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Hypertension in the San Antonio Heart Study and the Mexico City Diabetes Study: Clinical and Metabolic Correlates

SYNOPSIS

DESPITE THE GREATER OBESITY AND PREVALENCE of non-insulin-dependent diabetes mellitus (NIDDM) in Mexican Americans (MA) than in non-Hispanic whites (NHW), MA have a similar or slightly lower prevalence and incidence of hypertension than NHW. After adjustment for age, gender, obesity, and NIDDM, the prevalence of hypertension was significantly lower in MA than in NHW in both men and women. Mexican Americans, however, have lower rates of control than do non-Hispanic whites. The high rates of NIDDM, coupled with the poor control of hypertension in Mexican Americans, make efforts to control hypertension essential in this group. The prevalence of hypertension in low income residents of Mexico City is lower than in low income Mexican Americans from San Antonio, Texas.

Compared to non-Hispanic whites (NHW), Mexican Americans (MA) are generally more obese *(1,2)* and have an unfavorable body fat distribution *(2)*, as well as higher prevalence *(1)* and incidence *(3)* of non-insulin-dependent diabetes mellitus (NIDDM)—all of which are well known risk factors for hypertension. Despite these excess risk factors, Mexican Americans have a similar or lower prevalence of hypertension than non-Hispanic whites *(4,5)*. In this paper we review data from previously published papers on hypertension prevalence *(5)*, incidence *(6)*, and level of control of blood pressure *(7)* in Mexican Americans and non-Hispanic whites in the San Antonio Heart Study (SAHS). We will also review a recent comparative study of hypertension prevalence and level of control in low-income participants in the Mexico City Diabetes Study *(8)*.

Methods

The San Antonio Heart Study *(5,9)*. The SAHS is a population-based study of diabetes and cardiovascular disease in Mexican-American and non-Hispanic white men and women ages 25 to 64. Detailed descriptions of the study design, sampling procedures, response rates, and field procedures have appeared elsewhere *(1,5)*. Recruitment of participants in the SAHS occurred in two phases: the first encompassed the period from October 1979

to November 1982; and the second period, from October 1984 to October 1988. Both phases consisted of independent random samples conducted in different groups of census tracts. The data from both phases are pooled in the analyses presented in this paper.

We defined ethnicity using a previously published algorithm that considered parental surnames and birthplaces, stated ethnicity of grandparents, and the participant's preferred ethnic identity when a distinct national origin was indicated. Those identified as belonging to an ethnic group other than Mexican American or non-Hispanic white were excluded from the present analyses. Body mass index (BMI) was weight in kilograms divided by height in meters squared.

Blood pressure measurement protocols included determining systolic blood pressure (SBP) and diastolic blood pressure (DBP) by the first and fifth Korotkoff sounds, using a random zero sphygmomanometer to the nearest even digit on the right arm of the seated participant after at least a 5-minute rest period, taking three readings for each participant, and defining blood pressure as the average of the second and third readings.

We used four definitions of hypertension: 1) diastolic hypertension—DBP ≥95 mmHg or current use of antihypertensive medications; 2) Hypertension, Detection and Follow-up Program (HDFP definition)—SBP ≥160 mmHg or current use of antihypertensive medications; 3) combined hypertension—SBP ≥160 mmHg or DBP ≥95 mmHg or current use of antihypertensive medications; and 4) mild hypertension—SBP ≥140 mmHg or DBP ≥90 mmHg or current use of antihypertensive medications (1988 Joint National Committee definition). We included under each definition treated participants whose blood pressures were *controlled* (that is, below the stated cutpoint).

A 12-hour fast preceded the taking of blood specimens for plasma glucose and serum insulin, lipids and lipoprotein determinations. Following the fasting specimen, trained technicians administered a 75-gram glucose equivalent load and obtained blood samples 1 and 2 hours later for post-glucose load plasma glucose determinations. Diabetes was diagnosed according to National Diabetes Data Group criteria. In October 1987, an 8-year follow-up study was begun to determine the incidence of NIDDM and cardiovascular disease *(6)*.

Mexico City Diabetes Study. The Mexico City Diabetes Study is a population-based study of cardiovascular disease in both diabetic and non-diabetic men and women ages 35 to 64. Detailed descriptions have been previously published *(8)*. Methods similar to those used in the San Antonio Heart Study were employed. We restricted analyses of the San Antonio Mexican Americans in this section to participants ages 35 to 64 from low income areas (barrio) to increase comparability with the Mexico City populations. We defined mild hypertension as SBP ≥140 mmHg and (or) DBP ≥90 mmHg, DBP ≥90 mmHg, or current use of antihypertensive medications, which corresponds to the mild hypertension category of *The Fifth Report of the Joint National Committee on Detection, Evaluation, and Treatment of High Blood Pressure* (JNC-V) *(10)*.

