

## Research Article

# Impact of Comorbidities on Racial/Ethnic Disparities in Hypertension in the United States

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**Background.** Racial/ethnic disparities in hypertension (HTN) prevalence continue to persist in United States. We aimed in this study to examine the racial/ethnic disparities in hypertension prevalence and to determine whether or not health disparities may be explained by racial/ethnic disparities in co-morbidities. **Materials and Methods.** A cross-sectional design was used to examine the prevalence of hypertension among African Americans (AAs), Caucasians, and Hispanics in the National Health Interview Survey, 2003. The overall sample comprised 30, 852 adults. **Results.** There was a statistically significant racial/ethnic variability in hypertension prevalence, with AA/Blacks with the highest prevalence,  $\chi^2 = 393.0$  (3),  $P < 0.01$ . Hypertension was associated with co-morbidities, age, education, physical inactivity, marital status, income, sex, alcohol, and cigarette consumption, but not insurance. Relative to Caucasians, AAA/Blacks were 43% more likely while Hispanics were 40% less likely to report being diagnosed with high blood pressure, prevalence odds ratio (POR) = 1.43, 99% CI, 1.25–1.64,  $P = 0.002$ , and POR = 0.60, 99% CI, 0.55–0.66,  $P < 0.001$ , respectively. After adjustment for the relevant covariates including co-morbidities, racial/ethnic disparities in hypertension persisted; thus compared to Caucasians, African Americans were 61% more likely to be told by their health care providers that they were hypertensive, adjusted prevalence odds ratio (APOR) = 1.61, 99% CI, 1.39–1.86,  $P < 0.001$ . In contrast, Hispanics were 27% less likely to be diagnosed with hypertension compared to Caucasians, APOR = 0.73, 99% CI, 0.68–0.79,  $P < 0.001$ . **Conclusions.** There was racial/ethnic variability in hypertension prevalence in this large sample of non-institutionalized US residents, with the highest prevalence of hypertension observed among African Americans. These disparities were not removed after controlling for relevant covariates including co-morbidities.

## 1. Introduction

Hypertension remains one of the leading causes of cardiovascular disease mortality in the United States population, affecting disproportionately non-Hispanic Blacks [1]. The etiology of hypertension is multifactorial, and incidence, prevalence, and mortality vary by race/ethnicity [2]. Variability by race/ethnicity in hypertension prevalence had been shown by several studies [3–5]. These studies continue to identify the African American (Blacks) ethnicity with the highest prevalence [4] and the ethnic group in which hypertension-related death is highest [5, 6].

Whereas evidence continues to demonstrate these health disparities, these variabilities have not been completely explained. Also, it is not fully understood whether the observed variance is due to race/ethnicity or other factors associated with race, since race/ethnicity remains surrogate on the pathway of the factors directly contributing to elevated blood pressure such as high sodium intake, hyperlipidemia, overweight and obesity, high fat diet, hypercholesterolemia, stress, cigarette smoking, excessive alcohol consumption, and physical inactivity [7–9]. Explanatory factors for racial/ethnic differences in hypertension include differences in the racial/ethnic distribution of family income,

access and utilization of the health care, and insurance status [9–13]. Other predictors of hypertension include socioeconomic, demographic, and prognostic factors. The persisted burden of hypertension among racial/ethnic minorities, namely, AA/Blacks, may be due to variability in the racial/ethnic distribution of comorbidities [14–19].

Lowering hypertension-related mortality in the USA may substantially narrow mortality disparities between Caucasians (non-Hispanic Whites) and African Americans (non-Hispanic Blacks). Such attempt requires further examination of the racial/ethnic variability in the distribution of the predisposing factors to hypertension including other cardiovascular and related diseases. We are not aware of any study at population level or other designs and settings that have examined the contributing role of comorbidity in the racial/ethnic disparities in the prevalence or incidence of hypertension in the US population. This present study was designed to examine the nexus between race/ethnicity and hypertension, using the National Interview Health Survey (NIHS) data, which is an annual survey of community-based US adult residents [20], and to determine whether or not the postulated variance could be explained by the racial/ethnic disparities in comorbidities, which may be associated with access as insurance status and utilization of the health care, income, education, and other socioeconomic parameters that define racial/ethnic groups in the USA. We hypothesized that the racial/ethnic disparities in hypertension may be explained by the racial/ethnic variance in comorbidities, implying that controlling for the known comorbidities will remove the variance.

