# Labeling Death: The Link Between Race, Hypertension Prevalence and Hypertension Related Death [1]

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**Labeling Death: The Link Between Race, Hypertension Prevalence and Hypertension Related Death**

Racial disparities in hypertension, one of the principal modifiable risk factors leading to heart disease, stroke and renal failure, embody one mechanism which leads to the excess mortality among blacks. This paper examines the link between racial disparities in hypertension prevalence and disparities in diagnosing—or labeling—deaths as hypertension related. We use data from the National Center for Health Statistics Multiple Cause of Death File and the National Health Interview Survey Linked Mortality File to analyze the relationship between race, the probability of having ones death diagnosed as hypertension, and various social, economic and health-related characteristics. Our results reveal that: 1) blacks are two times more likely than whites to have their deaths labeled as hypertension across the adult life course, 2) the increased likelihood of a black person's death being labeled as hypertension is unrelated to group differences in education, place of death, number of multiple-causes on death certificate, diabetes as a related cause and county fixed effects, and 3) the increased odds of labeling a black death as hypertension is unrelated to pre-existing reports of high blood pressure, subjective health status, BMI, socioeconomic status and region of occurrence. Our results suggest the presence of statistical discrimination in cause of death diagnoses such that health professionals erroneously use underlying racial disparities in hypertension prevalence to inform decisions in diagnosing hypertension related death. We conclude with a discussion of the implications of our results for policies designed to curb racial disparities in health and vitality.

There is a long history of research documenting the relationship between racial classification and mortality. Early work cited the genetic deficiencies of minority groups as the source of the large disparities in vitality (Davenport 1911; Hoffman 1896; Gould 1996; Graves 2001). More recent research locates the source of racial mortality disparities in socio-environmental factors such as socioeconomic status (SES), neighborhood characteristics and access to quality health care (Collins & Williams 1999; Morenoff et al 2007; Weinick, Zuvekas & Cohen 2000; Williams & Jackson 2005). One arena that promises to shed additional light on the sources and solutions to racial mortality disparities is underlying causes of death. Underlying causes represent the proximate mechanisms of mortality disparities and can be used as a guide for policies that aim to improve minority health (i.e., Healthy People 2010).

The most widely discussed underlying cause of mortality disparities and the focus of many targeted health policies is heart disease (Elo & Drevenstedt 2004; Harper et al. 2007). This is a attributable to heart disease being the most prevalent cause of death accounting for approximately 35 percent of deaths among blacks and whites in 2005 (CDC Wonder Compressed Mortality File, ICD 10 codes I00 to I99). A principal risk factor for heart disease is hypertension (Henderson et al. 2007; Keil et al. 1993; Watson 2008). Blacks are more likely than whites to be diagnosed with hypertension (i.e., systolic blood pressure greater than 140 mm Hg systolic, greater than 90 mm Hg diastolic, or on hypertensive medication) and when on medication for hypertension blacks are significantly less likely than whites to have controlled their blood pressure (Centers for Disease Control and Prevention 2005; Howard et al. 2006; Morenoff et al. 2007; Safford et al. 2007). Blacks are also much more likely to die from hypertension than whites [see Table 1]. The wide racial disparities in hypertension prevalence, hypertension control, and the related significant heart disease, stroke, renal disease and hypertension mortality disparities has led to increasing scholarly and policy attention (Nesbitt 2006; Watson 2008).

In this paper, we investigate the link between racial disparities in hypertension preva-

lence and hypertension related death. More specifically, we use data from the National Center for Health Statistics Multiple-Cause of Death File and National Health Interview Survey Linked Mortality File to assess the degree to which pre-existing hypertension status and other socio-environmental factors contribute to racial disparities in having hypertension listed as a cause of death—hypertension related death.[1] Blacks could have a higher percentage of hypertension related deaths—and hypertension related mortality rates—because hypertension prevalence is truly higher among African Americans. The higher percentage of hypertension related deaths among blacks, however, could also be an artifact of racial differences in what gets labeled as an underlying/contributing cause of death (i.e., statistical discrimination). Our aim is to analyze the validity of the link between blacks' increased hypertension prevalence and the often cited hypertension related mortality statistics which are seen as their grave consequence (Henderson et al. 2007; Watson 2008).

In what follows, we highlight the degree to which racial disparities in having one's death labeled as hypertension is a product of disparities in pre-existing hypertension and other factors. Our results suggest that blacks' increased risk of having their death labeled as hypertension is attributable to statistical discrimination in cause of death diagnoses, and is unrelated to SES, pre-existing hypertension and several other socio-environmental factors. We conclude the paper with a discussion of the implications of these results for future research and health policy.

## Background

Race, a concept that relates features such as skin color and hair texture to subgroup membership, continues to shape the social outcomes and life chances of all U.S. residents. Despite drastic improvements in average socioeconomic and health outcomes in the past generation, the consequences of this system of privilege and disadvantage—racial stratification—are still manifest in racial disparities in health and vitality (Byrd & Clayton, 2000). Black children

can expect to live a significantly shorter and less healthy life than their white counterparts (Hayward & Heron 1999; Geronimus et al. 2001). Indeed, this disparity is related to blacks' disadvantage across an array of social dimensions such as the labor market and educational systems (Wiliams 1999). Racial health disparities, though, are pronounced even among persons of similar social and economic standing (Elo & Preston 1996; Menchik 1993; Rogers 1992; Rogers, Hummer & Nam 2000).

Hypertension prevalence is one such racial health disparity that is receiving increasing attention. Morenoff and colleages (2007) estimated that at the turn of the 21st century blacks were 1.8 times more likely to have hypertension than whites of similar socioeconomic status. Ferdinand and Armani (2007) further highlighted the significance of this hypertension disparity in their article on hypertension management. They write:

> The prevalence of hypertension in blacks in the United States is among the highest in the world. Compared with whites, blacks develop hypertension at an earlier age, their average blood pressures are much higher and they experience worse disease severity ... Consequently, blacks have a ... 1.8 times greater rate of fatal stroke ... [and] 4.2 times greater rate of end-stage renal disease (67).

Blacks, then, experience much worse levels of hypertension prevalence, hypertension control, and, as a consequence, have significantly higher cardiovascular and renal-disease mortality rates (Henderson et al. 2007; Howard et al. 2006; Keil et al. 1993; Safford et al. 2007; Willams & Jackson 2005).

Indeed, racial mortality disparities are seen in a number of underlying causes of mortality such as stroke and homicide. Table 1 shows age-adjusted mortality rates for selected causes of death during the period 2000 to 2004. Among men, blacks have much higher mortality rates for every cause of death. HIV and homicide mortality rates, for example, are 10 times higher among black males than among whites. HIV mortality rates among black women are also significantly higher than those for white women.

(Table 1 About Here)

The mortality rates from heart disease are particularly intriguing. The heart disease mortality rate in each race-gender group is larger than that for any other cause and the ischemic heart disease mortality rate for each group constitutes the majority of heart disease mortality. The black/white mortality disparities in overall heart disease [1.36 for men and 1.34 for women] and ischemic heart disease [1.19 for men and 1.25 for women], though, are fairly small in comparison to the disparities in hypertensive diseases. Black men and women are 3.4 and 2.8 times, respectively, more likely than whites to die from hypertension (ICD 10 codes I10 and I12[2]). When accounting for hypertension as a contributing cause of mortality (i.e., the multiple-cause mortality rate), we see that "hypertension related" mortality[3] is approximately two times higher among blacks than whites. This disparity holds even though the multiple-cause mortality rates for hypertension related diseases in each group are quite high.

A similar racial mortality disparity is seen in the relationship between hypertension and renal disease. Blacks are two times more likely to die from renal disease than whites. Blacks, however, are four times more likely to die from hypertensive renal disease than whites. While the pathophysiology of hypertension would suggest it should play a similar role in cardiovascular and renal disease in all racial and ethnic groups, these mortality rates suggest that hypertension plays a critical role in blacks' increased mortality from heart disease and renal disease.

The mortality statistics in Table 1 highlight the significance of hypertension in racial mortality disparities. These hypertension related mortality disparities are viewed as an essential outcome of the aforementioned disparities in hypertension prevalence (Howard et al. 2006; Keil et al. 1993; Safford et al. 2007; Willams & Jackson 2005). Henderson and colleagues (2007), for example, used data from the Multiethnic Cohort Study and found that "...diabetes and hypertension were the most important risk factors for the increased [Acute Myocardial Infarction] and [other forms of heart disease] amongAfrican American

men" (6). Additionally, Watson (2008) reaffirmed the importance of hypertension in her "Call to Action" for heart disease disparities. She writes "... hypertension in African Americans is associated with a 3-5 times higher cardiovascular mortality rate than in European Americans... and the risk of hypertensive end-stage renal disease is almost five times higher in African Americans" (19). Racial disparities in hypertension prevalence, then, are seen as a concrete mechanism that contributes to significant disparities in hypertension related mortality across the life course.

