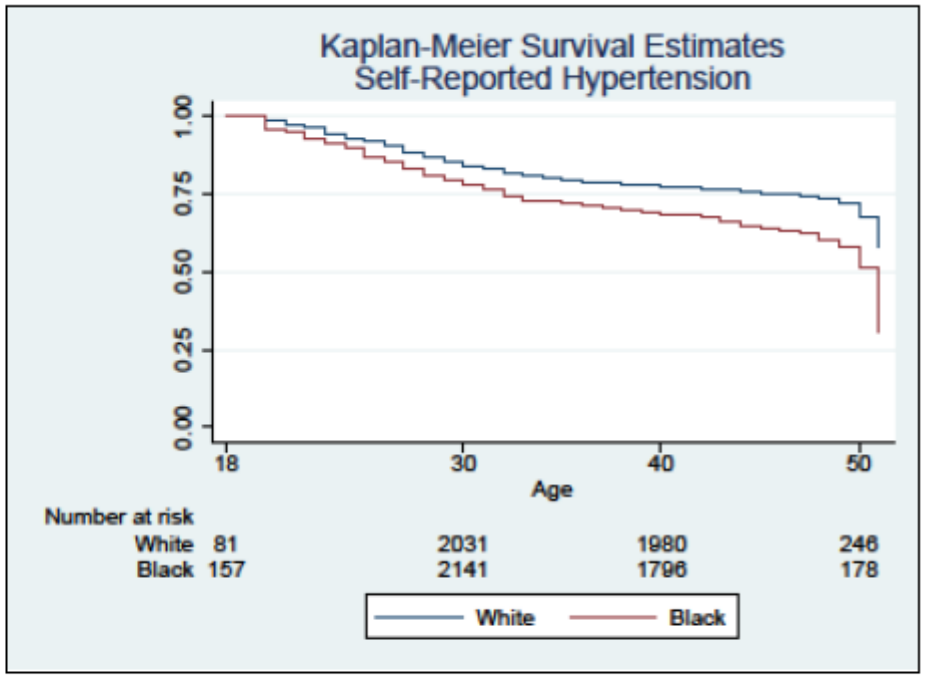
* $p<0.05$; ** $p<0.01$

Figure

1a

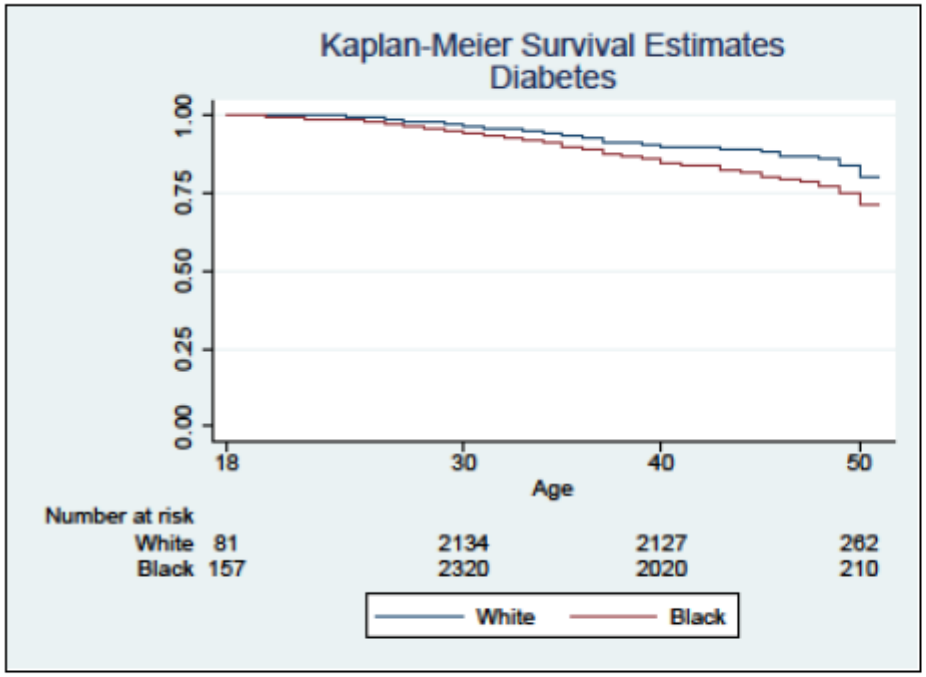

Kaplan-Meier Survival Estimates
Hypertension
0.00
0.25
0.50
0.75
1.00
18
30
40
50
Age
Number at risk
White 81 2030 1943 222
Black 157 2124 1644 145
White
Black

1b

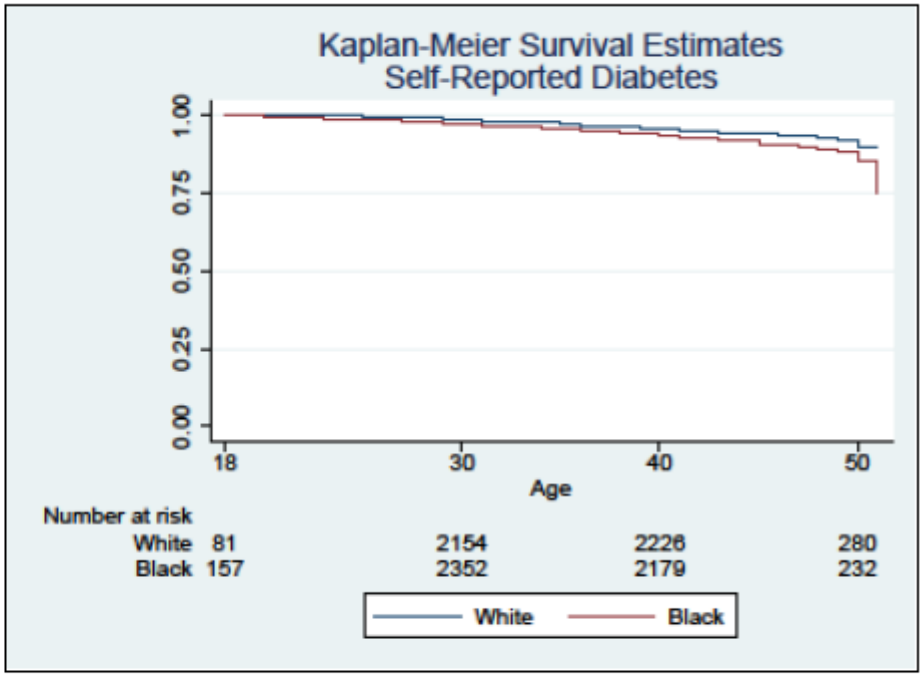

Kaplan-Meier Survival Estimates
Self-Reported Hypertension
0.00
0.25
0.50
0.75
1.00
18
30
40
50
Age
Number at risk
White 81 2031 1980 246
Black 157 2141 1796 178
White
Black

Figure

2a


Kaplan-Meier Survival Estimates
Diabetes
0.00
0.25
0.50
0.75
1.00
18
30
40
50
Age
Number at risk
White 81 2134 2127 262
Black 157 2320 2020 210
White
Black

2b


Kaplan-Meier Survival Estimates
Self-Reported Diabetes
0.00
0.25
0.50
0.75
1.00
18
30
40
50
Age
Number at risk
White 81 2154 2226 280
Black 157 2352 2179 232
White
Black