## 2. Materials and Methods

After an IRB approval, we conducted a cross-sectional study to examine the prevalence of hypertension in the US population, the racial/ethnic variance in this prevalence, and to determine whether or not this variance may be explained by the differences in the racial/ethnic distribution of comorbidities.

**2.1. Design.** This research utilized secondary data from the National Health Interview Survey (NHIS) to examine the relationship between the outcome variable hypertension and main predictor variables race/ethnicity, comorbidities, family income, insurance status, and other sociodemographic and prognostic relevant variables age, sex, education level, employment status, marital status, body mass index (BMI), cigarette smoking, physical activity, and alcohol drinking status.

**2.2. Study Population.** Using the NHIS, 2003, we examined 30,852 adults. Participants were non-Hispanic Whites,  $n = 20,169$  (65.37%), non-Hispanic Blacks,  $n = 4,168$  (13.51%), Hispanics  $n = 5,416$  (17.55%), and Others,  $n = 1,099$  (3.56%). Participants were either male,  $n = 13,427$  (43.52%), or female,  $n = 17,425$  (56.48%), ages 18 years and older.

**2.2.1. Data Collection Procedures.** Data were collected via a personal household interview by Census interviewers. The details of the sampling are published elsewhere [20].

### 2.3. Study Variables

**2.3.1. Outcome Variable.** The study outcome variable (hypertension) was measured as a self-reported variable and was dichotomized as “yes” and “no.” Participants were asked if they were ever told by their health care provider that they had hypertension.

**2.3.2. Main Predictor (Explanatory) Variables: Race/Ethnicity and Comorbidities.** The main predictor variable for this study is race/ethnicity. The variable race was categorized into non-Hispanic Whites, non-Hispanic Blacks, Hispanics, and Others. The secondary predictor variable was comorbidities, which was measured by the number of diseases related to hypertension. We examined the distribution of diabetes mellitus, depression, other endocrine disorders, circulatory disorders, and hypercholesterolemia by race/ethnicity. These variables were treated individually and were entered into the model after examining their individual association with hypertension in the total sample.

**2.3.3. Other Potential Explanatory Variables.** Insurance coverage was measured by any family members having insurance coverage and was categorized into “yes,” “no,” “refused,” “not ascertained,” and “do not know.” The responses “refused,” “not ascertained,” and “do not know,” because of the small numbers, were not included in the analysis. Income status was measured by family income greater than \$20,000 and less than \$20,000. This variable was categorized into “greater than \$20,000,” “less than \$20,000,” “refused,” “not ascertained,” and “do not know.” The responses “refused,” “not ascertained,” and “do not know,” were not included in the analysis.

The sociodemographic variables are age, sex, education level, employment, and marital status. The age of participants was originally measured as a continuous variable and later categorized into a binary scale ( $\geq 50$  years versus  $< 50$  year), given the clinical relevance of age in the predisposition to hypertension. Both males and females were eligible for the survey provided the age requirement was satisfied. Education level was measured by the years of school attainment. This variable was collected as categorical and was further transformed into suitable categories, namely, less or equal to high school, some college, and greater than or equal to a bachelor's degree. Employment status was measured by a categorical variable that elicited information on job profile. This variable was recoded in order to examine unemployment versus employment, with respect to racial/ethnic distribution and the association with the outcome variables. Marital status was measured by a categorical variable and was used to examine the influence of social support system on the outcome variable hypertension.

