Cardiovascular Disease Among Hispanics and Non-Hispanics in the Chronic Renal Insufficiency Cohort (CRIC) Study

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Summary

Background and objectives Hispanics are the largest minority group in the United States. The leading cause of death in patients with chronic kidney disease (CKD) is cardiovascular disease (CVD), yet little is known about its prevalence among Hispanics with CKD.

Design, setting, participants, & measurements We conducted cross-sectional analyses of prevalent self-reported clinical and subclinical measures of CVD among 497 Hispanics, 1638 non-Hispanic Caucasians, and 1650 non-Hispanic African Americans, aged 21 to 74 years, with mild-to-moderate CKD at enrollment in the Chronic Renal Insufficiency Cohort (CRIC) and Hispanic CRIC (HCRIC) studies. Measures of subclinical CVD included left ventricular hypertrophy (LVH), coronary artery calcification (CAC), and ankle-brachial index.

Results Self-reported coronary heart disease (CHD) was lower in Hispanics compared with non-Hispanic Caucasians (18% *versus* 23%, $P = 0.02$). Compared with non-Hispanic Caucasians, Hispanics had a lower prevalence of CAC >100 (41% *versus* 34%, $P = 0.03$) and CAC >400 (26% *versus* 19%, $P = 0.02$). However, after adjusting for sociodemographic factors, these differences were no longer significant. In adjusted analyses, Hispanics had a higher odds of LVH compared with non-Hispanic Caucasians (odds ratio 1.97, 95% confidence interval, 1.22 to 3.17, $P = 0.005$), and a higher odds of CAC >400 compared with non-Hispanic African Americans (odds ratio, 2.49, 95% confidence interval, 1.11 to 5.58, $P = 0.03$). Hispanic ethnicity was not independently associated with any other CVD measures.

Conclusions Prevalent LVH was more common among Hispanics than non-Hispanic Caucasians, and elevated CAC score was more common among Hispanics than non-Hispanic African Americans. Understanding reasons for these racial/ethnic differences and their association with long-term clinical outcomes is needed.

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Introduction

Hispanics are the largest and fastest growing minority group in the United States (1). Both the incidence and prevalence rates of end-stage renal disease (ESRD) in Hispanics are 1.5 times higher than in non-Hispanics (2). Although it is well established that chronic kidney disease (CKD) is an independent risk factor for cardiovascular disease (CVD) and all-cause mortality (3–5), limited data exist regarding the association of CVD and CKD in Hispanics. This is an area of interest because, in the general population, Hispanics appear to have a survival advantage compared with non-Hispanic Caucasians, despite a higher prevalence of cardiovascular risk factors (6). This observation is referred to as the "Hispanic paradox" and has been inconsistently found in CKD patients. In a large group of individuals with stage 3 or 4 CKD enrolled in Kaiser Permanente of Northern California, Hispanic ethnicity was associated with a lower risk of cardiovascular events and death relative to non-Hispanic Caucasians despite experiencing a higher risk of ESRD (7). In contrast, a study of a nationally representative sample of the U.S. population, the third National Health and Nutrition Examination Survey (NHANES III), demonstrated a nonsignificant trend toward higher mortality in Mexican Americans with CKD than non-Hispanic Caucasians (8). These heterogeneous findings may be due to differences in the populations studied and methodology but underscore the need for further investigation.

We characterize the prevalence of self-reported clinical CVD and measures of subclinical CVD among

Hispanics compared with non-Hispanic Caucasians and African Americans enrolled in two prospective observational studies of CKD: the Chronic Renal Insufficiency Cohort (CRIC) and Hispanic CRIC (HCRIC) Studies.

Materials and Methods

Study Design and Participants

We conducted cross-sectional analyses among Hispanic and non-Hispanic participants at enrollment in the CRIC and HCRIC studies. The design, methods, and baseline characteristics of the CRIC study participants have been previously published (9,10). Briefly, the CRIC study is a prospective cohort of 3612 individuals (169 Hispanics and 3443 non-Hispanics), aged 21 to 74 years, with mild-to-moderate CKD, recruited between May 2003 and March 2007 from seven clinical centers across the United States. HCRIC is a parallel study to the CRIC study that recruited 327 Hispanics from the Chicago area from October 2005 through June 2008, using the same inclusion criteria. Among all Hispanic participants, 69% were Mexican American, 16% were Puerto Rican, and 15% had other Latin American ancestry. We decided to combine all Hispanic participants in our analyses because no significant differences were found in the outcomes of interest between Hispanic subgroups. Protocols for both studies were approved by the Institutional Review Board of the participating centers and are in accordance with the principles of the Declaration of Helsinki. Informed consent was obtained from all participants.

Variables and Measurements

Sociodemographic characteristics, and medications were self-reported at entry. Hypertension was defined as systolic BP ≥140 mmHg, diastolic BP ≥90 mmHg, or use of antihypertensive medications. Diabetes was defined as fasting glucose ≥126 mg/dl, random glucose ≥200 mg/dl, or use of insulin or antidiabetic medication. Dyslipidemia was defined as fasting total cholesterol ≥240 mg/dl, low-density lipoprotein (LDL) ≥160 mg/dl, high-density lipoprotein (HDL) ≥40 mg/dl, triglycerides ≥200 mg/dl, or use of lipid-lowering medication. Family history of premature coronary heart disease (CHD) was defined as CHD in male first-degree relative <55 years, or CHD in female first-degree relative <65 years. History of CVD, including heart failure, peripheral arterial disease (PAD), stroke or CHD (defined as history of myocardial infarction [MI] or prior revascularization), was self-reported on a medical history questionnaire in the participant's language of preference (English or Spanish). Subclinical CVD measures included left ventricular hypertrophy (LVH), coronary artery calcification score (CAC), and ankle-brachial index (ABI). LVH was defined as left ventricular mass index ($g/m^{2.7}$) by transthoracic echocardiography (TTE) >50 in males and >47 in females. CAC was measured in a subcohort of the CRIC study using electron-beam computed tomography (EBCT) or multi-detector computer tomography. The CRIC subcohort was selected as a stratified, weighted random sample of approximately one third of all participants, based on the anticipated distribution within the full cohort. Subcohort participants were assigned to undergo additional, more intensive testing (including iothalamate clearance studies to measure GFR and EBCT). We considered two cut off points for CAC (>100 and >400) as consistent with subclinical CVD. TTE and CAC measurements were performed at the 1-year follow-up visit in the CRIC study. In HCRIC, all participants underwent TTE and CAC measurement at entry. GFR was estimated using the four-variable Modification of Diet in Renal Disease (MDRD) equation (11). Previous MI on electrocardiogram (ECG) was defined as the presence of Major 2 or 3 Minnesota code criteria (12).