Although hypertension is a definitive biological mechanism that leads to disparities in heart disease and renal failure, social and health scientists are still at a loss as to the sources of racial disparities in hypertension prevalence. Many scholars point to racism and discrimination—including segregation—as a source of hypertension disparities (Collins & Williams 1999; Din-Dzietham et al. 2004; Krieger 1990; Morenoff et al. 2007; Williams 1999). Others point to modifiable risk factors such as exercise, obesity and smoking, as well as to the intensity of hypertension treatment (Dennison et al. 2007; Safford et al. 2007). Still others look to genetic factors/theories of hypertension disparities such as salt sensitivity (He at al. 1998; Weinberger 1996; Wilson & Grim 1991). These mechanisms for increased hypertension among blacks are varied, work at multiple levels of biological and social causation, are not mutually exclusive, and likely act in concert to shape the observed racial disparities in hypertension prevalence and it's grave consequence, hypertension related mortality (Ferdinand & Armani 2007; Nesbitt 2005; Watson 2008).

In addition to the aforementioned sources of disparities in hypertension, statistical discrimination may also be a source of racial disparities in hypertension, and hypertension related mortality in particular. This type of discrimination is often discussed in economics and usually refers to employers' use of race as an index of the differential productivity of workers in the face of uncertainty (Darity 1982, 1998; Gould 1992; Loury 1995). In regards to health, Balsa, McGuire and Meridith (2005) write:

> The basic idea of statistical discrimination in the health context is that uncertainty about the patients severity of illness can induce the doctor to behave differently with otherwise identical members of different race/ethnic groups. If the underlying prevalence of the illness is associated with race, the doctor might take race into account in deciding about the diagnosis and treatment of a particular patient (229).

Statistical discrimination in diagnosing hypertension related death, then, may result from health professionals using race as a proxy for hypertension prevalence—or status—when diagnosing cause of death (Balsa & McGuire 2001; Burgess, Fu & van Ryn 2004).

Existing research suggests that statistical discrimination exists in clinical practice and contributes to disparities in both disease diagnosis and treatment (Balsa, McGuire & Meredith 2005; Loring & Powell 1988; van Ryn, Burgess, Malat & Griffin 2006). Perneger and colleagues (1995), for example, examined statistical discrimination in end-stage renal disease (ESRD) diagnosis using patient vignettes. They found that black patients were two times more likely to be diagnosed with "hypertensive" ESRD—as opposed to ESRD—than their white counterparts with similar clinical histories. More recently, Green and others (2007) found that clinicians are more likely to diagnose chest pain as coronary artery disease in blacks and less likely to treat blacks with thrombolysis when diagnosed. These results suggest that statistical discrimination exists in disease diagnosis and, by deduction, that high hypertension prevalence among blacks may lead to over-statements of hypertension related deaths among blacks and/or under-statements among whites.

While the determinants of pre-existing hypertension and hypertension related death are varied, the connection between hypertension prevalence and hypertension related death is arguably a concrete mechanism of blacks' increased cardiovascular and renal disease mortality risks. In what follows, we analyze the relationship between blacks' increased vulnerability to hypertension and hypertension related death. We aim to highlight the primary social and health-related determinants of the hypertension diagnosis on a person's death certificate. We are particularly interested in determining whether racial disparities in hypertension related

mortality is a product of differences in hypertension prevalence or if they are attributable to statistical discrimination in cause of death diagnosis.

**Data**

We use two data sets to examine the real, and potentially artifactual, role of hypertension in racial mortality disparities. The first data set, the National Center for Health Statistics Multiple Cause of Death File (MCD) for the years 2000 to 2004, contains information from all death certificates during the period. This data set consists of over 13 million deaths occurring in the U.S. between January 1, 2000 and December 31, 2004. We use data on the underlying cause of death, multiple contributing causes, place of occurrence (e.g., hospital outpatient), county of occurrence[4], and the age, race, sex, marital status and educational achievement of the decedent. We restrict our data to black and white deaths occurring in the U.S. during the five year period as our focus is on the black/white disparity in having ones death diagnosed as hypertension. To test the validity of our results, we further restricted our sample to heart disease deaths where hypertension labeling is a critical factor. We achieved the same results from the limited sample as in the full sample shown here. Detailed descriptions of the MCD variables we use appear in Table A1.

The dependent variable for our models using MCD data is having hypertension listed as an underlying or contributing (i.e., multiple) cause of death. For the contributing causes, we used the record axis conditions. Indeed, the record axis causes of death may be different than the actual conditions listed on the death certificate (i.e., entity axis conditions). The record axis conditions, however, are more reliable as they have been adjusted to conform to known pathways of mortality (Israel, Rosenberg & Curtin 1986; Wall 2005). For both the underlying and contributing causes of death, we define hypertension using the International Classification of Disease 10th revision (ICD-10) codes I10.0 to 10.9 [essential hypertension] and I12.0 to I12.9 [hypertensive renal disease].[5] We use these codes to be consistent with the

National Health Interview Survey Linked Mortality File definition. We performed additional analyses of the MCD data using ICD 10 codes I10.0 to I14.9—including hypertensive heart disease and secondary hypertension—which showed higher racial disparities at younger ages (results available upon request).

In addition to using information on hypertension and hypertensive diseases in the MCD data, we use data on diabetes as an underlying or contributing cause of death. We include diabetes in our analysis because it represents a potent cardiovascular risk, frequently coexists with hypertension, and is more prevalent in the black population in the United States (Watkins 2004). Incorporating the presence of diabetes in our models allows us to control for a major potential confounder accounting for increased hypertensive and cardiovascular deaths among blacks. We define the diabetes variable in the MCD data as having an underlying or contributing cause of death that is between the ICD-10 codes E10.0 and E14.9.

The second data set we use in our analysis is the National Health Interview Survey Linked Mortality File (NHIS). This data set consists of information collected in the NHIS surveys from 1986 to 2000 linked with the National Death Index from 1986 to 2002. We restrict our sample to survey respondents from the years 1986 to 1996—the survey questions changed in 1996—and to deaths occurring between 1995 and 2002. Our sample data includes information on underlying cause of death, multiple contributing causes, time of death (quarter), geographic region, and the age, race, sex, marital status, educational achievement, household income, subjective health status, BMI (i.e., body mass index) and pre-existing report of hypertension for each decedent.

The NHIS household income variable consists of 27 categories. In line with previous research, we use the midpoint of each category as an estimate for individual earnings below the median. For categories above the observed median, we fitted a Pareto curve to the distribution and estimated individual earnings as the average within the respective categories (see Parker & Fenwick 1983). For missing data on income, we imputed household income

using ordinal logistic regression since the initial income variable is ordinal—as opposed to multiple imputation. We include a dummy variable for missing income in all models using income to control for possible bias related to missing data on income.

The NHIS BMI variable is created using the height and weight measures from the NHIS Linked Mortality File. We estimate BMI using the equation: $BMI = [weight/(height^2)] * 703$. To account for missing values on either weight or height, we used multiple imputation and included a variable for BMI missing in our regression models. We imputed weight and height data using a Markov Chain Monte Carlo (MCMC) method (Schafer 1999) and used the average of five imputations as the final imputed value.

Indeed, many of the NHIS variables we use may have changed over the follow-up period. However, we replicated our results using the sample of deaths prior to 1995 [6], deaths occurring within five years of the survey, deaths between 1995 and 2002 that occurred within five years of the survey, and the entire sample of deaths (see Tables A3 and A4 for selected results). We use the more recent period as it overlaps with the MCD data. Detailed descriptions of the NHIS variables we use in the analysis appear in Table A2.

As in the MCD data, the dependent variable in our analysis of the NHIS data is having hypertension listed as an underlying or contributing cause of death. The NHIS data contains a dummy variable indicating whether hypertension is listed as an underlying or contributing cause of death. Additionally, the data contains a dummy variable indicating whether diabetes is listed as an underlying or contributing cause on the death certificate. We use the hypertension and diabetes cause of death dummy variables to replicate the aforementioned MCD variables in the NHIS data.

For us, the critical control variable in the NHIS data is pre-existing report of hypertension. This variable comes from the condition checklist where respondents are asked to report existing medical problems. Although these data do not contain specific information on blood pressure of respondent or medications, existing research indicates that both whites

and, more so, blacks are well aware of hypertension status (Center for Disease Control 2005; Howard et al. 2007; Morenoff et al. 2007). Thus, the condition checklist is a good proxy for having hypertension.