**2.3.4. Prognostic Variables.** Body mass index (BMI) was conceptualized by relationship between height and weight. This

variable was collected on a continuous scale and was recoded into four distinct categories to reflect normal BMI and overweight BMI, utilizing the Centers for Disease Control and Prevention's cut-off points for BMI. Smoking was conceptualized as a historical variable. This variable was collected as categorical with the main variable eliciting information on "ever smoked" and "never smoked." The responses "refused," "not ascertained," and "do not know," were not included in the analysis. This variable was recoded into a binary variable. Physical activity was measured by the frequency of exercise. This variable was categorized into ten groups with major categories including "never exercise," "exercise," and "unable to exercise." We recoded physical activity into "ever exercise" (including daily, weekly, monthly, and yearly basis) versus "never exercise" (including unable to exercise), in order to examine the outcome variables and the association with race/ethnicity. Alcohol drinking status was collected as a categorical variable and was measured by the number of drinks within a period of time. This variable was recoded into "lifetime abstainer," "former drinker," and "current drinker."

**2.4. Statistical Analysis.** Categorical variables were described using frequency and percentages, while continuous variables were summarized using mean and standard deviation. Pearson chi-square statistic was used to examine racial/ethnic differences with respect to the distribution of the outcome variable hypertension, comorbidities, family income, insurance coverage, socioeconomic factors, and other prognostic factors. The association between the main predictor variable race and hypertension was assessed using unconditional univariable logistic regression model for the prevalence odds ratio [21]. Similar technique was used to examine the association between comorbidities and hypertension as well as the distribution of comorbidities by race/ethnicity. In addition, multivariable survey logistic regression model was used to simultaneously adjust for the effects of the potential confounding variables including comorbidities on the association between race/ethnicity and hypertension. To enter into the multivariable model, a variable was expected to be significant at  $P < 0.25$  or  $P < 0.10$  for an interaction. However, we found no interaction and as such did not include in our model. Further, variables with biological or clinical relevance were entered into the multivariable model. All analyses were two-tailed, with  $P < 0.01$  (1% type I error tolerance) as the significance level. The analyses were performed using STATA statistical software, version 11, (StataCorp, College Station, TX).

### 3. Results

Caucasians represented the majority of the participants, 20,169 (65.4%), Hispanics, 5,416 (17.5%), African Americans, 4,168 (13.5%), and others, 1,099 (3.6%), 12,832 (41.6%) were younger than 50 years of age, and 13,427 were males (43.5%). With respect to education, 15,149 (49.1%) had less than high school education, 8,691 (28.2%) had high school education, 4,614 (15%) had some college or college education, while 2,398 (7.8%) had graduate (postcollege)

education. Income less than \$20,000 per annum was reported by 10,010 (32.4%), while 20,842 (67.6%) reported annual income household of \$20,000 or higher. There were 15,373 (49.8%) who reported not being married, while 15,479 (50.2%) reported that they were married. The majority of participants reported having health insurance coverage, 27,517 (89.2%). More than half of the participants had no history of cigarette smoking, 17,637 (57.2%), while the remaining had used cigarette in the past, 13,215 (42.8%). Almost two-thirds of participants had no history of alcohol consumption (based on current use and ever use response), 23,115 (74.9%), while 7,737 (25.1%) had used alcohol. Likewise, almost two-thirds had not exercised in the past, 22,601 (73.3%), while others had 8,251 (26.7%). Of those sampled, 583 (2%) were underweight, 11,351 (38.9%) were normal weight, 10,418 (35.7%) were overweight, and 6,858 (23.5%) were obese.

