INTRODUCTION

Hypertension is one of this century's most pressing public health challenges. Over the past two decades, its prevalence has increased within the United States, as well as across the globe (Watson, 2008). Defined as a sustained elevated blood pressure (systolic BP>140 mm/Hg and diastolic BP>90 mm/Hg), hypertension is arguably the most important modifiable risk factor for cardiovascular, cerebrovascular, and renal disease.

Hypertension affects approximately 29% of adults in the United States. African Americans have a higher prevalence rate than any other racial or ethnic group in this country- over 40%. African Americans also experience the onset of hypertension at earlier ages and are significantly less likely than Whites to have hypertension under control (Center for Disease Control [CDC], 2005; Howard, et al., 2006; Jamerson, 2004; Morenoff, et al., 2007). The disease is, on average, more severe in African Americans --- higher rates of morbidity and mortality associated with end-stage renal disease, congestive heart failure, and stroke are also observed in this group (Richardson & Piepho, 1999).

Despite decades of biomedical research, causal pathways of essential hypertension have not been fully identified, and the causes of differences in prevalence, onset, and severity of this disease among African Americans are not well understood. Generally, four pathways of blood pressure modulation have been identified – the rennin-angiotensin system which leads to vasoconstriction; nitric oxide *dependent* vasodilation pathways; nitric oxide *independent* vasodilation pathways; and sodium balance (Williams, et al., 2004). While it is unknown whether one pathway contributes more to the onset of hypertension than others, there is growing evidence to suggest that hypertension occurs as a result of interactions among a *variety of genes that are in disequilibrium* (Williams, et al., 2004) as opposed to the presence or absence of a

specific gene in a given subpopulation or the interaction of a specific gene with a specific environmental factor that triggers this response in a given subpopulation.

For over thirty years, the National Heart, Lung, and Blood Institute (NHLBI) has coordinated guidelines designed to increase the awareness, treatment, and control of hypertension. Beginning in 1984, with its Third Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure (JNC III), the NHLBI began listing Black patients as a "special population" and suggested that these patients responded better to diuretics than to β-blockers. In 1988, with its fourth report, the JNC repeated this recommendation and further placed Black hypertensive patients in direct comparison with their White counterparts, stating that the former group did not respond as well as Whites to β-blockers or ACE inhibitors. The JNC V, released in January of 1993 echoed these prior recommendations, pressing for research on whether other racial and ethnic groups responded differently than Whites to treatment for hypertension. Interestingly, the definition of who is Black or how race is defined in these studies is often unclear. Moreover, how individuals of mixed-race should be treated is not addressed.

Thus far, the terms *African American* and *Black* have been used interchangeably. While the authors acknowledge that the former describes a specific ethnic group and the latter a "racial" group, it is unclear given the ambiguity with which race is defined in previous studies whether researchers confine their Black sample to Americans of West African lineage who are native born, whose parents and grandparents are also native born, are descendants of slaves, and who report little or no racial admixture. Although we assume that the findings of our study pertain primarily to African Americans, we have no way of testing the accuracy of this claim, thus the use of the broader racial category *Black* in the rest of this article.

How Different is Different?

Recent research has highlighted the fact that Blacks and Whites respond similarly to antihypertensive drugs even when used as monotherapy agents. In a review of clinical trials conducted from 1984 to 1998, Sehgal (2004) showed that not only were Blacks in these studies more likely to have higher baseline blood pressures than their White counterparts --thus possibly skewing the results to show a weaker response to certain drugs than Whites-- but also that the magnitude of the Black-White differences is much smaller than the variation within each race group. In other words, the so-called Black-White difference in response to pharmacological interventions is more a matter of the direction and interpretation of the clinical trial results than real, significant differences in response rates by race.

In the same year, Mokwe and colleagues (2004) published a study on the use of one particular ACE inhibitor. When they examined response rates to the drug by race, they found a lesser response to treatment among the Black subjects compared to their White counterparts. However, once they controlled for individual characteristics such as obesity, kidney function, and diabetes, they found most of the difference in blood pressure response eliminated, leading them to conclude, as did Seghal, that most of the variability of blood pressure response was within and not between racial groups and that individual-level factors greatly contributed to the ostensible racial difference.

Given these findings, our project examines the extent to which race affects specific treatments for hypertension. Do medical practitioners prescribe different drugs for Black and White individuals that mirror the recommendations put forth by the JNC? Or is race less of a factor in treatment of hypertensive patients as suggested by the work of Seghal and others?

There are three main contributions our study makes to the literature. First, it adds to the growing literature on the role that national guidelines play in the day-to-day treatment of patients (Pedone and Lapane, 2003; Grimshaw and Russel, 1993; Lomas, et.al., 1989). Second, it contributes to the emergent literature on how practitioners use race in their treatment of specific diseases (see Balsa, McGuire, and Meredith, 2005; Institute of Medicine, 2003; Okelo,et.al., 2001; Peterson, et.al., 1997; Tamayo-Sarver, et.al., 2003; Todd, et.al., 1993; Van Ryn and Burke, 2000). Third, it functions as a cogent example of how, in the presence of uncertainty, race is used to dictate specific treatment of disease.

LITERATURE REVIEW

Do doctors prescribe different treatments for Blacks and Whites with hypertension and if so, why? The reasons for these racial differences map nicely onto prevailing theories about racial disparities in health. For the sake of brevity, we cluster these theories into three separate categories – physician adherence to guidelines; access to resources; and biological difference.