Statistical Analysis

Descriptive statistics were summarized as mean (SD) for continuous variables and frequency (proportion) for categorical variables. If the data distributions were skewed, natural logarithmic transformations were conducted and/or data were presented as median (interquartile intervals). Crude and adjusted (for age, gender, education, and estimated GFR [eGFR]) means for CVD risk factors were compared between racial/ethnic groups using ANOVA and *t* tests, as appropriate. Prevalence of self-reported clinical and subclinical measures of CVD was compared using chi-squared tests. To determine factors independently associated with clinical and subclinical measures of CVD, we fitted multivariate hierarchical logistic regression models that included *(1)* crude (unadjusted) measures, *(2)* demographic measures, and *(3)* clinical measures (see Table 4). These variables were chosen based on bivariate analyses and prior studies (5,13–18). The only variables with >3% missing values were proteinuria/24 h (6%), income (15%), and health insurance (12%); the latter two were not included in the regression models. To compare Hispanics with non-Hispanic groups, each model was run twice, once with non-Hispanic Caucasians as reference and once with non-Hispanic African Americans as reference. All hypothesis tests were two-sided with alpha level = 0.05. Among 3939 subjects enrolled in CRIC/HCRIC, 154 self-identified as member of "other" race/ethnicity were excluded from the analyses. We adopted a complete case analysis approach for all analyses (19); therefore, the final analytic cohorts varied among the different analyses presented: *(1)* 353 participants were excluded from the multivariate analysis of self-reported CVD due to missing data for one of the covariates, *(2)* 1857 participants were excluded from CAC analyses because they did not undergo CAC measurement as specified by study design (9), *(3)* 911 participants were excluded from LVH analyses due to missing data for the outcome (689) or covariates (222), and *(4)* 398 participants were excluded from ABI analyses because of missing data for this outcome (61) or covariates (337). All statistical analyses were conducted using SAS version 9.1 (SAS Institute, Cary, NC).

Results

Participant Characteristics and Unadjusted Prevalence of Cardiovascular Risk Factors

Among 3785 CRIC/HCRIC participants in our final analytic cohort (497 Hispanics [H]), 1638 non-Hispanic Caucasians [C], 1650 non-Hispanic African Americans [A]), 93% met criteria for CKD (eGFR <60 ml/min/1.73 m^2 or urine albumin-to-creatinine ratio >30 mg/g) (20). Compared with non-Hispanic Caucasians and African Americans, Hispanics were significantly younger (mean age 56 years [H], 59 years [C], 58 years [A]; $P < 0.01$; Table 1), more likely to have annual income <$20,000 (63% [H], 16%

[C], 39% [A]; $P < 0.001$), <7th grade education (37% [H], 0.4% [C], 1.2% [A]; $P < 0.001$), and no health insurance (23% [H]), 3% [C]), 6% [A]; $P < 0.001$). Only 53% of Hispanics reported ever being seen by a nephrologist, compared with 69% of non-Hispanics Caucasians and 67% of non-Hispanic African Americans ($P < 0.001$). Hispanics were also more likely to have lower mean eGFR in ml/min/1.73 m^2 (37.5 [H], 43.7 [C], 43.5 [A]; $P < 0.001$), and higher median proteinuria in g/24 h (0.7 [H], 0.1 [C], 0.2 [A]; $P < 0.001$). The following cardiovascular risk factors were significantly more common in Hispanics than non-Hispanic Caucasians and African Americans: diabetes

Table 1. Baseline characteristics of participants

Demographic Characteristics	Overall (n = 3785)	Hispanic (n = 497)	Non-Hispanic Caucasian (n = 1638)	P_1	Non-Hispanic African American (n = 1650)	P_2
Age, years	58.2 (11)	56.3 (12)	58.9 (11)	<0.01	58.1 (11)	<0.01
Gender, n (%)						<0.01
Male	2076 (55)	288 (58)	982 (60)	0.43	806 (49)	
Female	1709 (45)	209 (42)	656 (40)		844 (51)	
Annual household income, n (%)				<0.01		<0.01
<$20,000	1213 (32)	313 (63)	254 (16)		646 (39)	
$20,001–$50,000	925 (24)	92 (19)	416 (25)		417 (25)	
$50,001–$100,000	694 (18)	24 (5)	455 (28)		215 (13)	
>$100,000	369 (10)	12 (2)	295 (18)		62 (4)	
Missing	584 (15)	56 (11)	218 (13)		310 (9)	
Education, n (%)				<0.01		<0.01
<7th grade	210 (6)	183 (37)	7 (0.4)		20 (1.2)	
7th to 12th grade	610 (16)	110 (22)	83 (5)		417 (25)	
High school diploma	728 (19)	71 (14)	291 (18)		366 (22)	
Some college	1112 (29)	78 (16)	467 (29)		567 (34)	
College graduate	1125 (30)	55 (11)	790 (48)		280 (17)	
Health insurance, n (%)				<0.01		<0.01
Yes	3085 (82)	322 (65)	1442 (88)		1321 (80)	
No	256 (7)	113 (23)	48 (3)		95 (6)	
Missing	444 (12)	62 (12)	148 (9)		234 (14)	
Ever seen a nephrologist	2503 (66)	265 (53)	1131 (69)	<0.01	1107 (67)	<0.01
Kidney function measures						
Serum creatinine, mg/dl	1.75 (0.6)	1.88 (0.6)	1.59 (0.5)	<0.01	1.9 (0.6)	0.77
eGFR, ml/min/1.73 m^2	42.8 (13)	37.5 (13)	43.7 (13)	<0.01	43.5 (14)	<0.01
eGFR categories, ml/min/1.73 m^2, n (%)						
<30	734 (19)	151 (30)	278 (17)	<0.01	305 (18)	<0.01
30 to <40	939 (25)	162 (33)	389 (24)		388 (24)	
40 to <50	999 (26)	107 (22)	462 (28)		430 (26)	
50 to <60	722 (19)	51 (10)	349 (21)		322 (20)	
>60	391 (10)	26 (5)	160 (10)		205 (12)	
24-hour urine protein, g, median (IQR)	0.18 (0.1–0.9)	0.72 (0.1–3.3)	0.12 (0.1–0.5)	<0.01	0.24 (0.1–1.1)	<0.01
Traditional cardiovascular risk factors						
Current cigarette smoker, n (%)	504 (13)	29 (6)	155 (9)	0.01	320 (19)	<0.01
Hypertension, n (%)	3269 (86)	443 (89)	1293 (79)	<0.01	1533 (93)	0.01
Systolic BP, mmHg	129 (22)	136 (24)	122 (19)	<0.01	133 (23)	0.01[a]
Diastolic BP, mmHg	72 (13)	73 (13)	69 (11)	<0.01	74 (14)	0.07
Diabetes mellitus, n (%)	1831 (48)	334 (67)	649 (40)	<0.01	848 (51)	<0.01
Hemoglobin A1C	6.7 (2)	7 (2)	6.3 (1)	<0.01	6.9 (2)	0.29
BMI, kg/m^2	32 (8)	32 (7)	31 (8)	0.20	33 (8)	<0.01
Total cholesterol, mg/dl	184 (45)	189 (54)	180 (42)	<0.01[a]	186 (46)	0.14
LDL cholesterol, mg/dl	103 (36)	104 (40)	99 (32)	0.03[a]	106 (37)	0.24
HDL cholesterol, mg/dl	48 (15)	43 (13)	47 (15)	<0.01	49 (16)	<0.01
Family history of premature CHD, n (%)	603 (16)	50 (10)	322 (20)	<0.01	231 (14)	0.02
Nontraditional cardiovascular risk factors						
Hemoglobin, g/dl	12.6 (2)	12.1 (2)	13.2 (2)	<0.01	12.2 (2)	0.19
Serum calcium corrected for albumin,[b] mg/dl	9.2 (0.5)	9.2 (0.4)	9.2 (0.4)	0.56	9.3 (0.5)	0.01
Serum phosphorus, mg/dl	3.7 (0.7)	4 (0.7)	3.6 (0.6)	<0.01	3.8 (0.7)	<0.01
Total iPTH, pg/ml, median (IQR)	54 (35–90)	62 (41–102)	43 (30–69)	<0.01[a]	67 (41–115)	0.09
hs-CRP, mg/dl, Median (IQR)	2.6 (1.1–6.6)	2.5 (1.0–5.7)	2.15 (0.9–5.2)	0.26	3.3 (1.3–8.2)	<0.01