**Methodology**

The focus of our analysis is racial disparities in the proportion of deaths attributable to hypertension and hypertensive disease (i.e., underlying and contributing causes)—labeling disparities. We focus on labeling disparities because they are a critical part of the often cited hypertension related mortality disparities such as those shown in Table 1. This is best seen in the black/white ratio of cause specific mortality rates $[{}_nM_x^i]$. We write the cause specific mortality rate from cause i in race group j as:

$$ {}_nM_x^i(j) = \frac{{}_nD_x^i(j)}{{}_nN_x(j)} = \frac{{}_nD_x(j)}{{}_nN_x(j)} \cdot \frac{{}_nD_x^i(j)}{{}_nD_x(j)} = {}_nM_x^i(j) \cdot {}_n\pi_x^i(j) \tag{1} $$

where ${}_nM_x(j)$ is the mortality rate of race group j for ages x to x+n, ${}_n\pi_x^i$ is the proportion of deaths attributable to cause i among individuals aged x to x+n [i.e., ${}_nD_x^i/{}_nD_x$ where D is number of deaths], and ${}_nN_x$ is the number of person years lived between ages x and x+n (i.e., the midyear population). Equation 1 shows that the cause specific mortality rate for race group j is the product of the age-specific mortality rate and the proportion of deaths attributable to cause i in the age group.

Using Equation 1, we can express the black/white ratio in cause specific mortality rates as:

$$ {}_nR_x^i = \frac{{}_nM_x^i(B)}{{}_nM_x^i(W)} = \frac{{}_nM_x(B)}{{}_nM_x(W)} \cdot \frac{{}_n\pi_x^i(B)}{{}_n\pi_x^i(W)} = {}_nR_x \cdot {}_nL_x^i \tag{2} $$

where ${}_nR_x^i$ is the ratio of cause specific mortality rates from cause i for ages x to x+n. Equation 2 reveals that there are two mechanisms that lead to higher cause-specific mortality rates among blacks than among whites. The first is higher overall mortality among blacks [i.e., ${}_nM_x(B)/{}_nM_x(W)$]—rate disparities (${}_nR_x$). The second mechanism that leads to higher cause specific mortality rates, and the focus of our analysis, is racial disparities in the proportion of deaths from cause i [i.e., ${}_n\pi_x^i(B)/{}_n\pi_x^i(W)$]—labeling disparities (${}_nL_x^i$). When blacks have a greater proportion of deaths attributable to cause i than whites, the ratio of cause-specific mortality rates from cause i is larger than the ratio of overall mortality rates. Thus, racial disparities in the likelihood of having one's death diagnosed as cause i (i.e., hypertension) is necessarily linked to disparities in the likelihood of dying from cause i. The analysis below focuses on the sources of blacks' increased odds of having their death diagnosed as hypertension[7].

Our analysis of disparities in hypertension death diagnoses proceeds in three stages. First, we analyze racial disparities in hypertension labeling $[{}_nL_x^i]$ using descriptive statistics on the age distribution of hypertension deaths. We estimate these statistics using the MCD data for the years 2000 to 2004. This analysis highlights the baseline distribution of hypertension deaths among blacks and whites across the age distribution. In this first stage we define a hypertension death as one where the underlying cause is listed as essential hypertension or hypertensive renal disease (ICD codes I10.0 to I10.9 and I12.0 to I12.9). We focus on hypertension as an underlying cause at this point to enable comparisons with the age-distribution of deaths from two related cardiovascular causes of death—stroke and ischemic heart disease.

For the second stage of our analysis, we use the MCD data on deaths of individuals over age 20 to assess the extent to which having one's death diagnosed as "hypertension related"—hypertension listed as either an underlying or contributing cause—is associated with race of decedent, as well as other social and environmental factors. The dependent

variable in this stage is having hypertension appear somewhere on the death certificate.

We use logistic regression to estimate the racial disparity in diagnosing hypertension related deaths (i.e., residual race coefficient) net of the aforementioned personal characteristics of decedents. The logistic regressions in this stage of the analysis are stratified by age so as to account for the possible relationship between age and other control variables. The age strata consist of five-year age groups from 20-25 to 90-95, and an open-ended category of 100+. We also use county fixed effects in this stage of the analysis to account for geographic variation in the practices of reporting underlying causes of death and availability of medical specialists such as cardiologists, as well as the uneven distribution of the racial groups (Allison 2005). Thus, the second stage analysis highlights the extent to which racial disparities in diagnosing hypertension deaths persist among persons (i.e., decedents) with similar social and environmental characteristics.

The last stage of our analysis uses NHIS data to assess the degree to which racial differences in hypertension diagnosis at time of death is related to pre-existing health status. This analysis is restricted to respondents over age 20 and is performed in two steps. We begin by performing logistic regression of pre-existing hypertension on race and other characteristics using the baseline NHIS survey data—the sample is all NHIS survey respondents over age 20 from 1986 to 1996. This analysis highlights the nature and magnitude of racial disparities in pre-existing hypertension, as well as the relationship between hypertension, socioeconomic status, and subjective health status.

We then restrict our sample to deaths in the period 1995 to 2002 and use logistic regression to estimate the extent to which racial disparities in the proportion of hypertension related deaths is related to disparities in pre-existing hypertension and other social characteristics of the decedent. Due to the small sample sizes of blacks [see Table 2], we code age in these models as continuous and use an age-squared term to account for curvilinear variation in probability of diagnosing hypertension related deaths across the adult age dis-

tribution. We also use weights in the NHIS analysis to account for the stratified sampling techniques used in the survey. The weights are normalized so that they sum to the sample size. Altogether, the third stage of the analysis is designed to reveal whether pre-existing hypertension is a mechanism that leads to significantly higher reports of hypertension as an underlying/contributing cause of death among black decedents, or if statistical discrimination in cause of death diagnosis contributes to the wide racial disparities in the often cited hypertension mortality statistics. We now turn to our results.

**Results**

Table 2 shows selected descriptive statistics for men and women by race in each of our samples. The first panel, MCD data, indicates that blacks are more likely to have hypertension listed as a contributing or underlying cause of death than whites in the period 2000 to 2004. This result also holds in the NHIS sample where blacks report higher levels of pre-existing hypertension (from 1986-1996 surveys) and have a larger share of deaths diagnosed as hypertension related (i.e., underlying or contributing). Blacks in both data sets also have lower levels of education, a larger percentage of never married persons, and, in the MCD data, are more likely to die as either an in- or outpatient in a hospital. Thus, the descriptive statistics suggest that the high hypertension mortality among blacks may be attributable to higher levels of pre-existing hypertension. These raw statistics, however, may conceal age variation in racial disparities in hypertension labeling.

(Table 2 About Here)

Figures 1a and b show the black/white ratio in the age-specific proportion of deaths diagnosed as hypertension, stroke and ischemic heart disease by gender. The first panel shows that black men are 2 to 3 times more likely to have their death labeled as hypertension than white men. This disparity persists across the entire adult life course. The racial disparity in

stroke death diagnosis, on the other hand, increases from equality at age 20 to a peak of 2 in the middle-ages and then converges to parity at the oldest ages. The notable finding here is that stroke and hypertension mortality follow very different patterns among men.

(Figure 1 About Here)

The second panel of Figure 1 shows that racial disparities in the proportion of hypertension deaths among women follow a different pattern than those among men. Specifically, young black women were five times more likely to have their death diagnosed as hypertension than young white women. This disparity in hypertension labeling declined across the adult life course to a minimum of 1.5 in the 100+ age group. The results for stroke, however, are similar to those for men. Young and middle aged black women were approximately 1.5 times more likely than their white counterparts to have their death diagnosed as stroke, while older black and white women were equally likely to have their death diagnosed stroke. Although racial disparities in labeling hypertension deaths among women is different than among men, the disparities fail to converge in either population as was the case in labeling stroke deaths.

The lack of convergence in hypertension labeling disparities contradicts the age-pattern of racial mortality disparities in general, and for many diseases and causes of mortality (Berkman, Singer & Manton 1989). Rogers, Hummer and Nam (2000) as well as Elo and Preston (1994), for example, reported that blacks experience significant mortality disparities below age 65 and more modest—and often insignificant—disparities above that age. Additionally, Crimmins (2005) showed that racial and SES morbidity disparities are much larger among those below age 65, and disparities in the prevalence of risk factors—such as hypertension—are minimal at the oldest ages. In the case of hypertension, though, black/white disparities in diagnosing hypertension deaths remains near two up to the oldest ages (i.e., 100+). The lack of convergence suggests hypertension labeling disparities may be a product of social or environmental mechanisms that distinguish it from other causes of mortality.

We used logistic regression to further assess the extent to which racial disparities in reported hypertension deaths are related to other social and environmental factors. Figure 2 depicts the "baseline" and "full" logistic regression models.[8] We use figures to display these results because the models are stratified by age (i.e., we estimate a model separately for each age group). These models all use hypertension as an underlying or contributing cause of death as the dependent variable (i.e., hypertension related deaths). The baseline model includes controls for year. Thus, the results highlight the raw racial disparities in the likelihood that a death is diagnosed as hypertension related. The full model adds terms for number of contributing causes, education, place of death, size of MSA, marital status, diabetes as a contributing cause and county fixed effects.