Guideline Adherence

For over thirty years, the National Heart, Lung, and Blood Institute (NHLBI) has coordinated guidelines designed to increase the awareness, treatment, and control of hypertension. Adherence to these guidelines has been shown to vary and is generally rated as poor (Pedone and Lapane, 2003; Grimshaw and Russel, 1993; Lomas, et.al., 1989). For example, in an analysis of trends in antihypertensive drug use in the U.S. in 1992 and 1995, Siegel and Lopez show that the JNC V had little effect on prescribing patterns (1997). Self-reports of primary care physicians reveal that 41% of physicians a limited or no familiarity with JNC guidelines (Hyman & Pavlik, 2000). Regardless, physicians are more likely to follow guidelines for minority patients (see Ardery, et.al, 2007).

The guidelines suggest Black hypertensive patients respond differently to certain drugs than White patients. The guidelines do not mention race differences for diuretics, CCBs, or vasodilators such as α-blockers. If physicians followed the JNC recommendations, we would expect Black men and women less likely to take β-blockers and ACE inhibitors while no race difference should be observed for these other medications.

Access to Resources

The link between socioeconomic status (SES) and health conditions has been discussed since at least the mid-nineteenth century (Link and Phelan, 1995). In a seminal study completed in 1906, DuBois demonstrated that health disparities between Whites and Blacks were driven by the poorer economic, social, and environmental conditions faced by the latter group. More than a century later, Blacks on average are still disadvantaged along these lines compared with their White counterparts. In addition to economic factors, persistent race differences in access to health care also contribute to the race gap in health and longevity in the U.S.

Currently, 20% of Blacks are uninsured, compared to 11% of Whites (DeNavas-Walt, Proctor, & Mills, 2004). Availability and type of health insurance may affect the antihypertensive medications doctors prescribe. For example, patients with Medicaid or Medicare and uninsured patients are much more likely to be prescribed β-blockers and diuretics in accordance with the JNC recommendations than patients with HMO insurance plans (Guo, et.al., 2003), perhaps because these drugs are less costly than other antihypertensive drug classes.

Second, higher income provides patients with greater access to newer and more expensive technologies in healthcare, including drugs. Diuretics have been the least costly pharmacological intervention for hypertension for many years. Depending on the time frame examined, β-blockers are second lowest in cost, with CCBs and ACE inhibitors of comparable

cost (Liu and Wang, 2008). Patients with limited incomes may be more likely to take less expensive drugs. Given that Black adults on average have lower income than their White counterparts, income should also be a significant factor in their access to specific antihypertensive medications

Biological Difference

Salt Sensitivity and Nutrition

In conceptualizing biological difference by race, we do not suggest that race is a real biological category. We do note that structural constraints centuries in the making can come together to leave an indelible imprint within the bodies of those they affect. These biological responses are not genetic in nature but contingent on specific environmental and political factors that produce different health outcomes for those groups affected.

Approximately 60% of hypertensive individuals are responsive to sodium intake (Weinberger, 1996). That is, given a certain amount of sodium, an individual's mean arterial blood pressure will increase temporarily. This response to sodium indicates that the cells are releasing water to equilibrate the sodium level between the cells and the bloodstream, thus increasing pressure on the blood vessel walls. Individuals who exhibit an increased response to sodium intake are said to be *salt sensitive*. Given the high percentage of hypertensive individuals who are salt sensitive, the use of diuretics, which promote the loss of water volume in the body, as the first line of pharmacological intervention makes sense.

The fact that increased salt sensitivity is linked to hypertension has been well established. It has also been found in the U.S. that Blacks are more likely to be salt sensitive than their White counterparts (Kaplan, 1994; Taylor & Elis, 2002; Weinberger, 1996). While no agreement has been reached as to the mechanisms behind this biological difference, theories leaning towards an

inherent *genetic* predisposition in Blacks have been around since the 1960s (Kaufman & Hall, 2003). They have subsequently been summarily discredited (for some examples, see Cooper, Kaufman, & Ward, 2003; Curtin, 1992; Kaufman & Hall, 2003; Krieger, 2005), yet they keep resurfacing. They are still part of the rationale behind why some clinical trials treat Blacks as a distinct *genetic* population in studies of hypertension. The salt-slavery hypothesis was the subject of a controversial and widely publicized working paper by Fryer, Cutler, and Glaeser (2005). In the popular media, it was the subject of an episode of the *Oprah Winfrey Show* in 2007. With such prominent advocates, discredited genetic theories such as these nonetheless continue to exert an influence on both the popular imagination and the research community.

One mechanism of salt sensitivity that has received increased attention is dietary quality. Neighborhoods with high concentrations of Black residents have reduced access to healthy food options, regardless of income (Baker, et.al, 2006). In 1996, 28% of Blacks were reported to have a poor-quality diet, compared to 16% of Whites. Of all racial groups, Blacks were more likely to have diets high in fat and low in fruits, vegetables, and whole grains. Their diets also included lower levels of calcium and potassium intake.

Diet plays a significant role in mediating hypertension risk. In a study of the Dietary Approaches to Stop Hypertension (DASH) trial, Akita, et.al, (2003) observed that individuals who consumed the DASH diet were much less salt-sensitive than individuals on the control diet. The DASH diet's high potassium and calcium contents were credited for this difference. The higher percentage of Blacks who are salt sensitive, then, may be due to differences in dietary quality.