Values indicate mean (standard deviation) unless otherwise specified. eGFR, estimated glomerular filtration rate; hs-CRF, high-sensitivity C-reactive protein; IQR, interquartile range; P_1, Hispanic *versus* Non-Hispanic Caucasian; P_2, Hispanic *versus* Non-Hispanic African American.

[a]No longer significant after adjustment for age, gender, education, eGFR.

[b]Corrected calcium = Measured serum calcium + [0.8*(4–serum albumin)].

(67% [H], 40% [C], 51% [A]; $P < 0.001$); mean HDL in mg/dl (43 [H], 47 [C], 49 [A]; $P < 0.001$); and mean serum phosphate in mg/dl (4.0 [H], 3.6 [C], 3.8 [A]; $P < 0.001$). Compared with non-Hispanic Caucasians, Hispanics were more likely to have higher systolic BP in mmHG (136 [H] *versus* 122 [C], $P < 0.001$); diastolic BP in mmHG (73 [H] *versus* 69 [C], $P < 0.001$); hemoglobin A1C (7% [H] *versus* 6.3% [C], $P < 0.001$); and lower mean hemoglobin in g/dl (12.1 [H] *versus* 13.2 [C], $P < 0.001$). Differences in these cardiovascular risk factors were statistically significant even after adjustment for age, gender, education, and eGFR (data not shown). In contrast, only 6% of Hispanics reported current cigarette smoking, compared with 9% of non-Hispanic Caucasians and 19% of non-Hispanic African Americans ($P = 0.01$).

Prevalence of Clinical CVD

Compared with non-Hispanic Caucasians, Hispanics had lower prevalence of self-reported CHD (18% [H] *versus* 23% [C], $P = 0.02$; Table 2). The prevalence of any self-reported CVD, heart failure, and PAD was similar between Hispanics and non-Hispanic Caucasians. Compared with non-Hispanic African Americans, Hispanics had a significantly lower prevalence of any self-reported CVD (28% [H] *versus* 38% [A], $P < 0.001$), heart failure (7% [H] *versus* 13% [A], $P < 0.001$), and stroke (7% [H] *versus* 14% [A], $P < 0.001$). Self-reported CHD and PAD prevalence were similar between Hispanics and non-Hispanic African Americans (Table 2).

Prevalence of Subclinical Measures of CVD

Compared with non-Hispanic Caucasians, Hispanics had lower prevalence of CAC >100 (34% [H] *versus* 41% [C], $P = 0.03$); higher prevalence of LVH (71% [H] *versus* 40% [C], $P < 0.001$); and similar prevalence of abnormal ABI (15% [H] *versus* 13% [C], $P = 0.2$; Table 2). Compared with non-Hispanic African Americans, Hispanics had a significantly lower prevalence of ABI <0.9 (15% [H] *versus* 20% [A], $P = 0.007$), higher prevalence of LVH (71% [H] *versus* 63% [A], $P = 0.003$), and similar prevalence of CAC (>100). Using an alternative cutoff for CAC (>400), similar patterns between Hispanics and non-Hispanics were observed (Table 2).

Stratified Analyses of Clinical and Subclinical Outcomes

Among participants with eGFR <30 ml/min/1.73 m^2, Hispanics had significantly lower prevalence of self-reported CVD, CAC >100, and CAC >400, as compared with non-Hispanic Caucasians ($P < 0.05$; Table 3). Differences in high CAC prevalence was less pronounced when compared with non-Hispanic African Americans. Prevalent LVH was significantly higher in Hispanics than non-Hispanic Caucasians within every strata of abnormal eGFR ($P < 0.0001$; Table 3). Thirty-two percent of Hispanics with eGFR <30 ml/min/1.73 m^2 had CAC >100, compared with 53% of non-Hispanic Caucasians ($P = 0.003$) and 46% of non-Hispanic African Americans ($P = 0.05$; Table 3; Figure 1a).

In stratified analysis by gender, the prevalence of self-reported CHD was significantly lower among Hispanic males (19%) compared with non-Hispanic Caucasian males (30%, $P < 0.001$). Among participants with diabetes, the prevalence of CHD was also lower in Hispanics (21%) compared with non-Hispanic Caucasians (33%, $P < 0.001$; data not shown). ECG evidence of previous MI was similar across racial/ethnic groups (8% [H], 7% [C], 9% [A]; $P > 0.05$). Forty percent of Hispanics with diabetes had CAC >100, compared with 64% of non-Hispanic Caucasians ($P < 0.0001$; Figure 1b). This difference was not observed in participants without diabetes (data not shown).

Multivariate Analysis of Clinical and Subclinical Outcomes

In fully adjusted logistic regression models, the prevalence odds of LVH were two times higher among Hispanics compared with non-Hispanic Caucasians (95% confidence interval 1.2 to 3.2, $P = 0.005$) (Table 4). The fully adjusted prevalence odds of CAC >400 were 2.5 times higher among Hispanics compared with non-Hispanic African Americans (95% confidence interval 1.1 to 5.6, $P =$

Table 2. Prevalence of cardiovascular disease (CVD)*

Variables	Overall ($n = 3785$)	Hispanic ($n = 497$)	Non-Hispanic Caucasian ($n = 1638$)	P_1	Non-Hispanic African American ($n = 1650$)	P_2
Self-reported history of CVD						
Any CVD	1272 (34)	138 (28)	507 (31)	0.18	627 (38)	<0.01
CHD (MI or coronary revascularization)	827 (22)	90 (18)	376 (23)	0.02	361 (22)	0.07
Heart failure	371 (10)	37 (7)	117 (7)	0.82	217 (13)	<0.01
Stroke	382 (10)	37 (7)	118 (7)	0.86	227 (14)	<0.01
Peripheral arterial disease	257 (7)	35 (7)	105 (6)	0.62	117 (7)	0.97
Subclinical Measures of CVD						
CAC score >100	719 (37)	115 (34)	359 (41)	0.03	245 (35)	0.88
CAC score >400	415 (22)	65 (19)	225 (26)	0.02	125 (18)	0.52
Left ventricular hypertrophy	1663 (54)	282 (71)	538 (40)	<0.01	843 (63)	<0.01
Ankle-brachial index <0.9	611 (16)	72 (15)	206 (13)	0.23	333 (20)	<0.01

*Values indicate *n* (%) unless otherwise specified. CAC, coronary artery calcium score; IQR, interquartile range; P_1, Hispanic *versus* non-Hispanic Caucasian; P_2, Hispanic *versus* non-Hispanic African American.