(Figure 2 About Here)

The first panel of Figure 2 shows that racial disparities in diagnosing hypertension as a cause of death are partly related to the aforementioned social and environmental factors, but not in the expected direction. More specifically, the results indicate that racial disparities in diagnosing hypertension as a cause of death among men actually widen when we control for social and environmental characteristics. In contrast to men, racial disparities in hypertension labeling among women does not have a systematic relationship with social and environmental factors. At certain ages, the increased risk of diagnosing hypertension as a cause of death among black women widens after controlling for the aforementioned characteristics. The disparities at other ages decline after controlling for social and environmental characteristics. Racial disparities in diagnosing hypertension as a cause death, however, remain pronounced across the female life course.

As one can see, blacks are more likely to have their death labeled as hypertension—both contributing and underlying—and this disparity persists after controlling for a variety of social and environmental factors that appear on death certificates. These disparities, however,

may be related to blacks having significantly higher levels of hypertension prevalence than whites and having lower levels of health more broadly. We use the NHIS to assess the extent to which pre-existing hypertension and health status is the primary culprit behind racial disparities in hypertension labeling—and to assess the validity of often cited "grave consequences" of racial disparities in hypertension prevalence. We first, however, analyze racial disparities in hypertension prevalence. Tables 3 and 4 show the logistic regression results on pre-existing hypertension—the dependent variable is the aforementioned NHIS checklist variable.

Table 3 shows that black male NHIS respondents are roughly 1.4 times more likely to report having high blood pressure than their white counterparts. When we control for social and economic characteristics (Model 2), the racial disparity in hypertension among men slightly increased to 1.5. Adding controls for region (Model 3), as well as BMI and subjective health status (Model 4) also leads to slight increases in the racial disparity in hypertension among men. Altogether, black and white men with similar social, economic and health statuses have significant disparities in high blood pressure.

(Table 3 About Here)

Table 4 reveals that black women are approximately 1.7 times more likely to report having high blood pressure than white women. Like men, when we add controls for socioeconomic status (Model 2), region (Model 3), BMI and subjective health status (Model 4) the disparity among women slightly increases to 1.77. These results—and those above—replicate existing research findings which highlight the persistent racial disparity in hypertension prevalence (Morenoff et al. 2007; Safford et al. 2007). Indeed, these disparities are argued to be the source of wide racial disparities in heart disease and stroke mortality, and hypertension related mortality in particular (Henderson et al. 2007; Nesbitt 2005; Watson 2008).

(Table 4 About Here)

Table 5 shows the results from our analysis of the link between black men's higher levels of hypertension prevalence and their increased likelihood of having their death diagnosed as hypertension related. Model 1 shows that black men who died between 1995 and 2002 were roughly two times more likely to have their death diagnosed as hypertension related than their white counterparts. Pre-existing hypertension (Model 2) is also a significant determinant of having one's death diagnosed as hypertension related. Although pre-existing hypertension is a significant determinant of having one's death labeled as hypertension (Model 2), it only has a marginal impact on racial disparities in hypertension labeling. Adding controls for SES (Model 3), region (Model 4) as well as BMI, subjective health status and diabetes as a contributing cause (Model 5) does not affect the racial disparity in hypertension death labeling among men. Thus, the results here suggest that the higher prevalence of hypertension among black men is not related to their increased odds of having their death diagnosed as hypertension—black men are more likely to have their death diagnosed as hypertension related regardless of their pre-existing hypertension status.[9]

(Table 5 About Here)

The results from our analysis of hypertension prevalence and racial disparities in hypertension labeling among women appear in Table 6. The baseline model [1] indicates that black women are 1.7 times more likely to have hypertension listed as an underlying or contributing cause on their death certificate. Model 2 indicates that women with pre-existing hypertension are 1.5 times more likely to have their death diagnosed as hypertension related. Black female decedents, however, are still significantly more likely to be labeled as a hypertension related death after controlling for pre-existing high blood pressure. In addition to pre-existing high blood pressure and race, the other major contributors to having hypertension appear as an underlying or related cause of death are BMI and diabetes on death certificate (Model 5). Women who have diabetes listed as a cause of death are 3 times more

likely to also have their death diagnosed as hypertension related, while obese women are 1.3 times more likely to have their death labeled as hypertension related. Race, however, continues to be a significant determinant of the hypertension death diagnosis. Hence, black women are 1.7 times more likely to have their death labeled as hypertension than white women with similar social, economic and health characteristics, including pre-existing hypertension.

(Table 6 About Here)

**Discussion**

Racial differences in the prevalence of hypertension in the United States have been well established. The most recent data from NHANES puts the prevalence of hypertension at 29% of the adult population, 28% of non-Hispanic whites, and 41% of non-Hispanic blacks. (Ostchega et al, 2008b: 2) However, this 13 percent difference in hypertension prevalence is not adequate to explain a threefold difference in underlying cause hypertensive mortality among men and women. The results above indicate that pre-existing diagnosis of hypertension, while a significant determinant of having one's death labeled as hypertension, is not correlated to the significant racial disparity in diagnosing hypertension related deaths. The results also show that the racial disparity in diagnosing hypertension deaths remains pronounced across the entire life course even up to the oldest ages (i.e., 100+). Indeed, these results counter much of what we know about the mechanisms of increased cardiovascular mortality among blacks and the black/white mortality convergence at the oldest ages. They do, however, suggest that factors other than diagnosis of pre-existing hypertension are driving racial disparities in diagnosing hypertension deaths, and the reported hypertension mortality disparities more broadly. There are three major categories of explanation for such a difference.

First, it is possible that hypertension or other predictors of cardiovascular mortality are

worse in blacks than whites. (Ferdinand & Saunders 2006). Substantial work has been done exploring differences in pathophysiology of hypertension in blacks, particularly in the rennin-aldosterone system and salt-sensitivity (He at al. 1998; Stewart, Johnson & Saunders 2006; Weinberger 1996; Wilson & Grim 1991). Regarding other risk factors, we were able to control for diabetes in the NHIS models and achieved the same results.[10] However, limitations in the data did not allow us to control for smoking, hyperlipidemia, family history of cardiovascular disease, exercise, stress, and other lifestyle factors which would be relevant predictors of cardiovascular mortality, whether attributed to hypertension or not.[11] Poor access to quality health care could also play a role in making hypertension more severe, less adequately treated and more deadly for black Americans (Duru et al. 2007; Holmes et al. 2005; Ostchega et al. 2008a; Safford et al. 2007).

We tested the merits of the argument that the increased severity of hypertension among blacks contributes to observed disparities in diagnosing hypertension deaths using a race-by-pre-existing hypertension interaction effect. Our results indicated that pre-existing hypertension has the same relationship with hypertension labeling among black and white deaths. Although these results suggest severity may not be an issue, it may still play a role through other unobserved risk factors such as access to quality health care. Multilevel modeling has shown substantial facility-level effects whereby the hospitals which treat a disproportionate share of black patients have higher mortality rates for acute myocardial infarction (Skinner et al. 2005). This disparity may be related to poorer adherence to basic quality measures such as aspirin and beta blocker use, or may be due to lesser access to specialists and advanced treatment such as emergency cardiac catheterization. If the explanation that hypertension is more likely to lead to mortality in black Americans is due to greater intrinsic severity of hypertension, greater burden of cardiovascular risk factors or poor access to health care, then programs addressing control of cardiovascular risk factors would be the appropriate policy response to reduce the disproportionate burden of mortality from hypertensive diseases.

The second possible mechanism behind blacks' increased likelihood of having their deaths diagnosed as hypertension is labeling bias. This mechanism operates through either mis-diagnosis of hypertension in life or the exclusion of multiple causes in the death diagnosis. In the first case, under-diagnosis of hypertension in African Americans in life may contribute to the observed disparities in hypertension labeling. Indeed, research indicates that both blacks and whites are well aware of their hypertension status (Center for Disease Control 2005; Howard et al. 2007; Morenoff et al. 2007). High blood pressure, however, is a continuous variable that is positively associated with increased coronary, stroke and renal disease mortality risks (Roccella, Bowler & Horan 1987).[12] Mis-diagnosis implies that the current blood pressure levels we use to diagnose hypertension lead to underestimates of the *true* prevalence of hypertension among blacks, and in the U.S. population more broadly. The validity of this mechanism, though, rests on the presumption that: 1)the under-diagnosis of hypertension among blacks is significantly greater than among whites, and 2)the disparity in the under-diagnosis of hypertension persists to the oldest ages (i.e., 100+). The likelihood that both of these presumptions hold—especially the second—is questionable at best. If, however, labeling bias in the diagnosis of hypertension in life is operating, then a reassessment of the blood pressure levels that trigger initiation or intensification of treatment for hypertension should be driving policy goals.