Comorbidities

When deciding on treatment options for hypertension, some comorbidities are known to affect prescribing patterns. For example, while no antihypertensive therapy is specifically contraindicated for patients with diabetes, ACE inhibitors are generally preferred over diuretics and β-blockers, which can worsen glucose tolerance. Similarly, ACE inhibitors have been found to decrease mortality in patients with heart failure (JNC V). Blacks experience higher rates of diabetes and heart failure than Whites, which could increase their probability of getting an ACE inhibitor for hypertension. Finally, obesity is strongly associated with hypertension. No specific antihypertensive drug therapy is recommended for obese individuals.

METHODS

Sample

The analyses are based on data from the Third National Health and Nutrition Examination Survey (NHANES III), conducted by the National Center for Health Statistics from 1988 to 1994. NHANES III is a cross-sectional, stratified multistage probability sample of the civilian non-institutionalized U.S. population age 2 months to 90 years, with an oversample of several population segments including non-Hispanic Black respondents. Data were collected in household interviews, detailed clinical examinations, and laboratory tests. Details of the sampling design and protocol have been reported elsewhere (National Center for Health Statistics, 1994).

We restricted the sample to non-Hispanic White and Black adults age 25 to 75 years (N=9,922). There were no missing observations on most demographic variables, including age, sex, region of residence, and race/ethnicity. Self-reported hypertension was missing in only 0.3% of the analysis sample. The highest proportion of missing values was for systolic and diastolic

blood pressure measured during the clinical examination, with 10.7% missing observations. Respondents with missing values were excluded from the analyses. Older White non-married adults residing in urban areas were more likely to be missing blood pressure information, mostly because they failed to participate in the survey examination.

The time period in which the NHANES III data were collected overlapped with the publication of the JNC Reports that began documenting differences in response to pharmacological treatment for Blacks compared with Whites. While there is generally a lag time between the publication of treatment recommendations and their acceptance and implementation by physicians, the introduction of special treatment regimens for Blacks in 1984 provided ample time for physicians (4 to 10 years) to modify their prescription patterns accordingly.

Measures

Prevalence of hypertension. Three variables were used to define the prevalence of hypertension. First was a self-reported dichotomous variable "Has your doctor ever said you had high blood pressure?" Second was measured blood pressure (BP), calculated as the mean of three BP measurements collected during the survey examination. Individuals were classified as hypertensive if the mean systolic BP>140 mm/Hg or diastolic BP>90 mm/Hg. The third variable was a self-report of whether the subject was currently taking any antihypertensive medications.

Treatment of hypertension. During the household interview, interviewers asked a set of questions about prescription medication taken within the previous month. The respondents were asked to show all medication containers to confirm the accuracy of their reports, and report the health problems for which they took each medication. Pharmacological interventions for hypertension were classified as diuretics, vasodilators, calcium channel blockers, β-blockers, alpha blockers,

and ace inhibitors. Due to the small N, vasodilators and alpha blockers were collapsed into a single "other antihypertensive medications" category.

Note that although this study aims to explore the prescribing patterns of physicians, the data are based on the medications the patients report taking. While we do not have measures of compliance, we also do not have a reason to expect a pronounced systematic race-specific and medication class-specific non-compliance, which would bias our findings.

Socioeconomic position. Education was measured in years of schooling and used in models as a continuous predictor. Health insurance was dichotomized as any versus no insurance.

Demographic variables. Race, classified as non-Hispanic White and non-Hispanic Black, was the main demographic predictor. Age was measured in single years from 25 to 75. Sex and marital status were dichotomous, with male and married, respectively, as a reference. Census region was categorized as Northeast (reference), Midwest, South, and West. Rural/urban residence was a dichotomous classification based on the USDA codes, where urban (reference) included large metropolitan areas and their fringe counties; urban included all other areas.

Biological correlates of hypertension. We included 4 measures collected as a part of a blood biochemistry profile: Sodium (mmol/L), calcium (mmol/L), potassium (mmol/L), and vitamin D (mg/dL). For sodium, higher levels are associated with hypertension; the opposite association has been described for the other three.

Comorbidities. Two self-reported health conditions were included because they could impact the decision about hypertension medication. Diabetes and heart failure were assessed during the interview with a question "Have you ever been told you had diabetes?" and "Has a doctor ever told you that you had congestive heart failure?" Obesity was defined as BMI>30kg/m^2, using height and weight measured during the survey examination.

Statistical Analysis

Univariate and bivariate statistics were used to assess the distribution of key variables among White and Black men and women, and differences across these groups. Logistic models were used to estimate the effect of race on the odds of specific antihypertensive medication class. Sex-stratified models were estimated to test whether sex played a role in prescription patterns, given that much of previous literature relied on findings from studies conducted by the Veterans' Administration and included mostly men. All descriptive and multivariate analyses were adjusted for sampling weights and the complex sampling design. Analyses were conducted using Stata 10.1 (2007, StataCorp, College Station, TX).

RESULTS

Table 1 summarizes the distribution of key variables by race and sex. Black adults were younger, had less education, and were less likely to be insured than Whites. In the full sample, about a quarter of adults reported having been told by a doctor that they had hypertension – however, the proportion was somewhat higher among Black men and women than their White counterparts. Hypertension as measured during the survey medical examination had lower prevalence, likely due in part to the medication the respondents were prescribed – but Black adults had considerably higher prevalence of high BP than White adults.