Table 3. Clinical and subclinical measures of cardiovascular disease by estimated glomerular filtration rate (eGFR) in ml/min/1.73 m²*

eGFR, ml/min/1.73m²	Hispanic	Non-Hispanic Caucasian	P_1	Non-Hispanic African American	P_2
Self-reported Cardiovascular Disease, *n* (%)					
<30	42 (28)	115 (41)	<0.01	135 (44)	<0.01
30 to <40	52 (32)	143 (37)	0.30	174 (45)	<0.01
40 to <50	30 (28)	154 (33)	0.29	165 (38)	0.05
50 to <60	10 (20)	75 (21)	0.76	106 (33)	0.06
≥60	4 (15)	20 (13)	0.68	47 (23)	0.38
Coronary artery calcium score >100, *n* (%)					
<30	32 (32)	61 (53)	<0.01	47 (46)	0.05
30 to <40	43 (38)	91 (50)	0.05	43 (34)	0.44
40 to <50	26 (34)	99 (41)	0.25	69 (35)	0.80
50 to <60	11 (33)	83 (35)	0.81	59 (36)	0.75
≥60	3 (18)	25 (23)	0.60	27 (22)	0.67
Coronary artery calcium score >400, *n* (%)					
<30	18 (18)	37 (32)	0.02	28 (27)	0.12
30 to <40	24 (21)	61 (34)	0.03	27 (21)	0.95
40 to <50	14 (18)	61 (25)	0.20	31 (16)	0.65
50 to <60	7 (21)	52 (22)	0.90	27 (17)	0.52
≥60	2 (12)	14 (13)	0.88	12 (10)	0.80
Left ventricular hypertrophy, *n* (%)					
<30	104 (83)	121 (54)	<0.01	182 (76)	0.14
30 to <40	90 (70)	139 (45)	<0.01	223 (69)	0.72
40 to <50	57 (63)	151 (40)	<0.01	219 (63)	0.92
50 to <60	24 (65)	93 (31)	<0.01	141 (54)	0.20
≥60	7 (37)	34 (25)	0.26	78 (45)	0.48
Ankle-brachial index <0.9, *n* (%)					
<30	35 (24)	53 (20)	0.30	86 (28)	0.31
30 to <40	23 (15)	68 (18)	0.37	84 (22)	0.05
40 to <50	5 (5)	51 (11)	0.05	88 (21)	<0.01
50 to <60	7 (14)	32 (9)	0.26	62 (19)	0.39
≥60	2 (8)	2 (1)	0.04	13 (7)	0.82

*Values indicate *n* (%). P_1, Hispanic *versus* non-Hispanic Caucasian; P_2, Hispanic *versus* non-Hispanic African American.

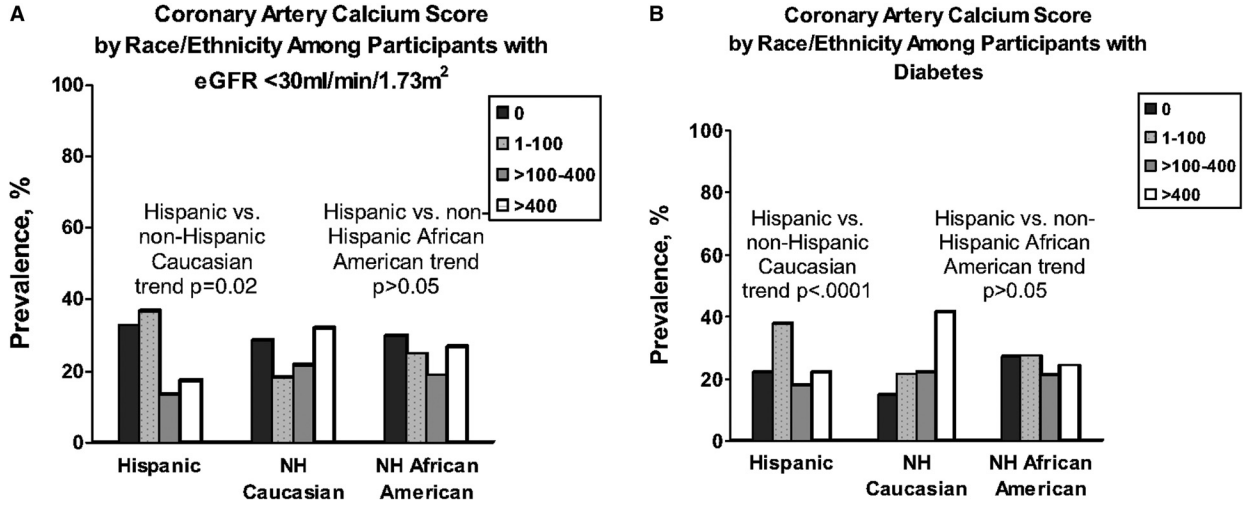

Figure 1. | Prevalence of coronary artery calcium scores (0, 1 to 100, >100 to 400, >400) by race/ethnicity among participants with eGFR <30ml/min/1.73m² (1a) and diabetes (1b). NH, non-Hispanic.

0.03). Adjusted prevalence odds of self-reported clinical CVD, CAC >100, and ABI <0.9 were similar across racial/ethnic groups.