Results

The San Antonio Heart Study. In Table 1 we present the crude prevalence of hypertension according to each of the four definitions defined above. Using the first

Table 1. Prevalence of hypertension and clinical characteristics of Mexican Americans and non-Hispanic whites

	Men		Women		P-Values for Ethnicity	
	Mexican Americans n=1391	Non-Hispanic Whites n=833	Mexican Americans n=1906	Non-Hispanic Whites n=1040	Men	Women
Hypertension						
DBP ≥95 or Meds	139	102	181	128	0.097	0.017
	(10.0%)	(12.2%)	(9.5%)	(12.3%)		
SBP ≥160 or Meds	125	102	196	132	0.014	0.067
	(9.0%)	(12.2%)	(10.3%)	(12.7%)		
SBP ≥160 or DBP ≥95 or Meds	152	109	206	132	0.124	0.125
	(10.9%)	(13.1%)	(10.8%)	(12.7%)		
Mild HBP						
SBP ≥140 or DBP ≥90 or Meds	247	146	278	165	0.897	0.353
	(17.8%)	(17.5%)	(14.6%)	(15.9%)		
Hypertensives on Meds (n)	110	94	169	124	—	—
Age (yrs)	42.9±0.3	44.7±0.4	42.9±0.3	44.8±0.4	<0.001	<0.001
BMI (kg/m^2)	28.0±0.1	26.7±0.1	28.4±0.1	25.5±0.2	<0.001	<0.001
Prevalence of NIDDM	156	45	240	51	<0.001	<0.001
	(11.2%)	(5.4%)	(12.6%)	(4.9%)		

Source: Adapted from ref 5.

three definitions, hypertension prevalence was lower in Mexican Americans than in non-Hispanic whites in both sexes, although this difference was statistically significant only in two of the six comparisons. There was little difference in the prevalence of mild hypertension between the two ethnic groups. Compared with non-Hispanic whites, Mexican Americans were younger, more obese, and had a higher prevalence of NIDDM, suggesting that confounding could have obscured a larger difference in hypertension prevalence between the two ethnic groups. Among hypertensive men (definition 1), Mexican Americans were less likely to be on antihypertensive therapy than were non-Hispanic whites (110 of 139 or 79.1% of Mexican Americans and 94 of 102 or 92.2% of non-Hispanic whites, P <0.01). Among hypertensive women ethnic differences did not affect the percent in treatment (169 of 181 or 93.4% of Mexican Americans and 124 of 128 or 90.9% of non-Hispanic whites were in treatment).

We also calculated a multiple logistic regression with DBP (definition 1) as the dependent variable, and ethnicity, age, BMI, and NIDDM as independent variables. The estimated hypertension prevalence was significantly lower in Mexican Americans than in non-Hispanic whites in both men (OR (MA/NHW)=0.72, 95% CI=0.54, 0.96 (P=0.028)) and in women (OR=0.62, 95% CI=0.47, 0.81, P=0.001).

Additional analysis *(9)* showed a significant but modest positive association of insulin concentrations with hypertension prevalence. These associations were similar in both ethnic groups. After adjusting for insulin concentrations, hypertension prevalence remained significantly lower in MA than in NHW.

The incidence of hypertension was 8.8% or 32 of 364 Mexican-American men and 10.2% or 26 of 254 non-Hispanic white men (P=0.592). Among women the incidence of hypertension was 9.3% or 47 of 503 Mexican Americans and 8.5% or 29 of 341 non-Hispanic whites (P=0.602). We also calculated a multiple logistic regression with hypertension incidence as the dependent variable and ethnicity, age, BMI, NIDDM, and educational status as independent variables. Estimated incidence rates of hypertension were slightly but not significantly lower in Mexican-American men (OR=0.72, 95% CI=0.38, 1.33) and women (OR=0.88, 95% CI=0.48, 1.58).

We also examined the correlations between obesity, glucose tolerance, and fasting insulin and the development of hypertension *(11)*. All three of these metabolic factors predicted the incidence of hypertension. In addition, the slope of the relation between fasting insulin and the incidence of hypertension was similar in Mexican Americans and non-Hispanic whites. The association between insulin and the incidence of hypertension, however, was stronger in lean than in obese participants. Prehypertensive participants had increased triglyceride and decreased HDL cholesterol, suggesting that hypertensive participants may have increased cardiovascular risk (particularly due to dyslipidemia) even before the onset of clinical hypertension.

In Table 2 we show the mean levels of anthropometric and metabolic variables and blood pressure according to ethnic group and gender in hypertensive participants. Hypertensive Mexican Americans were younger and had

Table 2. Mean levels and standard errors of anthropometric and metabolic variables and blood pressure in hypertensive participants according to ethnicity and gender