Among those whose doctor diagnosed hypertension, about half of both Black and White men were taking antihypertensive medication. In terms of specific pharmacological treatments, Blacks were more likely to take diuretics, CCBs, and the 'other' medication class, and less likely to take β-blockers. However, Black women were more likely to take ACE inhibitors than White women while Black men were *less* likely than White men to be on this medication.

Some of these differences could be due to comorbidities and biochemistry profile. A higher percentage of Blacks suffered from heart failure and diabetes, and a considerably larger percentage of Black women were obese than White women. There were relatively small differences in sodium, calcium, and potassium levels by race but White adults had considerably higher blood serum levels of vitamin D than Black adults.

Table 2 summarizes the race differences for specific antihypertensive medications net of potential confounding or mediating variables. Each coefficient in the table, showing the odds ratio of taking a medication for Black adults relative to White adults, is from an independently estimated model. Each line represents a specific antihypertensive medication category; each column a different set of control variables from age only in Model 1 to a full set of predictors in Model 6.

The race differences for most medications were substantial. Based on JNC recommendations, we did not expect differences in the use of diuretics by race. The data showed otherwise: Black men were 73% more likely to be taking diuretics, compared to White men of the same age. This difference was not explained by a comprehensive set of possible mediators. Through Model 6, the race effect gradually became stronger: all else equal, Black men were 128% more likely to be taking diuretics. The race differences among women were smaller – the odds ratio, although large in substantive terms (OR=1.35 in Model 1), was significantly different from zero only in the first model.

As with diuretics, we did not expect to see racial differences in the use of CCBs on the basis of the JNC recommendations. This prediction was borne out among men where no significant differences were found. However, Black women were more than twice as likely as

White women to take CCBs, and the odds ratio did not attenuate at all as we adjusted for a number of potential explanatory covariates.

Based on the JNC recommendations, we expected Black adults to be less likely to take β-blockers. The data supported this expectation: Black men and women were significantly less likely to be prescribed this class of medication than their white counterparts (OR=.65 for men and .61 for women in Model 1). Again, the race differences strengthened as more controls were added, suggesting that they were not due to differences between Black and White adults in socioeconomic status, comorbidities, or their blood biochemistry profile.

We also expected that Blacks would be less likely to take ACE inhibitors than Whites. However, the differences observed were not statistically significant. Interestingly, while the coefficients were in the expected direction of lower odds for Black men using an ACE inhibitor, models for women suggested the opposite pattern.

Finally, JNC recommendations did not mention race as a factor in prescribing drugs in the 'other' medication class. Black men and women, however, were significantly and substantially more likely to use a medication from this class, with ORs in the first Model of 1.73 for men and 2.09 for women. This class was the only series of nested models where the control variables, particularly the blood serum levels, explained the gross difference observed in Model 1. It should be noted, however, that the ORs remained substantively large even in the last model where they were not statistically significant.

Table 3 contains results from models identical to model 6 in the previous table, but presents the findings in detail to show the impact of all correlates on prescription patterns. Older adults were more likely to be prescribed all medications except for β-blockers. Patients who took multiple medications seemed most likely to get a diuretic and another class of medication.

There were regional differences. For example, women in rural areas were less likely to use β-blockers and women in the Northeast were least likely to use an ACE inhibitor than women in any other region. In contrast, no regional differences appeared for men. Education did not have an effect in any model while having insurance significantly increased the odds for a diuretic among men and for CCBs and 'other' class for women. These patterns are not intuitively clear given that diuretics are the least expensive class of antihypertensives and CCBs among the most expensive.

Comorbidities also reveal different patterns by sex. For example, ACE inhibitors, often prescribed for hypertensive patients with diabetes or heart failure, were significantly affected by diabetes among women and by heart failure among men. Diabetes also significantly increased the odds of women using CCBs but was not a significant factor for men. Only two of the blood serum markers revealed significant sex differences. Potassium levels significantly increased the odds of women using β-blockers but significantly reduced the odds of using 'other' medications. For men, potassium levels' only significant association was with ACE inhibitors, where it increased its odds. Finally, calcium levels significantly increased the odds for both men and women of using a diuretic and reduced their odds of using a CCB.

The models in Table 3 showed some unexpected differences between men and women. To assess whether differences observed hold for both races, we present race-stratified models in Table 4. The Table shows odds ratios of using a particular medication for women relative to men; each line shows a different antihypertensive medication category; each column a progressively larger set of control variables. Among both Whites and Blacks, women were substantially more likely to use diuretics than men. Adjusting for the full set of covariates, the odds ratio actually strengthened from 2.09 to 2.71 for White women and remained stable around

1.6 for Black women. White women were less likely to be prescribed CCBs than White men, but there was no statistically significant sex difference among Black adults for this medication class. Overall, White women differed more from White men in the odds of using a particular antihypertensive medication than Black women did from Black men.

DISCUSSION

The driving question behind this study was whether race affects treatment of hypertension. The data suggest that it does. The main insight is that the gross race differences in using each medication class was not explained by a host of likely explanatory variables. With the exception of the residual 'other' category, the confounding and mediating factors including socioeconomic status, comorbidities, and blood serum levels of key nutrients did not explain the race differences in medication types for men or women. An unexpected second insight is the considerable sex difference in how race affects the likelihood of being prescribed particular medication types.