Achievement of Therapeutic Goals for CVD Prevention

Several differences across racial/ethnic groups were found with respect to the unadjusted prevalence of

Table 4. Crude and adjusted odds ratio (OR) of clinical and subclinical measures of cardiovascular disease (CVD)

Variables	Any self-reported CVD		CAC >100		CAC >400		LVH		ABI <0.9	
	OR (95% CI)	*P* value	OR (95% CI)	*P* value	OR (95% CI)	*P* value	OR (95% CI)	*P* value	OR (95% CI)	*P* value
Unadjusted										
Hispanic *versus* NH Caucasian	0.86 (0.69, 1.07)	0.18	0.75 (0.58, 0.97)	0.03	0.69 (0.51, 0.94)	0.02	3.64 (2.86, 4.63)	<0.01	1.19 (0.89, 1.60)	0.23
Hispanic *versus* NH African American	0.63 (0.50, 0.78)	<0.01	0.98 (0.74, 1.29)	0.88	1.11 (0.80, 1.55)	0.52	1.44 (1.13, 1.83)	<0.01	0.68 (0.52, 0.90)	<0.01
Adjusted for demographic characteristics[a]										
Hispanic *versus* NH Caucasian	1.10 (0.75, 1.62)	0.63	0.89 (0.47, 1.69)	0.73	1.12 (0.56, 2.24)	0.74	2.16 (1.42, 3.27)	<0.01	1.26 (0.79, 2.03)	0.33
Hispanic *versus* NH African American	0.88 (0.61, 1.28)	0.51	1.45 (0.77, 2.74)	0.25	2.34 (1.17, 4.68)	0.02	1.08 (0.71, 1.62)	0.72	0.88 (0.56, 1.39)	0.60
Adjusted for demographic and clinical characteristics[b]										
Hispanic *versus* NH Caucasian	1.03 (0.68, 1.55)	0.90	0.63 (0.30, 1.33)	0.23	0.86 (0.39, 1.93)	0.72	1.97 (1.22, 3.17)	<0.01	1.29 (0.78, 2.13)	0.32
Hispanic *versus* NH African American	0.82 (0.55, 1.23)	0.34	1.35 (0.64, 2.82)	0.43	2.49 (1.11, 5.58)	0.03	1.44 (0.90, 2.31)	0.13	1.05 (0.65, 1.70)	0.84
Age	1.06 (1.05, 1.07)	<0.01	1.10 (1.08, 1.11)	<0.01	1.09 (1.07, 1.11)	<0.01	1.03 (1.02, 1.04)	<0.01	1.05 (1.04, 1.07)	<0.01
Gender (female *versus* male)	0.66 (0.54, 0.79)	<0.01	0.32 (0.24, 0.42)	<0.01	0.36 (0.26, 0.50)	<0.01	0.86 (0.70, 1.06)	0.15	1.03 (0.81, 1.29)	0.83
Education										
<7th grade *versus* college graduate	1.20 (0.75, 1.91)	0.45	0.72 (0.37, 1.40)	0.30	0.83 (0.39, 1.73)	0.61	2.50 (1.37, 4.53)	<0.01	1.12 (0.63, 1.99)	0.70
7th to 12th grade *versus* college graduate	1.23 (0.94, 1.60)	0.13	1.18 (0.75, 1.84)	0.50	1.47 (0.90, 2.40)	0.13	1.70 (1.24, 2.33)	<0.01	1.49 (1.07, 2.08)	0.02
HS diploma *versus* college graduate	1.29 (1.01, 1.64)	0.04	1.37 (0.94, 1.98)	0.10	1.58 (1.05, 2.38)	0.03	1.44 (1.10, 1.88)	<0.01	1.54 (1.13, 2.09)	<0.01
Some college *versus* college graduate	1.38 (1.12, 1.71)	<0.01	1.18 (0.86, 1.62)	0.30	1.05 (0.73, 1.50)	0.80	1.35 (1.07, 1.70)	0.01	1.34 (1.00, 1.78)	0.05
eGFR (10 ml/min/1.73 m^2 increase)	0.90 (0.84, 0.97)	<0.01	0.89 (0.79, 1.00)	0.05	0.95 (0.82, 1.09)	0.45	0.86 (0.79, 0.93)	0.02	0.78 (0.71, 0.86)	<0.01
Proteinuria (natural log)	1.08 (1.01, 1.15)	0.02	1.03 (0.93, 1.13)	0.58	1.06 (0.95, 1.18)	0.28	1.17 (1.09, 1.26)	<0.01	0.95 (0.88, 1.02)	0.17
Diabetes (yes *versus* no)	1.63 (1.37, 1.95)	<0.01	2.67 (2.02, 3.51)	<0.01	2.08 (1.52, 2.83)	<0.01	1.03 (0.84, 1.25)	0.81	1.76 (1.40, 2.21)	<0.01
Hyperlipidemia (yes *versus* no)	2.26 (1.76, 2.90)	<0.01	1.69 (1.21, 2.37)	<0.01	1.93 (1.25, 2.97)	<0.01	1.09 (0.86, 1.39)	0.48	2.04 (1.46, 2.84)	<0.01
BMI	1.01 (1.00, 1.02)	0.29	1.01 (0.99, 1.03)	0.47	1.00 (0.98, 1.02)	0.95	1.15 (1.13, 1.17)	<0.01	1.00 (0.99, 1.02)	0.77
Systolic BP (10-mmHg increase)	1.01 (0.97, 1.05)	0.80	1.07 (1.00, 1.14)	0.05	1.05 (0.97, 1.12)	0.23	1.21 (1.15, 1.28)	<0.01	1.06 (1.01, 1.12)	0.02
Current smoker (yes *versus* no)	1.33 (1.06, 1.69)	0.02	1.56 (1.03, 2.35)	0.03	1.43 (0.90, 2.28)	0.13	1.43 (1.09, 1.89)	0.01	2.19 (1.67, 2.88)	<0.01
Family history of premature CHD	1.80 (1.46, 2.20)	<0.01	1.84 (1.33, 2.56)	<0.01	1.91 (1.34, 2.73)	<0.01	1.27 (0.99, 1.62)	0.06	1.16 (0.90,1.51)	0.25
Serum hemoglobin	0.96 (0.91, 1.01)	0.14	0.91 (0.83, 0.99)	0.03	0.87 (0.79, 0.95)	<0.01	0.87 (0.82, 0.93)	<0.01	0.97 (0.91, 1.04)	0.45
hs-CRP (1 SD)	1.03 (0.96, 1.12)	0.40	1.00 (0.85, 1.16)	0.95	0.97 (0.81, 1.16)	0.71	1.00 (0.91, 1.09)	0.92	1.12 (1.03, 1.22)	<0.01
Serum phosphate	1.06 (0.93, 1.22)	0.37	1.35 (1.09, 1.66)	<0.01	1.41 (1.21, 1.78)	<0.01	—	—	1.08 (0.91, 1.27)	0.39
Corrected serum calcium	1.03 (0.86, 1.22)	0.79	1.04 (0.79, 1.38)	0.76	0.95 (0.70, 1.31)	0.77	—	—	0.93 (0.75, 1.15)	0.51
Total iPTH	—	—	1.00 (1.00, 1.00)	0.43	1.00 (1.00, 1.00)	0.63	—	—	—	—

CAC, coronary artery calcium; CVD, cardiovascular disease; LVH, left ventricular hypertrophy; ABI, ankle-brachial index; CI, confidence interval; OR, odds ratio; NH, non-Hispanic; eGFR, estimated glomerular filtration rate; HS, high school; BP, blood pressure; Hs-CRP, high-sensitivity C-reactive protein; SD, standard deviation; iPTH, intact parathyroid hormone; BMI, body mass index.