Table 1 presents the sociodemographics, lifestyle variables, risk, and prognostic factors that may be associated with hypertension as study characteristics, stratified by race/ethnicity. The Caucasians were statistically significantly more likely to be older, while the Hispanics were the youngest. There was a statistically significant difference in the distribution of sex by racial/ethnic group in the sample,  $\chi^2 = 63.7$  (3),  $P < 0.001$ . Irrespective of race/ethnicity, there were more females in the sample, with the ratio of male to female sample greatest among the African Americans (37.9% for male versus 62.1% for female), 1:1.64 (African American women relative to male were 64% more likely to be reached for response in the household, but may also reflect survivability of the female over the male in this racial/ethnic group). The race/ethnicity designated others, which represent predominantly Asians who were more likely to have college and graduate degrees, followed by Caucasians, while the Hispanics had the lowest participants with either college or graduate degrees, and this observation was statistically significant.

There was a statistically significant difference in income by race/ethnicity. The Caucasians (72.5%) and other racial/ethnic groups (72.6%) relative to African Americans (55.1%) and Hispanics (57.7%) were more likely to be in the income group,  $> \$20,000.00$  annual income, while African Americans (44.9%) and Hispanics (42.3%) were more likely to be in the income group,  $< \$20,000.00$  per annum. Marital status was significantly different by race/ethnicity, and African Americans were less likely to be married, with the ratio of unmarried (69.5%) to married (30.5%) being 2.2:1 (implying that African Americans are two times as likely not to be married). There were no racial/ethnic differences in smoking status and insurance status, but alcohol, physical activities, and BMI demonstrated significant racial/ethnic variability.

Table 2 presents the prevalence of hypertension among the community-based United States residents, stratified by race/ethnicity. Though not shown in table, estimated one-third of adult United States population reported being told by their health care provider that they were hypertensive, 8,243 (26.7%). The prevalence of hypertension in this sample differed significantly by race/ethnicity,  $\chi^2 = 393.0$  (3),

TABLE 1: Characteristics of study participants by race/ethnicity, National Health Interview Survey.

Variable	Caucasians no. (%)	Hispanics no. (%)	African Americans no. (%)	Others no. (%)	$\chi^2$ (df)	P
Age (yrs)					927.7 (3)	<0.001
<50	7,244 (35.9)	3,125 (57.7)	1,878 (45.1)	585 (53.2)		
≥50	12,925 (64.1)	2,291 (42.3)	2,290 (54.9)	514 (46.8)		
Sex					63.7 (3)	<0.001
Male	8,955 (44.4)	2,392 (44.2)	1,578 (37.9)	502 (45.7)		
Female	11,214 (55.6)	3,024 (55.8)	2,590 (62.1)	597 (54.3)		
Education					1840 (6)	<0.001
<HS	8,672 (43.0)	3,852 (71.2)	2,257 (54.1)	368 (33.5)		
HS	6,090 (30.2)	1,046 (19.3)	1,278 (30.7)	277 (25.2)		
College	3,559 (17.6)	352 (6.5)	444 (10.7)	259 (23.6)		
Graduate	1,848 (9.2)	166 (3.1)	189 (4.5)	195 (17.4)		
Income (US\$)					772.3 (3)	<0.001
<20,000	5,547 (27.5)	2,290 (42.3)	1,872 (44.9)	301 (27.4)		
≥20,000	14,622 (72.5)	3,126 (57.7)	2,296 (55.1)	798 (72.6)		
Marital status					744.0 (3)	<0.001
Nonmarried	9,407 (46.6)	2,566 (47.4)	2,895 (69.5)	505 (46.0)		
Married	10,762 (53.4)	2,850 (52.6)	1,273 (30.5)	594 (54.0)		
Insurance coverage					4.40 (3)	0.22
No	2,164 (10.7)	617 (11.4)	451 (10.8)	103 (9.4)		
Yes	18,005 (89.3)	4,799 (88.6)	3,717 (89.2)	996 (90.6)		
Smoking					673.9 (3)	<0.001
No	10,503 (52.1)	3,786 (69.9)	2,596 (62.3)	752 (68.4)		
Yes	9,666 (47.9)	1,630 (30.1)	1,572 (37.7)	347 (31.6)		
Alcohol					957.6 (3)	<0.001
No	16,211 (80.4)	3,465 (64.0)	2,795 (67.1)	644 (58.6)		
Yes	3,958 (19.6)	1,951 (36.0)	1,373 (32.9)	455 (41.4)		
Exercise					182.0 (3)	<0.001
No	14,353 (71.2)	4,329 (79.9)	3,143 (75.4)	776 (70.6)		
Yes	5,816 (28.8)	1,087 (20.1)	1,025 (24.6)	323 (29.4)		
BMI					575.3 (6)	<0.001
<18.5	421 (2.2)	62 (1.2)	47 (1.2)	53 (5.1)		
18.5–24.9	7,812 (40.8)	1,782 (35.0)	1,169 (29.9)	588 (56.0)		
25.0–29.9	6,776 (35.4)	1,969 (38.7)	1,384 (35.4)	289 (27.5)		
>30	4,152 (21.7)	1,280 (25.1)	1,307 (33.4)	119 (11.3)		