The prescription pattern may have been influenced by multiple factors. The influential JNC recommendations from this time period suggested that Blacks responded less to β-blockers and ACE inhibitors, compared to Whites, and no race differences were described for diuretics and CCBs. The data, however, were not consistent with these recommendations. Where no race differences were predicted, we found that Black men were substantially more likely to use diuretics and Black women significantly more likely to use CCBs than their White counterparts. Where the recommendations predicted lower probability of taking ACE inhibitors for Blacks, the data showed no significant difference across race for this medication class. The only drug that was consistent with the JNC recommendations on race for both sexes was β-blockers, which were less likely to be taken by Black adults.

In addition to using race as a factor in selecting treatment for hypertension, the JNC also cites comorbidities, dietary assessment, and environmental factors (JNC V, 1993). In our analyses, we gradually adjusted for all these factors, from education and health insurance to comorbidities and blood levels of sodium, calcium, potassium, and vitamin D. However, these covariates did not significantly change the observed race patterns for most medication classes. Among the covariates, there were some that we expected to play a prominent role in the race differences. For instance, given the debate about the high sodium intake among Black adults and its connection to hypertension, we expected that controlling for sodium levels would explain a part of the race differential in the prescription patterns. The data did not support the expectation: the levels of sodium, as well as potassium and Vitamin D blood levels, suggested little about race and antihypertensive medication use. It is unclear what role the blood serum levels of these key nutrients plays in what drugs are being used for hypertension. Similarly, comorbidities including obesity, heart failure, and diabetes, which we expected to impact the gross race differentials, explained only a small proportion of the differences in the odds of using a specific antihypertensive drug.

Above we concluded that a range of potentially important covariates did not have a substantive effect on the race differences. This does not imply that the covariates did not influence the prescribing patterns. Some differences emerged in which covariates significantly predict prescription patterns for men and women. For example, having insurance was a positive predictor in the prescription of diuretics for men. This was not the case for any of the other drugs, which is odd given that diuretics are generally the least expensive of the antihypertensives, nor is it significant for women. Having insurance, however, was a significant predictor in the prescription of CCBs and the 'other' medication class for women. The data are

from 1988-1994 data, before Part D of Medicare, so this variable did not directly measure prescription drug coverage for a significant portion of our sample. Insurance status does serve as a rough marker of SES, as well as a signal to doctors about whether a patient is able to maintain her treatment, especially for a chronic condition like hypertension. Rural status and region were also significant predictors of specific prescription patterns for women but not for men. And the three comorbidities yielded different levels of influence on prescription patterns by sex as well.

The fact that the covariates did not change the race differences in the prescription patterns does not necessarily imply that they had the same effect on Blacks' and Whites' medication use either. However, we found (using race-stratified models not shown here) few systematic race differences in the factors that affect drug-prescription patterns. For instance, insurance status was a significant predictor of using CCBs or drugs in the 'other' category for Blacks. This makes sense given that CCBs and α-blockers at the time were relatively expensive treatment options and, in the case of the latter, generally prescribed in conjunction with another drug (Hilleman, et.al., 1994). However, insurance status had no effect on any of the drug classes for Whites. Insurance status increased the odds of Blacks using more expensive drugs, but not Whites. Why might this be so? Ubel, et.al. (2003) showed that pharmaceutical companies that produce more expensive medications are more likely to provide free samples to doctors, thus increasing their use. Given that Blacks, on average, have lower incomes than their White counterparts, doctors who primarily serve these communities may be more likely to receive these free samples, *and* more likely to dispense them as a means to keep their patients' health care costs down.

In conclusion, our analyses showed clear, systematic racial differences in the prescription of antihypertensive drugs. These differences persist after controlling for a host of variables typically associated with prescription patterns, from socioeconomic status to comorbidities. The

patterns and extent of these race differences vary by sex, a result we did not anticipate on the basis of existing literature.

The existing literature and JNC recommendations for pharmacological interventions for hypertension do not discuss race and sex interactions. Our findings suggest that race as well as sex plays a significant role in the treatment of hypertension. At the very least, they show that more women should be included in clinical trials and in epidemiological research regarding hypertension and its treatment. More generally, they call for an examination of the meaning of race and sex in medical practice and their effect on patient care.

While our models suggest mechanisms for explaining differences in the treatment of hypertension, for each medication, different patterns emerge across race and sex. In some cases, we find access to resources as predictive of patterns by race or sex (i.e. insurance coverage). In others, possible cultural and structural considerations come into play (i.e. regional and rural predictors for sex). And in some cases, race and sex themselves were the strongest predictors of using a specific drug, suggesting that doctors think of certain therapies as raced or gendered.

The analyses have several shortcomings that limit our ability to draw causal conclusions about how physicians use race and sex in prescribing medications. First, this study is based on cross-sectional data. Longitudinal data would allow for an analysis of treatment from the first diagnosis of hypertension, strengthening our ability to identify mechanisms determining treatment. Additionally, the demographic and socioeconomic variables are relatively crude, which may result in the underestimation of their effects. We did not include income due to the high percentage of missing data for this variable. Similarly, the use of census region as opposed to smaller geographic units may obscure the effects of location on prescription patterns – given the regional effects across race and sex for some drug classes we observed, this pattern should be

explored in more detail. More recent studies have incorporated practice-level data in their analyses of treatment patterns by race (e.g. Sequist, et.al, 2008), revealing significant differences in patient outcomes both across- practice, within-practice, and within-doctor. And studies examining the neighborhoods where Blacks and Whites live have shown that social and environmental exposures explain a substantial portion of the race gap in hypertension prevalence (Morenoff, et al., 2007; Thorpe, Brandon, & LaVeist, 2008). A nationally representative sample that allowed for this micro-level analysis would contribute to our ability to identify the mechanisms responsible for race and sex differences in the treatment of hypertension.