	Mexican American		*Non-Hispanic White*		*P-Values*		
	Men n=152	*Women n=206*	*Men n=109*	*Women n=132*	*Ethnic*	*Sex*	*Ethnic x Sex*
Age (yrs)	50±1	53±1	53±1	55±1	0.002	0.002	0.595
BMI (kg/m^2)	29.9±0.5	31.9±0.5	28.2±0.4	29.2±0.6	<0.001	0.076	0.054
Centrality[a]	1.72±0.05	1.19±0.02	1.60±0.06	1.05±0.03	<0.001	<0.001	0.730
Fasting Glucose (mg/dl)	108.9±3.5	116.4±4.0	98.8±1.9	100.9±2.7	<0.001	0.178	0.452
2-Hr Glucose (mg/dl)	167.5±7.9	189.7±7.8	134.3±5.3	143.9±6.8	<0.001	0.040	0.416
Fasting Insulin (microunits/ml)	17.6±1.1	16.9±1.0	12.7±1.1	12.3±1.0	<0.001	0.589	0.947
2-Hr Insulin (microunits/ml)[b]	84.4±1.2	111.9±1.0	73.0±1.4	72.1±1.0	0.008	0.221	0.180
SBP (mmHg)	136±2	135±1	130±2	128±2	<0.001	0.284	0.588
DBP (mmHg)	84±1	80±1	79±1	75±1	<0.001	<0.001	0.653
% In Poor Control[c]	46/152 (30.3%)	42/206 (20.4%)	15/109 (13.8%)	11/132 (8.3%)	<0.001	0.012	—
% On Antihypertensive Medicine	110/152 (72.4%)	169/206 (82.0%)	94/109 (86.2%)	124/132 (93.9%)	0.014	0.688	—
% SBP ≥160 mmHg	17/152 (11.2%)	29/206 (14.1%)	6/109 (5.5%)	9/132 (6.8%)	0.009	0.325	—
% DBP ≥95 mmHg	34/152 (22.4%)	17/206 (8.3%)	9/109 (8.3%)	3/132 (2.3%)	<0.001	<0.001	—

[a] Centrality=ratio of subscapular-to-triceps skinfolds.
[b] San Antonio Heart Study only phase II.
[c] SBP ≥160 mmHg, DBP ≥95 mmHg, or both.
Source: Adapted from ref 7.

greater adiposity (body mass index), higher subscapular-to-triceps skinfold ratios (centrality), higher fasting and 2-hour plasma glucose and fasting serum insulin concentrations, and higher systolic and diastolic blood pressure levels than hypertensive non-Hispanic whites. Women, however, had higher 2-hour glucose concentrations than men. Mexican-American hypertensive participants were significantly more likely to have poorly controlled hypertension, to have SBP >160 mmHg or DBP >95 mmHg, and less likely to be on antihypertensive therapy than non-Hispanic white hypertensive participants.

We also calculated a multiple logistic regression model in which the dependent variable was poor versus good control of hypertension in hypertensive participants, and the independent variables were ethnicity, age, gender, BMI, centrality, fasting glucose, and education. Only Mexican-American ethnicity (OR (MA/NHW)=0.36, 95% CI=0.21, 0.62, P<0.001) was significantly associated with poor control. When diabetic participants were excluded, the results were nearly identical.

Mexico City Diabetes Study. Low-socioeconomic-status Mexicans have a significantly lower prevalence of both mild and moderate hypertension (JNC-V definitions) than barrio Mexican Americans. In men ages 35 to 64, the prevalence of mild hypertension is 17.1% in Mexico and 24.4% in San Antonio (P=0.001), and in women ages 35 to 64 it is 17.4% in Mexico and 22.0% in San Antonio (P=0.005). The SBP is significantly higher in San Antonio participants than in Mexico City participants, but there is no significant difference in DBP or age between the participants of the two cities. We computed multiple logistic regression analyses with mild hypertension as the dependent variable and age, BMI, WHR, city, NIDDM, genetic admixture and educational attainment. The city odds ratio (Mexico City to San Antonio) for the prevalence of mild hypertension was 0.55 (95% CI=0.39, 0.77, P <0.001) in men, 0.81 (95% CI=0.59, 1.42, P=0.201) in women, and 0.67 (95% CI=0.53, 0.84, P=0.001) in both sexes pooled.

Discussion

We have shown in this review that Mexican Americans have a modest deficit (about 15%) in crude prevalence of hypertension relative to non-Hispanic whites in San Antonio. After adjustment for the greater obesity and NIDDM present in Mexican Americans, the deficit in hypertension becomes somewhat larger (about 30%) and statistically significant. Mexican Americans also have a modest deficit in hypertension incidence, although these results are not statistically significant perhaps because of decreased statistical power. The cause of the "relative" protection from hypertension is currently unknown and needs further study. Because Mexican Americans have a high prevalence *(1)* and incidence *(3)* of NIDDM and because hypertension is an important risk factor for diabetic complications, hypertension has a major public health impact in this ethnic group even if it is somewhat less common than in non-Hispanic whites. This observation is highlighted by our work showing that Mexican-American hypertensive participants have more poorly controlled hypertension than non-Hispanic white hypertensive participants.

We have recently shown that the prevalence of hypertension among barrio residents in Mexico City is lower than in barrio Mexican Americans in San Antonio. Although Mexicans are leaner and have less NIDDM than Mexican Americans, these confounding variables do not explain the differences in hypertension prevalence. It is possible that other environmental factors such as salt or calcium intake or other dietary habits may explain this interesting difference.

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