In addition to mis-diagnosis, labeling bias may result via excluding multiple causes on the death certificate. Studies of death certification show that hypertension, diabetes, and other underlying causes of death are frequently absent from death certificates, which simply list the proximate cause of death (Lu et al. 2005; Goldacre 1993). If the dramatic difference in black/white hypertensive deaths represents in part a difference in the likelihood that hypertension will be listed as an underlying cause of death, then we should see persistent racial disparities in diagnosing hypertension mortality. In kind, the policy takeaway would be that more systematic training of certifying physicians might allow more accurate assessments

of underlying causes of death both within racial groups and across the population. We tested the merits of the "exclusion" mechanism in two ways. First, we analyzed the covariation between race, hypertension death diagnosis and the number of multiple causes listed on the death certificate. If blacks' increased likelihood of having hypertension appear as an underlying cause of death is driving our results, then controlling for the number of multiple causes should lead to a dramatic decline in the observed racial disparities in diagnoses of hypertension related death. We found that the number of multiple causes listed is positively related to having hypertension appear on the death certificate. However, we also found that the number of multiple causes on the death certificate is unrelated to blacks' increased odds of having their death labeled as hypertension related.

The second way we assessed the validity of the "exclusion" explanation was by using a Heckman selection model. This model allows us to examine the hypertension death diagnosis as a two step process. The first step involves having hypertension listed on the death certificate as an underlying/contributing cause of death, while the second step entails having hypertension listed as the underlying cause as opposed to a contributing cause of death. Overall, the Heckman model allows us to estimate the likelihood that a death is diagnosed with hypertension as an underlying cause given that hypertension appears on the death certificate while accounting for the factors that lead to hypertension being listed on the death certificate. Our results revealed that blacks are significantly more likely to have hypertension appear somewhere on the death certificate—as shown above. Furthermore, when hypertension was listed on the death certificate blacks were approximately two-times more likely to have hypertension listed as an underlying cause across the entire life course. While the exclusion of multiple causes on the death certificate is a potentially viable mechanism leading to higher diagnoses of hypertension related mortality among blacks, our supplemental analysis suggests that this mechanism plays a minimal role at best.

The third—and most plausible in our opinion—explanation for significant racial dispar-

ities in diagnosing hypertension related deaths is statistical discrimination. As mentioned above, Balsa and colleagues assert that "[if] the underlying prevalence of the illness is associated with race, the doctor might take race into account in deciding about the diagnosis" (229). In the case of death diagnoses, statistical discrimination implies that doctors draw on the widely known relationship between race and hypertension prevalence to inform their decision to diagnose a death as hypertension related. In other words, race becomes a proxy for a decedent's hypertension status among health professionals which readily leads to higher reports of hypertension related deaths among blacks than among whites. Thus, black deaths would be erroneously shifted from ischemic or other heart disease categories to hypertensive heart disease, and from nephritis, nephrosis and other renal disease categories to hypertensive renal disease—and/or vice versa for whites.

We believe that statistical discrimination is the most plausible of the three aforementioned explanations for racial disparities in labeling hypertension deaths for three reasons. First, the racial disparities in labeling hypertension deaths persist up to the oldest ages (i.e., 100+) where risk factors such as hypertension play a minimal role (i.e., disparities in mortality and risk factor prevalence generally converge). Second, controlling for a variety of factors related to the aforementioned explanations (e.g., number of multiple causes, hypertension as an underlying cause, hypertension prevalence, race-by-hypertension interactions to test severity arguments) had no effect on the racial disparity in diagnosing hypertension mortality. And third, existing research indicates that statistical discrimination exists in health care settings, and, more importantly, has been documented in hypertension diagnoses (Balsa, McGuire & Meredith 2005; Loring & Powell 1988; Green et al. 2007; Perneger et al. 1995; van Ryn, Burgess, Malat & Griffin 2006). If statistical discrimination in hypertension related deaths is an extension of a broader racial bias operating among health professionals and within the medical community, then programs designed to undermine erroneous beliefs about the connections between race, disease and mortality would be the appropriate policy response—

which would lead to more accurate hypertension related mortality estimates.

## Conclusion

Blacks in the U.S. face a widespread system of disadvantage where they are often treated differently than their white counterparts (Bonilla-Silva 1997; Loring & Powell 1988; Pager 2003; Pager & Quillian 2005; Royster 2003; Yinger 1993). These differences in treatment within and across a variety of social institutions undoubtedly shape the observed racial disparities in socioeconomic status, health and mortality. Our analysis reveals that hypertension, both a cause and consequence of other racial disparities, is more readily invoked as a cause of death among blacks. The results further suggest that the importance of disparities in hypertension prevalence for mortality disparities may be overstated, and that policies designed to curb disparities in prevalence may not translate into similar reductions in black/white mortality disparities. There are a variety of potential sources of this labeling disparity, such as severity of hypertension and access to quality health care, all of which have real consequences for health research and policies that focus on race. Future research should critically analyze the aforementioned sources of racial disparities in diagnosing hypertension related deaths so as to better inform policies designed to alleviate the persistent racial inequities in vitality.

## Notes

[1]We define hypertension related death as deaths that are directly attributed to hypertension (i.e., underlying) and deaths where hypertension is listed as a contributing cause of death—deaths from cardiovascular disease, stroke and renal disease which represent the bulk of mortality attributable to hypertension.

[2]The disparities are nearly synonymous to those using ICD 10 codes I10 to I15.

[3]The term "hypertension related" mortality refers to the mortality rate where the numerator consists of all deaths with hypertension is listed as an underlying or contributing cause, and the denominator is the person years lived in the interval.

[4]The county of occurrence variable consists of FIPS codes for counties with populations greater than 100,000. Counties with populations less than 100,000 are lumped together into one category that contains deaths from all counties with small populations in the same state.

[5]This definition excludes pulmonary hypertension.

[6]The final model for men in the sample of deaths prior to 1995 shows an insignificant racial disparity in hypertension labeling.

[7]We also used Cox proportional hazards to see if hypertension prevalence contributed to blacks' increased odds of dying. The results parallel those presented here. We focus on the sources of labeling disparities because they are an essential aspect of the cause-specific mortality rates that are often cited by scholars when discussing the consequences of racial disparities in hypertension prevalence.

[8]We do not report confidence intervals because the MCD data contains the entire population—as opposed to a sample—of deaths between 2000 and 2004.

[9]We also ran logistic regression models that included an interaction term between race and pre-existing hypertension status to ascertain whether the significant disparity in diagnosing hypertension related deaths was exclusive to those with pre-existing hypertension. The results (available upon request) revealed an insignificant negative interaction between race and pre-existing hypertension (i.e., the disparity was modestly smaller among men with hypertension). Hence, black men's increased odds of having their death diagnosed as hypertension is a product of significant labeling disparities among men with *and* without pre-existing hypertension.

[10]We are unable to control for both hypertension and diabetes in the same model as they appear on different condition checklists in the NHIS data. The diagnosis of diabetes on the death certificate, however, is a very good proxy for the effect of diabetes on hypertension labeling disparities (i.e., those with diabetes are approximately six times more likely to have diabetes listed as an underlying/contributing cause of death).

[11]A number of these risk factors appear on NHIS supplements in different years. Smoking, for example, appears on an NHIS supplement in a few years, but is not included in the primary survey. The exclusion of these risk factors from the larger survey for a number of years and the already small sample size led us to exclude them from the analysis here.

[12]We recognize that low blood pressure (i.e., blood pressure less than 90 mm Hg systolic, or less than 40 mm Hg diastolic) is inversely related to mortality risks. This relationship, though, shifts to positive once we move beyond the minimum blood pressures needed to

sustain life.

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Figure 1: Racial Disparities in the Age-Specific Proportion of Deaths Attributable to Hypertension, Stroke and Ischemic Heart Disease by Sex, 2000-2004