We would like to mention the significance of the timeframe in which the data were collected. We do not wish to suggest that the findings from these data, collected some 15 to 20 years ago, mirror the prescribing patterns of doctors today. However, the findings do illustrate significant racial differences in prescribing patterns at one point in time that may have serious implications for morbidity and mortality outcomes of individuals being treated for hypertension to date.

To conclude, this study showed that doctors treated hypertensive patients along racial lines that do not follow patterns suggested by the JNC and other literature of the time. Our findings have no bearing on the quality of care individuals receive, only that the type of pharmacological intervention differs. Information on physicians would greatly enhance our understanding of what role race plays in the doctor's office and why. Studies that can incorporate some or all of these refinements would provide not just an empirical contribution to the study of race, sex, and medicine, but also a theoretical one to the fields of medicine and clinical research in which both race and sex are under-theorized.

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Table 1. Distribution of select sample characteristics, White and Black adults age 25-75: Third National Health and Nutrition Examination Survey.

	White men	White women	Black men	Black women	Difference[1]
N	2,684	3,089	1,907	2,242	
Age -- mean (s.e.)	45.5 (.4)	46.3 (.5)	42.8 (.3)	43.4 (.5)	***
Education -- mean (s.e.)	13.0 (.1)	12.8 (.1)	11.6 (.1)	11.8 (.1)	***
South	32.7	31.6	54.5	52.4	***
Non-metro area	54.8	56.8	42.2	41.2	**
Not married	21.0	29.6	42.5	57.8	***
Health insurance[2]	91.1	92.1	84.8	87.2	***
Reported HBP[3]	24.0	24.5	27.8	34.8	***
Measured HBP[4]	19.9	14.3	25.8	22.6	***
Any antihypertensive med.	49.9	57.4	49.2	60.5	***
Number of antihypertensive medication[5]					***
0	50.1	42.6	50.8	39.5	
1	30.0	36.2	29.3	35.5	
2	14.4	17.1	13.3	18.4	
3+	5.5	4.2	6.6	6.5	
Specific antihypertensive medication					
Diuretics	14.2	27.3	19.7	29.2	***
Calcium blockers	17.8	14.2	19.8	23.5	**
Beta blockers	17.4	18.4	10.8	11.2	**
ACE inhibitors	16.8	14.2	14.0	15.1	n.s.
Other	9.7	9.1	13.2	14.2	**
Multiple antihypert.	19.9	21.2	19.9	24.9	n.s.
Comorbidities					
Diabetes	5.0	5.3	6.2	9.8	***
Heart failure	2.1	1.5	2.8	2.5	**
Obese	21.3	24.3	21.6	39.0	***
Plasma levels of -- mean (s.e.)					
Sodium (mmol/L)	141.3 (.1)	140.8 (.2)	141.3 (.2)	141.0 (.2)	**
Calcium (mmol/L)	2.3 (.0)	2.3 (.0)	2.3 (.0)	2.3 (.0)	n.s.
Potassium (mmol/L)	4.1 (.0)	4.0 (.0)	4.0 (.0)	3.9 (.0)	***
Vitamin D (mmol/L)	80.1 (1.7)	68.2 (1.1)	52.9 (1.6)	46.1 (1.2)	***

* p<0.1, ** p<0.05, *** p<0.01

Note: Shown are proportions unless specified otherwise. Adjusted for sampling design.

[1]Design-adjusted Wald tests and chi-square tests are used to assess difference among the four groups.

[2]Proportion with any health insurance, including Medicaid, Medicare, VA, or employer insurance.

[3]Proportion who reported that their doctor told them at least once that they had high blood pressure.

[4]Proportion with systolic pressure >140 mm/Hg or diastolic > 90mm/Hg measured during examination.

[5]Proportion on any hypertensive medication of those who self-reported hypertension.

Table 2. The effect of race on the odds of getting a given drug class for hypertension, OR (95% CI).

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Men						
Diuretics	1.73***	1.86***	1.86**	2.01***	2.01***	2.28***
	(1.26,2.38)	(1.25,2.76)	(1.17,2.96)	(1.21,3.31)	(1.23,3.30)	(1.29,4.03)
Calcium channel blockers	1.29	1.26	1.29	1.35	1.39	1.42
	(0.85,1.96)	(0.82,1.94)	(0.80,2.08)	(0.82,2.24)	(0.84,2.30)	(0.83,2.44)
Beta blockers	0.65*	0.55**	0.59**	0.60*	0.61*	0.45**
	(0.40,1.06)	(0.33,0.93)	(0.35,1.00)	(0.35,1.02)	(0.36,1.04)	(0.22,0.91)
Ace inhibitors	0.92	0.87	0.94	0.96	0.94	0.93
	(0.62,1.38)	(0.55,1.38)	(0.59,1.50)	(0.59,1.56)	(0.59,1.49)	(0.51,1.68)
Other	1.73***	1.82***	1.75**	1.54*	1.56*	1.36
	(1.17,2.57)	(1.19,2.77)	(1.13,2.70)	(0.96,2.45)	(0.99,2.45)	(0.78,2.34)
Women						
Diuretics	1.35*	1.15	1.21	1.23	1.21	1.15
	(0.98,1.87)	(0.79,1.68)	(0.82,1.78)	(0.84,1.80)	(0.82,1.79)	(0.72,1.81)
Calcium channel blockers	2.30***	2.24***	2.52***	2.59***	2.53***	2.37***
	(1.56,3.39)	(1.50,3.35)	(1.62,3.93)	(1.70,3.94)	(1.66,3.86)	(1.45,3.86)
Beta blockers	0.61***	0.51***	0.51***	0.50***	0.51***	0.55***
	(0.43,0.88)	(0.36,0.72)	(0.35,0.74)	(0.35,0.74)	(0.35,0.75)	(0.36,0.83)
Ace inhibitors	1.26	1.14	1.23	1.25	1.21	1.11
	(0.87,1.83)	(0.78,1.65)	(0.82,1.85)	(0.84,1.87)	(0.81,1.81)	(0.72,1.71)
Other	2.09***	1.96***	1.73**	1.68**	1.66**	1.57
	(1.45,3.01)	(1.31,2.92)	(1.11,2.69)	(1.05,2.68)	(1.03,2.68)	(0.89,2.76)