Abbreviations and notes: No.: numbers, BMI: body mass index and was calculated given the height (meters) and weight (Kg) of participants. HS: high school. The significance level is <0.01. "Others" which represents predominantly Asians were more likely to have college and graduate degree, followed by Caucasians, while the Hispanics had the lowest participants with either college or graduate degrees, BMI (Kg/m<sup>2</sup>): body mass index was calculated given the height (meters) and weight (Kg) of participants. The first BMI group (<18.5 Kg/m<sup>2</sup>) is underweight, 18.5–24.9 is normal, and 25–29.9 is overweight, while >30 is obese. df: degrees of freedom.

TABLE 2: Hypertension prevalence in a sample of community-based United States residents, National Health Interview Survey.

Race/ethnicity	Hypertensive		Nonhypertensive		$\chi^2$ (393.0)	df (3)	P (<0.001)
	Number	%	Number	%			
Caucasians	5,552	27.5	14,617	72.5	—	—	—
Hispanics	1,009	18.6	4,407	81.4	—	—	—
African Americans	1,481	35.5	2,687	64.5	—	—	—
Others	201	18.3	898	81.7	—	—	—

Crude and unadjusted prevalence (percentage) of hypertension. df: degrees of freedom.  $\chi^2$ : chi square.



TABLE 3: The prevalence of hypertension in a sample of United States community-based residents by race/ethnicity with Caucasian as the reference race/ethnicity.

Race/ethnicity	Prevalence odds ratio*	99% Confidence Interval	P
Caucasians	1.00	Referent	Referent
Hispanics	0.60	0.55–0.66	<0.001
African Americans	1.43	1.25–1.64	0.002
Others	0.57	0.50–0.66	<0.001

The race/ethnicity “others” is predominantly Asian Americans. The significance level is 0.01 (1% type 1 tolerable error). \*Crude and unadjusted prevalence odds of having been told that an adult is hypertensive using survey logistic regression model.

$P < 0.001$ . The prevalence of hypertension was highest among African Americans (35.5%), intermediate among Caucasians (27.5%), and lowest among Hispanics (18.6%) and others (18.3%).

Table 3 presents the unadjusted or crude prevalence of hypertension by race and ethnicity using Caucasian as the reference race or group. Compared with Caucasians, African Americans were 43% more likely to report being diagnosed with high blood pressure, prevalence odds ratio (POR) = 1.43, 99% CI, 1.25–1.64,  $P = 0.002$ . Hispanics, relative to Caucasians, were 40% less likely to report having been told by their health care providers that they were hypertensive, POR = 0.60, 99% CI, 0.55–0.66,  $P < 0.001$ .

Table 4 presents the factors associated with hypertension prevalence in community-based United States residents in a univariable survey logistic regression model. The older age group relative to the younger group was six times as likely to be hypertensive, and this association was statistically significant, POR = 6.33, 95% CI, 5.77–6.94. There was no significant association between sex and hypertension.