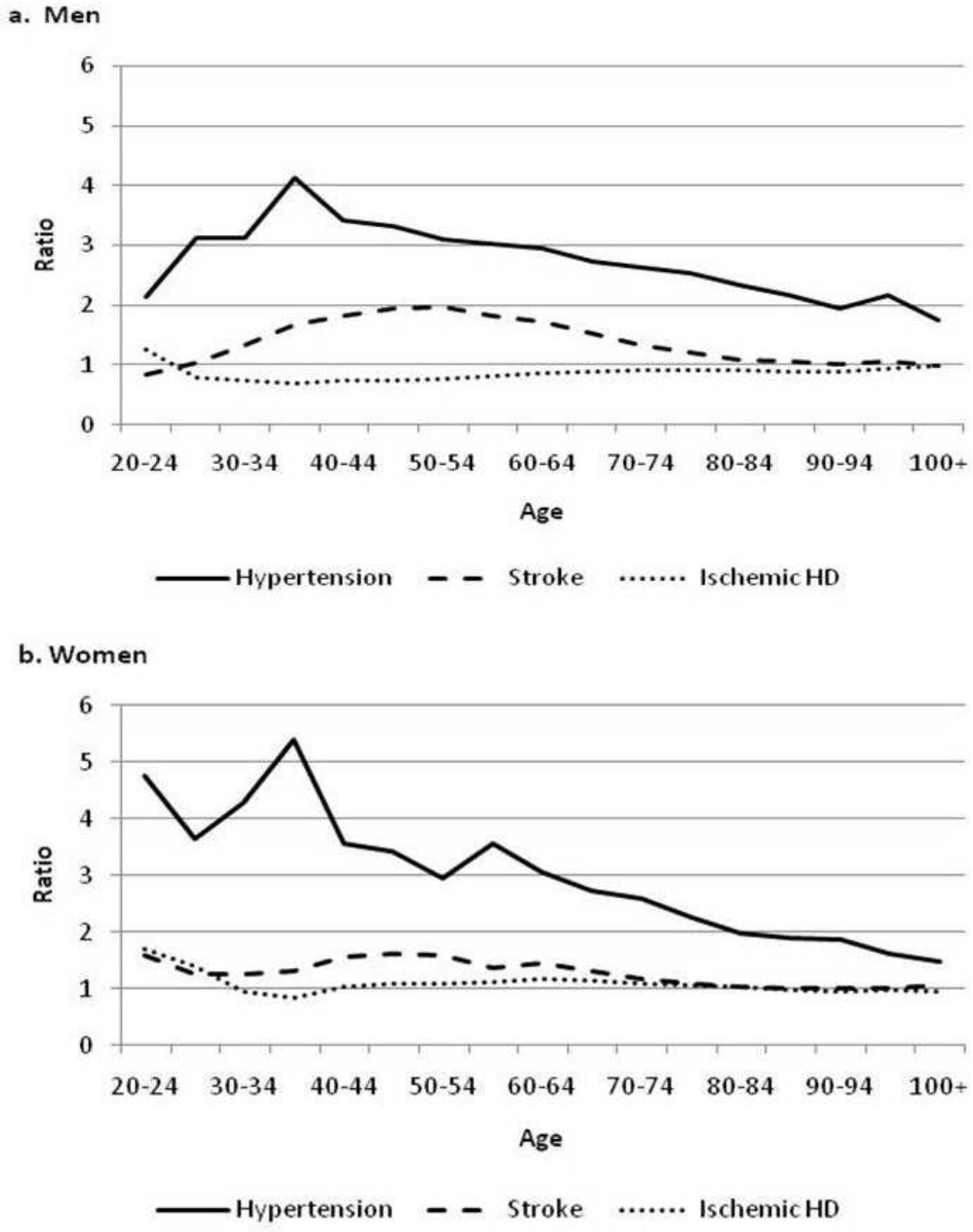
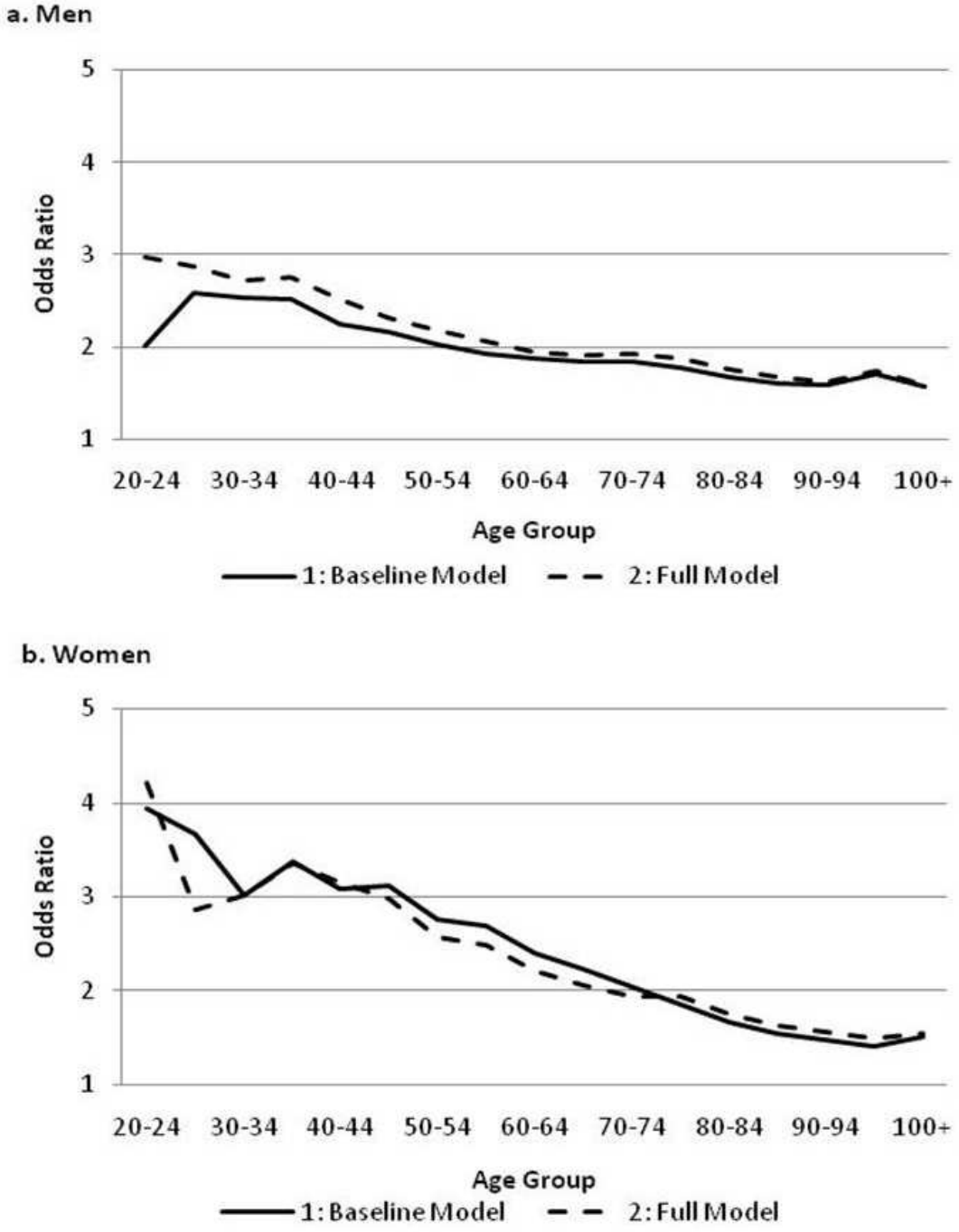


Figure 2: Logistic Regression of Hypertension as an Underlying/Contributing Cause on Race, SES and Other Characteristics by Sex, 2000-2004




Notes: Baseline model contains terms for year. The full model contains terms for number of multiple causes, education, place of death, marital status, diabetes as an underlying/contributing cause of death, and county fixed effect.

**Table 1: Age-Adjusted Cause-Specific Mortality Rates by Race and Sex**

| | | Men | | |
|---|---|---|---|---|
| *Cause* | *White* | *Black* | *Ratio (B/W)* | *Diff. (B-W)* |
| HIV | 1.3 | 13.2 | 9.8 | 11.9 |
| Cancer | 92.4 | 126.7 | 1.37 | 34.2 |
| Diabetes | 9.9 | 20.8 | 2.1 | 10.9 |
| Homicide | 1.5 | 15.1 | 10.3 | 13.7 |
| Stroke | 46.9 | 77.7 | 1.7 | 30.8 |
| Heart Dis. | 287 | 390.3 | 1.36 | 103.3 |
| *Isch. HD* | 199.4 | 237.3 | 1.19 | 38.0 |
| *Hypertension* | 5.9 | 20.1 | 3.39 | 14.1 |
| *MC Hyp*[a] | 74.8 | 170.4 | 2.3 | 95.6 |
| *Renal Dis.* | 16.7 | 39.1 | 2.3 | 22.3 |
| *Hyp. Ren. Dis.* | 2.5 | 11.2 | 4.4 | 8.7 |
| | | Women | | |
| *Cause* | *White* | *Black* | *Ratio (B/W)* | *Diff. (B-W)* |
| HIV | 0.2 | 5.4 | 21.81 | 5.2 |
| Cancer | 83.6 | 97.6 | 1.17 | 14 |
| Diabetes | 9.8 | 24.8 | 2.53 | 15 |
| Homicide | 0.7 | 2.6 | 3.57 | 1.9 |
| Stroke | 74.2 | 97 | 1.31 | 22.8 |
| Heart Dis. | 294.3 | 393.3 | 1.34 | 99 |
| *Isch. HD* | 182.5 | 227.8 | 1.25 | 45.3 |
| *Hypertension* | 19.3 | 53.9 | 2.79 | 34.5 |
| *MC Hyp*[a] | 106.3 | 204.1 | 1.92 | 97.8 |
| *Renal Dis.* | 17.3 | 41.7 | 2.41 | 24.4 |
| *Hyp. Ren. Dis.* | 3.37 | 13.5 | 4.01 | 10.1 |

*Note: The Heart Disease category does not include Stroke deaths.*
*a. MC Hyp stands for Multiple Cause Hypertension Mortality and refers to mortality rates with the number of deaths where hypertension appeared on the death certificate as an underlying or contributing cause.*