[a]Adjusted for age, gender, education, and clinical site.

[b]Additionally adjusted for all variables listed. CVD, CAC, and ABI models were additionally adjusted for serum phosphate and calcium. Intact PTH (iPTH) was only analyzed in the CAC models.

achievement of guideline-recommended goals for secondary prevention of CVD (Table 5) (21). Compared with non-Hispanic Caucasians and African Americans, Hispanics with self-reported CVD were less likely to take daily aspirin (48% [H], 68% [C], 58% [A]; $P < 0.04$) and to have hemoglobin A1C <7% (39% [H], 62% [C], 53% [A]; $P < 0.001$). Compared with non-Hispanic Caucasians, Hispanics with self-reported CVD were less likely to exercise regularly (39% [H] *versus* 53% [C], $P = 0.005$) and less likely to have BP <130/80 mmHg (40% [H] *versus* 62% [C], $P < 0.001$). Compared with non-Hispanic African Americans, Hispanics were more likely to have a waist circumference <40 in for men and <35 in for women (35% [H] *versus* 22% [A], $P = 0.003$). Use of renin-angiotensin system (RAS) blockers and β-blockers was similar between groups.

Discussion

At the time of enrollment in the CRIC/HCRIC studies, Hispanics with CKD were at significant socioeconomic disadvantage and had a higher prevalence of most traditional and nontraditional CVD risk factors compared with non-Hispanics, even after accounting for differences in kidney function. However, Hispanics generally had a lower prevalence of most self-reported and subclinical measures of CVD, with the exception of LVH, which was exceptionally common among Hispanics. Furthermore, Hispanics with self-reported CVD were less likely to achieve therapeutic goals for secondary prevention of CVD, including regular exercise, BP, and glycemic control. To our knowledge, our study is the first systematic evaluation of the clinical epidemiology of CVD in Hispanics with CKD across a wide range of age and in comparison with other racial/ethnic groups.

Similar to our findings, in a general population sample from NHANES III, the prevalence of self-reported CHD was lower in Mexican Americans despite the higher prevalence of diabetes, hypertension, and physical inactivity compared with non-Hispanic Caucasians (22). Possible explanations for these disparities between Hispanics and non-Hispanics include biologic or genetic differences, and cultural variation in disease self-report. In addition, limited access to medical care might decrease the possibility of being diagnosed with a mild cardiovascular event.

Nearly two thirds of Hispanics with CKD had poorly controlled hypertension (BP >130/80 mmHG), a rate which was almost twofold higher than that observed in non-Hispanic Caucasians in our cohort. Similarly, within NHANES III, Mexican Americans had the highest rate of uncontrolled hypertension (23). In addition to poorly achieved recommended BP goals, Hispanics were less likely to achieve many other recommended goals for secondary prevention of CVD. The generally poor rates of use of these preventive measures may be related to lack of access to medical care, as evidenced by lack of health insurance in 23% of Hispanics and late or no referral for nephrology care, ineffective patient-provider communication, factors associated with acculturation, and low adherence to medication and medical advice (24).

We observed a significantly higher prevalence of LVH among Hispanics compared with non-Hispanic Caucasians, as the adjusted prevalence was nearly twice that in the former compared with the latter group. Given the known relationship between hypertension and LVH, this finding is likely best attributed to the higher rates of poor BP control among Hispanics (25,26). Other factors that may explain this increased risk include higher body mass index and lower hemoglobin observed in Hispanics compared with non-Hispanic Caucasians (16). Our findings are consistent with prior studies of non-CKD populations in which Hispanics have been found to have higher prevalence of LVH compared with non-Hispanics (27–29). In the Multi-Ethnic Study of Atherosclerosis (MESA), Hispanics were similarly almost twice as likely as non-Hispanic Caucasians to have LVH after adjustment for hypertension and other covariates (28). These results have clinical relevance because LVH is known to be an independent predictor of cardiovascular morbidity and mortality, and, therefore, more aggressive BP control among Hispanics could contribute to reducing LVH among this group (25,30).

We considered two cut-off points for CAC (>100 and >400) as consistent with subclinical CVD based on prior

Table 5. Achievement of guideline-recommended goals for secondary prevention of CVD among participants with any self-reported CVD at baseline

Variables	Hispanic $n = 138$	Non-Hispanic Caucasian $n = 507$		Non-Hispanic African American $n = 627$	
	n (%)	n (%)	P	n (%)	P
Not current smoker	132 (96)	454 (90)	0.03	488 (78)	<0.01
BP <130/80 mmHG	55 (40)	316 (62)	<0.01	244 (39)	0.84
LDL ≤100	101 (73)	361 (71)	0.65	352 (56)	<0.01
Physical activity 30 min/day, at least 5 d/wk	51 (39)	257 (53)	<0.01	259 (44)	0.23
BMI <25 kg/m^2	13 (9)	74 (15)	0.12	76 (12)	0.37
Waist circumference: men <40 in, women <35 in	47 (35)	154 (31)	0.35	138 (22)	<0.01
Hg A1C <7%	54 (39)	313 (62)	<0.01	333 (53)	<0.01
Aspirin use	66 (48)	343 (68)	<0.01	363 (58)	0.03
RAS blocker use	100 (73)	397 (79)	0.13	479 (77)	0.30
β-blocker use	104 (76)	346 (69)	0.11	452 (73)	0.40

BMI, body mass index; Hg, hemoglobin; RAS, renin-angiotensin system.

studies suggesting that these cut-off points are associated with increased risk of cardiovascular disease events and all-cause mortality (31–38). Consistent with the lower prevalence of self-reported CHD among Hispanics with CKD compared with non-Hispanic Caucasians, we also found a lower prevalence of CAC >100. Similar results were observed in the subgroups of participants with advanced CKD and diabetes. In adjusted analyses of the combined CRIC and HCRIC cohorts, this difference was attenuated and no longer statistically significant, which could be due to the small number of Hispanics who underwent CAC measurement. Similarly, in a non-CKD cohort, the prevalence of coronary calcification (CAC >0) was significantly lower in Hispanics compared with Caucasians, after adjustment for coronary risk factors (31). In contrast, the risk for CAC >400 was two times greater in Hispanics than non-Hispanic African Americans, even after adjustment for sociodemographic and clinical factors. Few studies have compared CAC in Hispanics with CKD with other racial/ethnic groups. A cross-sectional study of 90 nondialyzed patients with diabetes (60 Hispanics and 30 African Americans) reported more prevalent and severe CAC among individuals with diabetic nephropathy but did not provide a direct comparison between Hispanics and African Americans (37).

It has been reported previously that the prevalence of peripheral arterial disease in the general population is higher among African Americans than Hispanics (13,39,40). Our study is the first to report the prevalence of measured peripheral arterial disease in a cohort of Hispanics with CKD not yet on dialysis. Similar to prior studies in patients without CKD, we found that Hispanics had lower prevalence of abnormal ABI than non-Hispanic African Americans, which was not present in adjusted analyses and appeared to be explained by differences in sociodemographic factors.