There was a significant association between education, income, marital status, alcohol consumption, smoking, and physical activities and the prevalence of hypertension,  $P < 0.01$ . Though not shown on the table, Hispanics (59.9%) and African Americans (68.1%) compared to Caucasians (72.6%) in our sample were less likely to check their cholesterol level,  $P < 0.001$ . Thus, compared to Caucasians, Hispanics were 44% less likely to check their cholesterol level, while African Americans were 19% less likely as well, OR = 0.56, 99% CI, 0.53–0.60,  $P < 0.001$ , and OR = 0.81, 99% CI, 0.75–0.87,  $P < 0.001$ , respectively. The persistency of high cholesterol prevalence as a result of absence of awareness of such a risk factor in individuals who are not checking their cholesterol level reflects increased predisposition to hypertension and hence elevated prevalence of hypertension in the populations at risk. Second, compared to Caucasians (31.2%), Hispanics (23.9%) and African Americans (24.4%) had lower prevalence of high cholesterol level,  $P < 0.001$ . African Americans and Hispanics were 28% and 30% less likely to have high cholesterol level compared to Caucasians, OR = 0.72, 99% CI, 0.66–0.79,  $P < 0.001$  and OR = 0.70, 99% CI, 0.65–0.77,  $P < 0.001$ , respectively.

TABLE 4: Factors associated with hypertension prevalence in a sample of community-based United States residents, National Health Interview Survey, 2003.

Covariate	Prevalence odds ratio	99% Confidence Interval	P
Age (years)			
<50	1.00	Referent	Referent
≥50	6.33	5.77–6.94	<0.001
Sex			
Male	1.00	Referent	Referent
Female	1.16	1.01–1.34	0.04* (NS)
Education			
<High school	1.00	Referent	Referent
High school	0.70	0.66–0.74	<0.001
College	0.51	0.48–0.55	<0.001
Graduate degree	0.72	0.68–0.76	<0.001
Income (US\$)			
<20,000.00	1.00	Referent	Referent
≥20,000.00	0.67	0.64–0.70	<0.001
Insurance coverage			
No	1.00	Referent	Referent
Yes	1.02	0.84–1.23	0.82* (NS)
Marital status			
No	1.00	Referent	Referent
Yes	0.86	0.82–0.91	0.001
Alcohol			
No	1.00	Referent	Referent
Yes	1.26	1.19–1.33	<0.001
Smoking			
No	1.00	Referent	Referent
Yes	1.28	1.17–1.40	0.002
Physical activity			
No	1.00	Referent	Referent
Yes	0.86	0.82–0.91	0.001

Univariable survey logistic regression model, with 0.01 as the significance level.

**3.1. Comorbidities.** African Americans (10.8%) had the highest prevalence of diabetes mellitus (DM), where hypertension is more prevalent compared to Caucasians (7.8%) and Hispanics (7.5%). In our data, diabetes mellitus was associated with hypertension, with those who had diabetes mellitus 6 times as likely to have hypertension compared to those without, OR = 6.34, 99% CI, 5.83–6.94. Also, compared to Caucasians (40.8%), African Americans (55.5%) and Hispanics (58.6%) were more likely to be diagnosed with diabetes mellitus at younger age (<50 years),  $P < 0.001$ . Though not a significant finding in our sample, African Americans (87.3%) and Hispanics (85.0%) compared to Caucasians (88.9%) were less likely to check their blood sugar level,  $P = 0.08$ . In addition, among African Americans, diabetes mellitus appeared to be poorly controlled, with more of the

TABLE 5: Multivariable survey logistic regression of the association between race/ethnicity in the prevalence of hypertension among community-based United States residents, National Health Interview Survey.

Race/ethnicity	Adjusted prevalence odds*	99% Confidence Interval	P
Caucasians	1.00	Referent	Referent
Hispanics	0.73	0.68–0.79	<0.001
African Americans	1.61	1.39–1.86	0.001
Others	0.74	0.63–0.87	<0.001

The race/ethnicity “others” is predominantly Asian Americans. The significance level is 0.01 (1% type 1 tolerable error). \*Adjusted prevalence odds of having been told that an adult is hypertensive using survey logistic regression model. Adjusted factors were age, education, marital status, smoking, alcohol, income, exercise, cholesterol level, and comorbidities (diabetes mellitus and depression).