**Table 2: Selected Descriptive Statistics for MCD and NHIS data by race and sex**

| | MCD | | | |
|---|---|---|---|---|
| | Men | | Women | |
| | *White* | *Black* | *White* | *Black* |
| N | 4641068 | 674158 | 5001534 | 668167 |
| *Hypertension* | | | | |
| % Contributing | 8.5 | 12.8 | 11.7 | 17.3 |
| % Underlying | 0.7 | 1.5 | 1.1 | 2.2 |
| % of Contributing that are Underlying | 7.9 | 11.9 | 9.3 | 12.5 |
| *Education (%)* | | | | |
| LTHS | 26.3 | 36.6 | 27.9 | 36.9 |
| HS Dip. | 38.2 | 36 | 42.5 | 33.8 |
| Some Coll. | 10.6 | 7.7 | 10.5 | 8.5 |
| College + | 19.2 | 8.7 | 13.4 | 10.2 |
| Marital Status (%) | | | | |
| Single | 9.6 | 23.7 | 6.5 | 15.1 |
| Married | 57.1 | 42.6 | 24.9 | 20.7 |
| *Place of Death (%)* | | | | |
| Inpatient | 38.7 | 44 | 35.6 | 46.9 |
| Outpatient | 9.9 | 16.5 | 5.9 | 12.8 |
| Residence | 45 | 32 | 53.5 | 36.1 |
| | NHIS | | | |
| | Men | | Women | |
| | *White* | *Black* | *White* | *Black* |
| N (Baseline) | 34204 | 5038 | 46244 | 9533 |
| % Pre-Ex. Hyp. | 20.4 | 26.4 | 18.8 | 29.2 |
| Deaths (95-02) | 8303 | 1297 | 10245 | 1828 |
| % Hyp. Related | 8.4 | 14.1 | 11.2 | 16 |
| *Education (%)* | | | | |
| LTHS | 30.4 | 50.4 | 30.2 | 48.2 |
| HS Dip. | 33.4 | 28.6 | 38.2 | 29.6 |
| Some Coll. | 16.5 | 13.1 | 17.2 | 14 |
| College + | 18.9 | 6.2 | 13.4 | 6.5 |
| Missing | 0.9 | 1.7 | 0.9 | 1.7 |
| Mean Income | 29144 | 19494 | 25537 | 18161 |
| *Marital Status (%)* | | | | |
| Single | 9.4 | 17.1 | 7.2 | 16.8 |
| Married | 76.1 | 56.2 | 54.4 | 32.5 |
| *BMI (%)* | | | | |
| 25-30 | 41.6 | 39.3 | 26.8 | 30.7 |
| 30< | 20 | 22.8 | 22.1 | 38.4 |

Note: NHIS estimates are not weighted.

**Table 3: Logistic Regression of Pre-Existing Hypertension on Race, SES and Health of NHIS Respondents - Men (Odds Ratios)**

| | Model 1 | Model 2 | Model 3 | Model 4 |
|---|---|---|---|---|
| Race | 1.385*** | 1.564*** | 1.560*** | 1.686*** |
| Age | 1.086*** | 1.072*** | 1.072*** | 1.076*** |
| Age-Squared | 0.999*** | 0.999*** | 0.999*** | 0.999*** |
| *Education (Ref = HS)* | | | | |
| LTHS | | 0.805*** | 0.801*** | 0.915** |
| Some College | | 1.015 | 1.03 | 0.985 |
| College+ | | 0.985 | 0.999 | 0.918* |
| Missing | | 0.674** | 0.670** | 0.753 |
| Log Income | | 1.139*** | 1.139*** | 1.073*** |
| Missing Inc. | | 1.276*** | 1.274*** | 1.162*** |
| *Marital Status (Ref = Single)* | | | | |
| Married | | 1.411** | 1.413*** | 1.315*** |
| Divorced | | 1.180** | 1.188** | 1.162* |
| Widowed | | 1.315*** | 1.314** | 1.225** |
| Missing | | 1.276 | 1.285 | 1.309 |
| *Region (Ref = West)* | | | | |
| Northeast | | | 1.216** | 1.154 |
| South | | | 1.157 | 1.185** |
| Midwest | | | 1.182 | 1.122 |
| *Subj.Health (Ref = Exc)* | | | | |
| Very Good | | | | 1.158*** |
| Good | | | | 0.895** |
| Fair | | | | 0.574*** |
| Poor | | | | 0.356*** |
| Missing | | | | 0.519** |
| *BMI (Ref= less than 25)* | | | | |
| 25-30 | | | | 1.501*** |
| 30< | | | | 1.904*** |
| Missing | | | | 1.147 |
| -2 Log-Lik. | 39925 | 39654 | 39628 | 38492 |
| df | 13 | 23 | 26 | 34 |

Notes: * = p<0.05, **= p<0.01, ***= p<0.001. All models include dummy variables for year of NHIS survey.

**Table 4: Logistic Regression of Pre-Existing Hypertension on Race, SES and Health of NHIS Respondents - Women (Odds Ratios)**

| | Model 1 | Model 2 | Model 3 | Model 4 |
|---|---|---|---|---|
| Race | 1.659*** | 1.747*** | 1.716*** | 1.766*** |
| Age | 1.100*** | 1.095*** | 1.095*** | 1.094*** |
| Age-Squared | 0.999*** | 0.999*** | 0.999*** | 0.999*** |
| *Education (Ref = HS)* | | | | |
| LTHS | | 0.861*** | 0.852*** | 0.942* |
| Some College | | 0.882*** | 0.893*** | 0.866*** |
| College+ | | 0.776*** | 0.783*** | 0.747*** |
| Missing | | 0.736* | 0.736* | 0.792 |
| Log Income | | 1.020*** | 1.023 | 1.004 |
| Missing Inc. | | 1.081* | 1.075* | 1.075* |
| *Marital Status (Ref = Single)* | | | | |
| Married | | 1.129** | 1.130** | 1.133** |
| Divorced | | 0.94 | 0.949 | 0.978 |
| Widowed | | 1.004 | 1.005 | 0.99 |
| Missing | | 0.821 | 0.828 | 0.903 |
| *Region (Ref = West)* | | | | |
| Northeast | | | 1.166 | 1.108 |
| South | | | 1.201** | 1.236*** |
| Midwest | | | 1.187* | 1.145 |
| *Subj.Health (Ref = Exc)* | | | | |
| Very Good | | | | 0.98 |
| Good | | | | 0.851*** |
| Fair | | | | 0.561*** |
| Poor | | | | 0.342*** |
| Missing | | | | 0.608** |
| *BMI (Ref= less than 25)* | | | | |
| 25-30 | | | | 1.444*** |
| 30< | | | | 1.698*** |
| Missing | | | | 0.951 |
| -2 Log-Lik. | 54169 | 54044 | 54010 | 52756 |
| df | 13 | 23 | 26 | 34 |

Notes: * = p<0.05, **= p<0.01, ***= p<0.001. All models include dummy variables for year of NHIS survey.

**Table 5: Logistic Regression of Hypertension as Underlying/Contributing Cause of Death on Race, SES and Health of NHIS Respondents - Men (Odds Ratios)**

| | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|
| Race | 2.047*** | 1.980*** | 2.061*** | 2.044*** | 2.135*** |
| Pre-Existing Hyp. | | 1.699*** | 1.700*** | 1.701*** | 1.694*** |
| Age | 1.140*** | 1.136*** | 1.107** | 1.107** | 1.089* |
| Age-Squared | 0.999*** | 0.999*** | 0.999** | 0.999** | 1.000* |
| *Education (Ref = HS)* | | | | | |
| LTHS | | | 1.097 | 1.094 | 1.125 |
| Some College | | | 1.018 | 1.015 | 0.924 |
| College+ | | | 1.167 | 1.16 | 1.214 |
| Missing | | | 0.624 | 0.629 | 0.807 |
| Log Income | | | 0.957 | 0.958 | 0.962 |
| Missing Inc. | | | 0.881 | 0.882 | 0.873 |
| *Marital Status (Ref = Single)* | | | | | |
| Married | | | 2.222*** | 2.208*** | 2.120*** |
| Divorced | | | 2.010** | 2.003** | 1.865** |
| Widowed | | | 1.786* | 1.767* | 1.628* |
| Missing | | | 0.001 | 0.001 | 0.001 |
| *Region (Ref = West)* | | | | | |
| South | | | | 0.984 | 0.943 |
| Midwest | | | | 1.019 | 1.062 |
| West | | | | 0.939 | 0.913 |
| *Subj. Health (Ref = Exc.)* | | | | | |
| Very Good | | | | | 0.826 |
| Good | | | | | 1.149 |
| Fair | | | | | 0.997 |
| Poor | | | | | 0.772 |
| Missing | | | | | 2.711* |
| *BMI (Ref= less than 25)* | | | | | |
| 25-30 | | | | | 1.472*** |
| 30< | | | | | 1.590*** |
| Missing | | | | | 0.142 |
| Diabetes Contributing | | | | | 3.392*** |
| -2 Log-Lik. | 5550 | 5512 | 5485 | 5484 | 5242 |
| df | 6 | 7 | 17 | 20 | 29 |

Notes: * = p<0.05, **= p<0.01, ***= p<0.001. All models control for year of NHIS survey, year of survey squared and year of death.