Each coefficient shows the effect of being black on the odds of taking the specific medication, estimated in a separate sex-stratified model.
Model 1 adjusts for age.
Model 2 adjusts for age and multiple antihypertensive medications.
Model 3 adjusts for above, plus demographics: region, non-metropolitan residence, and not married.
Model 4 adjusts for above, plus socioeconomic indicators: education and health insurance.
Model 5 adjusts for above, plus medical conditions: diabetes, heart failure, and obesity.
Model 6 adjusts for above, plus blood serum levels of K, Ca, Na, and vitamin D.

Table 3. The effect of race on the odds of getting a given drug class for hypertension, OR (95% CI).

	Women					Men				
	Diuretics	Calcium	Beta bl.	Ace	Other	Diuretics	Calcium	Beta bl.	Ace	Other
Black	1.15	2.37***	0.55***	1.11	1.57	2.28***	1.42	0.45**	0.93	1.36
	(0.72,1.82)	(1.46,3.86)	(0.36,0.83)	(0.72,1.71)	(0.89,2.77)	(1.30,4.01)	(0.84,2.43)	(0.22,0.91)	(0.51,1.68)	(0.78,2.34)
Age	1.05***	1.03***	1.01	1.02***	1.06***	1.04***	1.03***	1.02	1.03**	1.05***
	(1.03,1.06)	(1.01,1.04)	(1.00,1.03)	(1.01,1.04)	(1.04,1.09)	(1.02,1.06)	(1.01,1.05)	(0.99,1.04)	(1.00,1.05)	(1.03,1.08)
Multiple meds	16.61***	9.68***	5.97***	3.45***	5.11***	19.30***	6.66***	11.26***	5.61***	9.65***
	(10.84,25.45)	(6.17,15.17)	(4.21,8.46)	(2.39,4.98)	(2.95,8.87)	(9.92,37.54)	(3.95,11.23)	(5.91,21.44)	(3.61,8.73)	(6.12,15.21)
Rural	1.12	1.25	0.61**	0.98	1.15	1.07	1.22	0.84	0.99	0.79
	(0.74,1.70)	(0.79,1.98)	(0.40,0.92)	(0.59,1.63)	(0.63,2.07)	(0.52,2.21)	(0.67,2.22)	(0.48,1.49)	(0.53,1.83)	(0.47,1.34)
Midwest	0.95	0.94	0.89	1.66*	1.28	1.40	0.83	0.84	0.97	1.42
	(0.50,1.82)	(0.44,2.02)	(0.59,1.34)	(0.96,2.88)	(0.52,3.16)	(0.67,2.95)	(0.35,1.97)	(0.43,1.63)	(0.35,2.67)	(0.53,3.85)
South	0.73	0.68	0.96	1.58*	2.27**	0.87	1.40	0.65	1.07	1.52
	(0.39,1.35)	(0.38,1.20)	(0.61,1.50)	(0.93,2.68)	(1.11,4.65)	(0.37,2.01)	(0.56,3.47)	(0.33,1.30)	(0.42,2.73)	(0.63,3.67)
West	0.73	0.83	1.15	1.97*	0.97	0.71	1.10	0.68	0.72	0.85
	(0.37,1.45)	(0.36,1.95)	(0.64,2.06)	(0.96,4.05)	(0.30,3.11)	(0.30,1.68)	(0.48,2.54)	(0.33,1.44)	(0.28,1.88)	(0.26,2.79)
Not married	0.75	0.95	0.88	0.92	1.19	0.73	0.74	0.74	0.74	0.88
	(0.51,1.10)	(0.61,1.48)	(0.57,1.37)	(0.65,1.30)	(0.72,1.98)	(0.38,1.42)	(0.44,1.24)	(0.37,1.46)	(0.44,1.26)	(0.39,2.01)
Education	1.05	1.02	0.98	1.03	0.96	1.08	1.03	1.01	1.02	0.96
	(0.97,1.14)	(0.92,1.12)	(0.90,1.06)	(0.94,1.13)	(0.89,1.04)	(0.98,1.18)	(0.93,1.13)	(0.93,1.10)	(0.97,1.07)	(0.89,1.04)
Insurance	0.61	2.05*	1.70	0.68	2.82**	6.99**	0.93	1.52	0.97	2.27
	(0.33,1.13)	(0.89,4.75)	(0.78,3.70)	(0.33,1.38)	(1.03,7.71)	(1.20,40.75)	(0.35,2.47)	(0.53,4.35)	(0.34,2.79)	(0.37,14.04)
Diabetes	0.69	1.56*	0.98	1.77**	1.20	1.27	0.88	1.34	1.00	1.58
	(0.38,1.26)	(0.97,2.52)	(0.54,1.77)	(1.06,2.94)	(0.70,2.04)	(0.46,3.55)	(0.47,1.64)	(0.69,2.58)	(0.46,2.15)	(0.63,3.94)
Heart failure	2.21*	2.36**	0.30**	1.19	2.37*	1.37	1.06	0.34**	2.17**	3.10**
	(0.89,5.48)	(1.00,5.55)	(0.10,0.86)	(0.53,2.64)	(0.94,5.99)	(0.64,2.95)	(0.46,2.42)	(0.15,0.80)	(1.03,4.54)	(1.27,7.54)
Obese	1.63**	0.85	0.74	0.94	1.46*	1.49	1.33	1.36	0.91	1.38
	(1.07,2.47)	(0.58,1.27)	(0.42,1.31)	(0.61,1.45)	(0.95,2.26)	(0.85,2.61)	(0.83,2.12)	(0.81,2.29)	(0.50,1.66)	(0.80,2.39)
Potassium	0.10***	0.96	2.06**	1.45	0.41**	0.10***	1.15	1.18	2.03**	1.00
	(0.06,0.18)	(0.61,1.48)	(1.17,3.65)	(0.83,2.53)	(0.21,0.81)	(0.04,0.24)	(0.49,2.68)	(0.64,2.18)	(1.12,3.69)	(0.57,1.76)
Calcium	17.51***	0.08***	3.13	1.62	0.47	37.90***	0.79	5.04	1.67	1.42
	(3.96,77.44)	(0.02,0.36)	(0.32,30.38)	(0.34,7.73)	(0.09,2.49)	(4.28,335.3)	(0.07,8.94)	(0.26,98.70)	(0.32,8.64)	(0.16,12.51)
Sodium	0.95	1.01	0.96	0.97	0.99	1.01	0.97	0.99	0.94	0.99
	(0.88,1.02)	(0.93,1.09)	(0.90,1.03)	(0.91,1.04)	(0.91,1.07)	(0.92,1.11)	(0.88,1.07)	(0.89,1.09)	(0.85,1.03)	(0.89,1.10)
Vitamin D	1.00	1.00	1.00	1.00	1.01*	1.01	1.00	0.99	1.00	1.00
	(0.99,1.01)	(0.99,1.01)	(0.99,1.01)	(0.99,1.00)	(1.00,1.01)	(1.00,1.02)	(0.99,1.01)	(0.98,1.01)	(0.99,1.01)	(0.99,1.00)