African Americans taking insulin relative to diabetic pill (36.1% and 66.4%), compared to Caucasians (27.6% and 68.0%) and Hispanics (24.2% and 74.7%). Also, compared with African Americans without diabetes mellitus, those with diabetes mellitus were almost 13 times as likely to have hypertension, OR = 12.73, 99% CI, 7.82–20.70,  $P < 0.001$ , but among Caucasians and Hispanics without diabetes mellitus, those with diabetes mellitus were 7 times as likely to have hypertension, OR = 7.20, 99% CI, 5.83–8.92, and OR = 7.10, 99% CI, 5.64–8.92, respectively.

Chronic circulatory problem may predispose to hypertension and other cardiovascular conditions as a result of blood vessel occlusion and subsequent increase in peripheral resistance. Compared with Caucasians (89.3%) in our sample, African Americans (96.7%) and Hispanics (97.0%) were more likely to have chronic circulatory problem,  $P = 0.70$ .

Whereas there was no significant difference in the racial/ethnic prevalence of depression, Caucasians (7.9%) had the lowest prevalence of depression, anxiety, and emotional problems, compared with African Americans (8.3%) and Hispanics (8.8%),  $P = 0.41$ . The prevalence of this condition may be higher among the minorities especially African Americans, but due to the stigma associated with it, it is always underreported as reflected on its overall prevalence in a survey of this nature, self-reported emotional problems (8.1%). There was a marginally statistically significant difference in the racial/ethnic prevalence of those who can afford mental care/counseling for this condition,  $P = 0.03$ .

**3.2. Effect of Comorbidity on Racial/Ethnic Variability in Hypertension.** Table 5 presents the adjusted or controlled association between hypertension and race/ethnicity in a multivariable survey logistic regression model. After adjustment for the factors that were associated with hypertension (age, education, marital status, smoking, alcohol, income, comorbidities, and exercise/physical activities) in our univariable model, and those associated with race/ethnicity in our chi-square for independence test, the significant racial/ethnic disparities in hypertension prevalence persisted

in this model (multivariable survey logistic regression). Compared to Caucasians, African Americans were 61% more likely to be told by their health care providers that they were hypertensive, adjusted prevalence odds ratio (APOR) = 1.61, 99% CI, 1.39–1.86,  $P < 0.001$ . In contrast, Hispanics were 27% less likely to be diagnosed with hypertension compared to Caucasians, APOR = 0.73, 99% CI, 0.68–0.79,  $P < 0.001$ .

## 4. Discussion

Because hypertension is a single most potent predictor of cardiovascular-related mortality, and racial/ethnic variability in its incidence and prevalence had been observed in the US population, we aimed in this study to validate this variability and to determine whether or not racial/ethnic differences in comorbidities can remove such disparities. There are some relevant findings in this study. First, there were racial/ethnic disparities in the prevalence of hypertension. Secondly, hypertension was most prevalent among African Americans. Thirdly, some chronic diseases (selective comorbidities) were associated with high prevalence of hypertension. Fourthly, comorbidities varied by race/ethnicity, and African Americans had the highest prevalence of metabolic diseases mainly diabetes mellitus. Finally, after adjustment for the confounding factors including comorbidities in the association between hypertension and race/ethnicity, the racial/ethnic disparities in hypertension prevalence persisted.

This large sample of noninstitutionalized US residents demonstrated health disparities in hypertension prevalence. While this finding is not novel, it is worth noticing that racial/ethnic disparities in hypertension prevalence continue to persist in the USA, and that racial/ethnic minorities bear the burden of hypertension [3–5]. This finding supports previous literature in similar populations and samples in the USA [3–5, 22–25]. The issue remains as to whether or not there is racial/ethnic predisposition to hypertension, which renders it impossible to remove the observed disparities. African Americans/Blacks are more predisposed to increased volume loading and decreased volume depletion, which affirms genetic predisposition or gene-environment interaction in HTN. Consequently, since hypertension runs in families, preventing HTN in the first place among the high risk populations such as AA/Blacks remains a feasible approach to racial/ethnic hypertension prevalence narrowing in the USA.