**Table 6: Logistic Regression of Hypertension as Underlying/Contributing Cause of Death on Race, SES and Health of NHIS Respondents - Women (Odds Ratios)**

| | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|
| Race | 1.730*** | 1.691*** | 1.745*** | 1.846*** | 1.704*** |
| Pre-Existing Hyp. | | 1.490*** | 1.487*** | 1.488*** | 1.485*** |
| Age | 1.035 | 1.030 | 1.017 | 1.017 | 1.000 |
| Age-Squared | 1.000 | 1.000 | 1.000 | 1.000 | 1.000 |
| *Education (Ref = HS)* | | | | | |
| LTHS | | | 0.938 | 0.969 | 0.978 |
| Some College | | | 1.073 | 1.041 | 0.993 |
| College+ | | | 0.789* | 0.805 | 0.814 |
| Missing | | | 1.154 | 1.175 | 1.428 |
| Log Income | | | 0.986 | 0.985 | 0.985 |
| Missing Inc. | | | 0.92 | 0.933 | 0.935 |
| *Marital Status (Ref = Single)* | | | | | |
| Married | | | 1.369* | 1.371* | 1.313 |
| Divorced | | | 1.017 | 0.993 | 0.937 |
| Widowed | | | 1.405 | 1.412* | 1.343 |
| Missing | | | 1.543 | 1.499 | 1.701 |
| *Region (Ref = West)* | | | | | |
| South | | | | 0.728* | 0.666** |
| Midwest | | | | 0.700*** | 0.712 |
| West | | | | 0.895 | 0.869 |
| *Subj. Health (Ref = Exc.)* | | | | | |
| Very Good | | | | | 0.879 |
| Good | | | | | 0.749* |
| Fair | | | | | 0.806 |
| Poor | | | | | 0.631*** |
| Missing | | | | | 0.521 |
| *BMI (Ref= less than 25)* | | | | | |
| 25-30 | | | | | 1.185* |
| 30< | | | | | 1.355*** |
| Missing | | | | | 0.521 |
| Diabetes Contributing | | | | | 3.220*** |
| -2 Log-Lik. | 8445 | 8410 | 8389 | 8365 | 8069 |
| df | 6 | 7 | 17 | 20 | 29 |

Notes: * = p<0.05, **= p<0.01, ***= p<0.001. All models control for year of NHIS survey, year of survey squared and year of death.

**Table A1: Multiple Cause of Death File (MCD) Variable Descriptions**

| Variable | Description |
|---|---|
| *Hypertension* | |
| Underlying | Underlying Cause coded as ICD-10 codes I10 and I12. |
| Contributing | Any Record Axis Condition coded as ICD-10 codes I10 and I12 |
| Diabetes | Any Record Axis Condition coded as ICD-10 codes E10.0 to E14.9 |
| County of Occurrence | Fips codes for county populations greater than 100,000. Counties less than 100,000 are grouped within states. |
| Place of Occurrence | Place of death coded as 4 categories (Hospital Inpatient, Hospital Outpatient, Residence, Other) |
| Autopsy Status | Dummy variable indicating whether an autopsy was performed. Only available for 2003 and 2004. |
| *Decedent Chars.* | |
| Age | Age of decedent at death |
| Race | Race of decedent (i.e., black and white) |
| Education | Education on death certificate coded as four categories (Less than High School, High School, Some College, College) |
| Marital Status | Marital status coded as 4 categories (Never Married (i.e., Single), Married, Widowed, Divorced) |

Notes: These variables are derived from the 2000 to 2005 MCD data sets from the National Center for Health Statistics

**Table A2: National Health Interview Study Linked Mortality File (NHIS) Variable Descriptions**

| Variable | Description |
|---|---|
| *Hypertension* | |
| Pre-Existing Report | Hypertension identified as an existing medical condition on heart disease checklist. |
| Cause of Death | Hypertension listed as an underlying or contributing cause of death. Uses ICD-10 codes I10 and I12. |
| Diabetes | Diabetes listed as an underlying or contributing cause of death. |
| Region | Region of Residence grouped into 4 categories (Northeast, South, Midwest, West) |
| *Age* | |
| Survey Age | Age of respondent at time of NHIS survey. |
| Follow-up Age | Age at death used in analyses of NHIS deaths. |
| Race | Race of respondent (i.e., black and white) |
| Education | Education of NHIS respondent coded as four categories (Less than High School, High School, Some College, College) |
| Marital Status | Marital status coded as 4 categories (Never Married (I.e., Single), Married, Widowed, Divorced) |
| Household Income | Household income grouped into 27 categories. We use midpoints for categories less than the median, and fit a Pareto curve to estimate mean incomes above the median. We use ordinal logistic regression to impute missing income. |
| Subj. Health Status | Health status indicated by respondent in NHIS interview consisting of 5 categories (excellent, very good, good, fair, poor). |
| BMI | Body Mass Index calculated using the height and weight of respondent separated into 3 categories (less than 25, 25-29, greater than 30) |

Notes: The NHIS survey variables are derived from the years 1986 to 1996, while the information on deaths (i.e., year, cause of death) come from the Mortality Follow-up File from the years 1995 to 2002.

**Table A3: Selected Results from Logistic Regression of Hypertension as Underlying/Contributing Cause of Death on Race, Socioeconomic Status and Health of NHIS Respondents - Five Samples: Males (Odds Ratios Shown)**

Sample 1: Deaths After 1995

| | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|
| Race | 2.047*** | 1.980*** | 2.061*** | 2.044*** | 2.135*** |

Sample 2: Deaths Before 1995

| | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|
| Race | 1.612** | 1.553* | 1.596** | 1.699** | 1.323 |

*Sample 3: Deaths Less Than 5 Yrs after Survey*

| | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|
| Race | 1.655*** | 1.597*** | 1.789*** | 1.851*** | 1.587** |

*Sample 4: Deaths After 1995 and Less than 5 Yrs after Survey*

| | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|
| Race | 1.585* | 1.538* | 1.920** | 1.899** | 1.889** |

*Sample 5: All Deaths 1986 to 2002*

| | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|
| Race | 1.917*** | 1.853*** | 1.949*** | 1.969*** | 1.924*** |
| *Controls* | | | | | |
| Hypertension | N | Y | Y | Y | Y |
| Education | N | N | Y | Y | Y |
| Income | N | N | Y | Y | Y |
| Marital Status | N | N | Y | Y | Y |
| Region | N | N | N | Y | Y |
| Subj. Health | N | N | N | N | Y |
| Diab. Cont. | N | N | N | N | Y |
| BMI | N | N | N | N | Y |

Notes: * = p<0.05, **= p<0.01, ***= p<0.001. All models include dummy variables for year of NHIS survey.

**Table A4: Selected Results from Logistic Regression of Hypertension as Underlying/Contributing Cause of Death on Race, Socioeconomic Status and Health of NHIS Respondents - Five Samples: Females (Odds Ratios Shown)**

| | Model 1 | Model 2 | Model 3 | Model 4 | Model 5 |
|---|---|---|---|---|---|
| Sample 1: Deaths After 1995 | | | | | |
| Race | 1.730*** | 1.691*** | 1.745*** | 1.846*** | 1.704*** |
| Sample 2: Deaths Before 1995 | | | | | |
| Race | 2.045*** | 2.016*** | 2.112*** | 2.109*** | 1.902*** |
| *Sample 3: Deaths Less Than 5 Yrs after Survey* | | | | | |
| Race | 1.830*** | 1.798*** | 1.875*** | 1.874*** | 1.706*** |
| *Sample 4: Deaths After 1995 and Less than 5 Yrs after Survey* | | | | | |
| Race | 1.835*** | 1.806*** | 1.878*** | 1.861*** | 1.594** |
| *Sample 5: All Deaths 1986 to 2002* | | | | | |
| Race | 1.824*** | 1.786*** | 1.830*** | 1.903*** | 1.756*** |
| *Controls* | | | | | |
| Hypertension | N | Y | Y | Y | Y |
| Education | N | N | Y | Y | Y |
| Income | N | N | Y | Y | Y |
| Marital Status | N | N | Y | Y | Y |
| Region | N | N | N | Y | Y |
| Subj. Health | N | N | N | N | Y |
| Diab. Cont. | N | N | N | N | Y |
| BMI | N | N | N | N | Y |

Notes: * = p<0.05, **= p<0.01, ***= p<0.001. All models include dummy variables for year of NHIS survey.