Our study has limitations. First, the majority of Hispanics were recruited at a single center and, therefore, findings may not be generalizable to all U.S. Hispanics. However, the composition of the HCRIC cohort is reflective of the heterogeneity of the U.S. Hispanic population in terms of country of origin and education (1,8,41). Moreover, the socioeconomic disadvantages observed in HCRIC are similar to those found in Hispanics with CKD in a recent NHANES III analysis (8). Also, findings from our cohorts may not be generalizable to all types to kidney disease, such as glomerulonephritis. However, the CRIC study is representative of the ESRD population in which diabetes and hypertension are the primary diagnosis for over 70% of patients (10). Second, clinical CVD was ascertained by self-report of prior history of MI or coronary revascularization, which might be influenced by recall bias. However, this methodology is commonly used in clinical research, and the excellent or substantial agreement between self-reported CHD and the medical record has been previously demonstrated in patients with ESRD (42). Third, we estimated GFR using the MDRD formula, which has not been validated in Hispanics; however, this formula has been used in other studies of Hispanics with CKD (8,43).

Hispanics in CRIC and HCRIC had worse BP control and a higher prevalence of LVH compared with non-Hispanic Caucasians. Moreover, Hispanics with CKD had the lowest rate of achievement of CVD therapeutic targets. The health disparities found in our study are likely the result of a complex interplay among multiple factors at the individual and community levels, the health care system, and society as a whole. The long-term impact of our findings on renal and cardiovascular outcomes will be evaluated in subsequent longitudinal analyses. Future research should be dedicated to examine etiologic factors that may explain our findings.

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References

1. US Census Bureau. Hispanic Heritage Month 2010: Sept 15-Oct 15. US Census Bureau Newsroom Web site. http://www.census.gov/newsroom/releases/archives/facts_for_features_special_editions/cb10-ff17.html. Accessed March 1, 2011
2. U.S. Renal Data System, USRDS 2010 Annual Data Report: Atlas of Chronic Kidney Disease and End-Stage Renal Disease in the United States, National Institutes of Health, National Institute of Diabetes and Digestive and Kidney Diseases, Bethesda MD, 2010
3. Weiner DE, Tighiouart H, Amin MG, Stark PC, MacLeod B, Griffith JL, Salem DN, Levey AS, Sarnak MJ: Chronic kidney disease as a risk factor for cardiovascular disease and all-cause mortality: A pooled analysis of community-based studies. *J Am Soc Nephrol* 15: 1307–1315, 2004
4. Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY: Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. *N Engl J Med* 351: 1296–1305, 2004
5. Culleton BF, Larson MG, Wilson PW, Evans JC, Parfrey PS, Levy D: Cardiovascular disease and mortality in a community-based cohort with mild renal insufficiency. *Kidney Int* 56: 2214–2219, 1999
6. Franzini L, Ribble JC, Keddie AM: Understanding the Hispanic paradox. *Ethn Dis* 11: 496–518, 2001
7. Peralta CA, Shlipak MG, Fan D, Ordonez J, Lash JP, Chertow