We have also demonstrated that some conditions or comorbidities were associated with hypertension in our sample. For example, individuals with DM were more likely to be hypertensive regardless of race/ethnicity. However the predisposition to HTN given DM was stronger among AA/Blacks compared to their white counterparts. HTN and DM tend to coexist, and it expected that uncontrolled DM may lead to microvascular compromise including arterial stenosis and hence elevated blood pressure. It is also plausible to expect DM to result from uncontrolled HTN since this could result in end organ damage involving the renal system and liver [26]. DM as comorbidity was shown in our data to vary by race/ethnicity, with AA/Blacks with the highest

prevalence of DM. Therefore, given the nexus between DM and HTN, the highest prevalence of HTN among AA/Blacks may be explained in part by the higher prevalence of DM among AA/Blacks. Controlled glycemia or blood glucose level may be implicated in the HTN prevalence, since studies have shown poor glycemic control among AA/Blacks compared to whites [27–30].

The process of adjusting for confounding is to balance this factor between racial/ethnic group while observing the effect of the variable of interest (HTN prevalence) on race/ethnicity (independent variable). Applying this technique we controlled for comorbidities as well as other factors known to be associated with HTN and race/ethnicity in an attempt to determine whether or not the observed racial/ethnic disparities observed in the crude association could be removed. After this adjustment, the racial/ethnic disparities though removed to some extent did persist.

We reexamined our research question as to whether or not comorbidities explain the racial/ethnic disparities in HTN in the USA. The data indicate that higher HTN prevalence among AA/black cannot be explained by racial/ethnic variability in comorbidities among racial/ethnic groups. Race/ethnicity portrays biologic as well as environmental, social, and psychosocial attributes. While it may be a direct measure of outcomes in some diseases such as hypertension where physiologic variance in volume loading and volume depletion had been observed [31], it remains a surrogate in many outcomes of interest such as COPD, asthma, and bronchitis. Remarkably, comorbidities were higher among AA/Blacks, and after adjustment for these conditions the racial/ethnic disparities in HTN persisted.

Despite the strength of this study, there are some limitations. First, we attempted to explain racial/ethnic variance in HTN prevalence in a large sample of US noninstitutionalized residents by observing variability in comorbidities among racial/ethnic groups. Since we used the cross-sectional data temporality remains a big issue. Specifically, it is unclear if DM was diagnosed before HTN, or depression, circulatory disorders, and so forth diagnosed before HTN. Secondly, because we used preexisting data for this secondary analysis, there are variables known to influence HTN such as dietary profile which were not available in our data for possible assessment as a confounder or effect measure modifier. The availability of such data could have provided some explanation to the observed racial/ethnic disparities in HTN prevalence. Thirdly, we adjusted for factors associated with race/ethnicity and hypertension. However, like in most epidemiologic studies, we cannot rule out the possibility of unmeasured or residual confounding in these findings, since no matter how sophisticated a statistical model used is, residual confounding remains [32]. But it is not likely that our findings are driven solely by these unmeasured or residual confounding. *Finally, hypertension was self-reported, which might have introduced a misclassification, selection, and information biases into HTN prevalence by race/ethnicity. But assuming nondifferential misclassification bias, it is unlikely that the evidence in this study is substantially influenced by such biases.*

In summary racial/ethnic disparities exist in HTN prevalence which were not completely removed after controlling for comorbidities. These findings are suggestive of the need to examine other risk and predisposing factors in HTN incidence and prevalence, as well as comorbidities in an attempt to understand racial/ethnic disparities hypertension in the US.

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