* p<0.1, ** p<0.05, *** p<0.01

Note: These are full results from model 6 shown in table 2.

Table 4. The effect of sex on the odds of getting a given drug class for hypertension, OR (95% CI)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
White adults						
Diuretics	2.09***	2.86***	3.08***	3.19***	3.13***	2.71***
	(1.62,2.68)	(1.99,4.13)	(2.14,4.44)	(2.20,4.62)	(2.15,4.57)	(1.75,4.22)
Calcium channel block.	0.67*	0.64**	0.67*	0.68*	0.67*	0.70
	(0.44,1.01)	(0.42,0.98)	(0.43,1.05)	(0.43,1.07)	(0.43,1.07)	(0.44,1.13)
Beta blockers	1.00	1.03	1.09	1.09	1.06	1.00
	(0.74,1.37)	(0.75,1.41)	(0.79,1.52)	(0.78,1.51)	(0.76,1.50)	(0.71,1.43)
Ace inhibitors	0.75	0.75	0.76	0.78	0.78	0.81
	(0.52,1.09)	(0.50,1.13)	(0.50,1.15)	(0.51,1.19)	(0.51,1.21)	(0.51,1.28)
Other	0.79	0.80	0.79	0.76	0.77	0.67
	(0.53,1.18)	(0.51,1.26)	(0.49,1.26)	(0.47,1.24)	(0.48,1.24)	(0.39,1.16)
Black adults						
Diuretics	1.62***	1.67***	1.66***	1.67***	1.52**	1.60**
	(1.23,2.13)	(1.18,2.36)	(1.15,2.39)	(1.19,2.35)	(1.05,2.18)	(1.05,2.45)
Calcium channel block.	1.18	1.09	1.10	1.13	1.17	1.13
	(0.86,1.62)	(0.79,1.51)	(0.77,1.56)	(0.80,1.60)	(0.82,1.66)	(0.78,1.65)
Beta blockers	0.96	0.87	0.90	0.91	0.87	1.11
	(0.59,1.58)	(0.52,1.46)	(0.56,1.47)	(0.55,1.49)	(0.53,1.43)	(0.63,1.97)
Ace inhibitors	1.09	0.99	1.04	1.05	1.08	1.09
	(0.70,1.68)	(0.63,1.56)	(0.64,1.68)	(0.65,1.68)	(0.69,1.70)	(0.68,1.74)
Other	0.97	0.88	0.88	0.90	0.84	0.77
	(0.71,1.34)	(0.63,1.24)	(0.61,1.26)	(0.62,1.31)	(0.57,1.24)	(0.52,1.13)

Each coefficient shows the effect of being female on the odds of taking the specific medication, estimated in a separate race-stratified model.
The models 1-6 gradually adjust for the same variables are listed in the footnote below table 2.