GM, Go AS: Risks for end-stage renal disease, cardiovascular events, and death in Hispanic versus non-Hispanic white adults with chronic kidney disease. *J Am Soc Nephrol* 17: 2892–2899, 2006
8. Mehrotra R, Kermah D, Fried L, Adler S, Norris K: Racial differences in mortality among those with CKD. *J Am Soc Nephrol* 19: 1403–1410, 2008
9. Feldman HI, Appel LJ, Chertow GM, Cifelli D, Cizman B, Daugirdas J, Fink JC, Franklin-Becker ED, Go AS, Hamm LL, He J, Hostetter T, Hsu CY, Jamerson K, Joffe M, Kusek JW, Landis JR, Lash JP, Miller ER, Mohler ER, III, Muntner P, Ojo AO, Rahman M, Townsend RR, Wright JT: The Chronic Renal Insufficiency Cohort (CRIC) Study: Design and methods. *J Am Soc Nephrol* 14: S148–S153, 2003
10. Lash JP, Go AS, Appel LJ, He J, Ojo A, Rahman M, Townsend RR, Xie D, Cifelli D, Cohan J, Fink JC, Fischer MJ, Gadegbeku C, Hamm LL, Kusek JW, Landis JR, Narva A, Robinson N, Teal V, Feldman HI: Chronic Renal Insufficiency Cohort (CRIC) Study: Baseline characteristics and associations with kidney function. *Clin J Am Soc Nephrol* 4: 1302–1311, 2009
11. Levey AS, Bosch JP, Lewis JB, Greene T, Rogers N, Roth D: A more accurate method to estimate glomerular filtration rate from serum creatinine: A new prediction equation. Modification of Diet in Renal Disease Study Group. *Ann Intern Med* 130: 461–470, 1999
12. Karnegis JN, Matts J, Tuna N: Development and evolution of electrocardiographic Minnesota Q-QS codes in patients with acute myocardial infarction. *Am Heart J* 110: 452–459, 1985
13. Allison MA, Criqui MH, McClelland RL, Scott JM, McDermott MM, Liu K, Folsom AR, Bertoni AG, Sharrett AR, Homma S, Kori S: The effect of novel cardiovascular risk factors on the ethnic-specific odds for peripheral arterial disease in the Multi-Ethnic Study of Atherosclerosis (MESA). *J Am Coll Cardiol* 48: 1190–1197, 2006
14. Allison MA, Budoff MJ, Wong ND, Blumenthal RS, Schreiner PJ, Criqui MH: Prevalence of and risk factors for subclinical cardiovascular disease in selected US Hispanic ethnic groups: The Multi-Ethnic Study of Atherosclerosis. *Am J Epidemiol* 167: 962–969, 2008
15. Kramer H, Toto R, Peshock R, Cooper R, Victor R: Association between chronic kidney disease and coronary artery calcification: The Dallas Heart Study. *J Am Soc Nephrol* 16: 507–513, 2005
16. Lauer MS, Anderson KM, Kannel WB, Levy D: The impact of obesity on left ventricular mass and geometry. The Framingham Heart Study. *JAMA* 266: 231–236, 1991
17. Menon V, Gul A, Sarnak MJ: Cardiovascular risk factors in chronic kidney disease. *Kidney Int* 68: 1413–1418, 2005
18. Moran A, Katz R, Jenny NS, Astor B, Bluemke DA, Lima JA, Siscovick D, Bertoni AG, Shlipak MG: Left ventricular hypertrophy in mild and moderate reduction in kidney function determined using cardiac magnetic resonance imaging and cystatin C: The multi-ethnic study of atherosclerosis (MESA). *Am J Kidney Dis* 52: 839–848, 2008
19. Schafer JL, Graham JW: Missing data: Our view of the state of the art. *Psychol Methods* 7: 147–177, 2002
20. National Kidney Foundation: K/DOQI clinical practice guidelines for chronic kidney disease: Evaluation, classification, and stratification. *Am J Kidney Dis* 39[Suppl 1], S1–S266, 2002
21. Smith SC, Jr, Allen J, Blair SN, Bonow RO, Brass LM, Fonarow GC, Grundy SM, Hiratzka L, Jones D, Krumholz HM, Mosca L, Pasternak RC, Pearson T, Pfeffer MA, Taubert KA: AHA/ACC guidelines for secondary prevention for patients with coronary and other atherosclerotic vascular disease: 2006 update. Endorsed by the National Heart, Lung, and Blood Institute. *Circulation* 113: 2363–2372, 2006
22. Nguyen HT, Stack AG: Ethnic disparities in cardiovascular risk factors and coronary disease prevalence among individuals with chronic kidney disease: Findings from the Third National Health and Nutrition Examination Survey. *J Am Soc Nephrol* 17: 1716–1723, 2006
23. He J, Muntner P, Chen J, Roccella EJ, Streiffer RH, Whelton PK: Factors associated with hypertension control in the general population of the United States. *Arch Intern Med* 162: 1051–1058, 2002
24. Smedley BD, Stith AY, Nelson AR: *Unequal treatment: Confronting racial and ethnic disparities in health care*, Washington DC, National Academy Press, 2002
25. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP: Prognostic implications of echocardiographically determined left ventricular mass in the Framingham Heart Study. *N Engl J Med* 322: 1561–1566, 1990
26. Rodriguez CJ, Lin F, Sacco RL, Jin Z, Boden-Albala B, Homma S, Di Tullio MR: Prognostic implications of left ventricular mass among Hispanics: The Northern Manhattan Study. *Hypertension* 48: 87–92, 2006
27. Rodriguez CJ, Sciacca RR, Diez-Roux AV, Boden-Albala B, Sacco RL, Homma S, DiTullio MR: Relation between socioeconomic status, race-ethnicity, and left ventricular mass: The Northern Manhattan study. *Hypertension* 43: 775–779, 2004
28. Rodriguez CJ, Diez-Roux AV, Moran A, Jin Z, Kronmal RA, Lima J, Homma S, Bluemke DA, Barr RG: Left ventricular mass and ventricular remodeling among Hispanic subgroups compared with non-Hispanic blacks and whites: MESA (Multi-ethnic Study of Atherosclerosis). *J Am Coll Cardiol* 55: 234–242, 2010
29. Zabalgoitia M, Ur Rahman SN, Haley WE, Oneschuk L, Yunis C, Lucas C, Yarows S, Krause L, Amerena J: Impact of ethnicity on left ventricular mass and relative wall thickness in essential hypertension. *Am J Cardiol* 81: 412–417, 1998
30. Mitchell BD, Hazuda HP, Haffner SM, Patterson JK, Stern MP: Myocardial infarction in Mexican-Americans and non-Hispanic whites. The San Antonio Heart Study. *Circulation* 83: 45–51, 1991
31. Bild DE, Detrano R, Peterson D, Guerci A, Liu K, Shahar E, Ouyang P, Jackson S, Saad MF: Ethnic differences in coronary calcification: The Multi-Ethnic Study of Atherosclerosis (MESA). *Circulation* 111: 1313–1320, 2005
32. Block GA, Raggi P, Bellasi A, Kooienga L, Spiegel DM: Mortality effect of coronary calcification and phosphate binder choice in incident hemodialysis patients. *Kidney Int* 71: 438–441, 2007
33. Budoff MJ, Shaw LJ, Liu ST, Weinstein SR, Mosler TP, Tseng PH, Flores FR, Callister TQ, Raggi P, Berman DS: Long-term prognosis associated with coronary calcification: Observations from a registry of 25,253 patients. *J Am Coll Cardiol* 49: 1860–1870, 2007
34. Budoff MJ, Nasir K, McClelland RL, Detrano R, Wong N, Blumenthal RS, Kondos G, Kronmal RA: Coronary calcium predicts events better with absolute calcium scores than age-sex-race/ethnicity percentiles: MESA (Multi-Ethnic Study of Atherosclerosis). *J Am Coll Cardiol* 53: 345–352, 2009
35. Chiu YW, Adler SG, Budoff MJ, Takasu J, Ashai J, Mehrotra R: Coronary artery calcification and mortality in diabetic patients with proteinuria. *Kidney Int* 77: 1107–1114, 2010
36. Greenland P, Bonow RO, Brundage BH, Budoff MJ, Eisenberg MJ, Grundy SM, Lauer MS, Post WS, Raggi P, Redberg RF, Rodgers GP, Shaw LJ, Taylor AJ, Weintraub WS: ACCF/AHA 2007 clinical expert consensus document on coronary artery calcium scoring by computed tomography in global cardiovascular risk assessment and in evaluation of patients with chest pain: A report of the American College of Cardiology Foundation Clinical Expert Consensus Task Force (ACCF/AHA Writing Committee to Update the 2000 Expert Consensus Document on Electron Beam Computed Tomography) developed in collaboration with the Society of Atherosclerosis Imaging and Prevention and the Society of Cardiovascular Computed Tomography. *J Am Coll Cardiol* 49: 378–402, 2007
37. Mehrotra R, Budoff M, Christenson P, Ipp E, Takasu J, Gupta A, Norris K, Adler S: Determinants of coronary artery calcification in diabetics with and without nephropathy. *Kidney Int* 66: 2022–2031, 2004
38. Watanabe R, Lemos MM, Manfredi SR, Draibe SA, Canziani ME: Impact of cardiovascular calcification in nondialyzed patients after 24 months of follow-up. *Clin J Am Soc Nephrol* 5: 189–194, 2010
39. Collins TC, Petersen NJ, Suarez-Almazor M, Ashton CM: The

prevalence of peripheral arterial disease in a racially diverse population. *Arch Intern Med* 163: 1469–1474, 2003
40. Selvin E, Erlinger TP: Prevalence of and risk factors for peripheral arterial disease in the United States: Results from the National Health and Nutrition Examination Survey, 1999–2000. *Circulation* 110: 738–743, 2004
41. Sundquist J, Winkleby M: Country of birth, acculturation status and abdominal obesity in a national sample of Mexican-American women and men. *Int J Epidemiol* 29: 470–477, 2000
42. Merkin SS, Cavanaugh K, Longenecker JC, Fink NE, Levey AS, Powe NR: Agreement of self-reported comorbid conditions with medical and physician reports varied by disease among end-stage renal disease patients. *J Clin Epidemiol* 60: 634–642, 2007
43. Coresh J, Selvin E, Stevens LA, Manzi J, Kusek JW, Eggers P, Van Lente F, Levey AS: Prevalence of chronic kidney disease in the United States. *JAMA* 298: 2038–2047, 2007

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