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Differences in Albuminuria Between Hispanics and Whites: An Evaluation by Genetic Ancestry and Country of Origin

The Multi-Ethnic Study of Atherosclerosis

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Abstract

Background— Reports show higher prevalence of albuminuria among Hispanics compared with whites. Differences by country of origin or genetic background are unknown.

Methods and Results— In Multi-Ethnic Study of Atherosclerosis, we studied the associations of both genetic ancestry and country of origin with albumin to creatinine ratio among 1417 Hispanic versus white participants using multivariable linear regression and back transforming beta coefficients into relative difference (%RD, 95% CI). Percentage European, Native American, and African ancestry components for Hispanics were estimated using genetic admixture analysis. The proportions of European, Native American, and African genetic ancestry differed significantly by country of origin ($P<0.0001$); Mexican/Central Americans had the highest Native American ($41\pm 13\%$), Puerto Ricans had the highest European ($61\pm 15\%$), and Dominicans had the highest African ($39\pm 21\%$) ancestry. Hispanic ethnicity was associated with higher albumin/creatinine ratio compared with whites, but the association varied with the country of origin (adjusted P interaction=0.04). Mexican/Central Americans and Dominicans had higher albumin/creatinine ratio compared with whites after adjustment (RD 19%, 2% to 40% and RD 27%, 1% to 61%), but not Puerto Ricans (RD 8%, -12% to 34%). Higher Native American ancestry was associated with higher albuminuria after age and sex adjustment among all Hispanics (RD 11%, 1% to 21%) but was attenuated after further adjustment. Higher European ancestry was independently associated with lower albumin/creatinine ratio among Puerto Ricans (-21%, -34% to -6%) but not among Mexican/Central Americans and Dominicans.

Conclusions— Hispanics are a heterogeneous group with varying genetic ancestry. Risks of albuminuria differ across the country of origin groups. These differences may be due, in part, to differences in genetic ancestral components.

Key Words:

[genetics](#)

[kidney](#)

[albuminuria](#)

[ancestry](#)

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Introduction

Hispanics have a higher incidence of end-stage renal disease compared with whites in the United States,¹ despite reports that Hispanics have lower prevalence of chronic kidney disease (CKD).^{2,3}

Although this discrepancy may be explained by Hispanics having faster rates of progression from CKD to end-stage renal disease,⁴ it may also be complicated by the use of different definitions to describe “Hispanics.” For example, CKD prevalence estimates have focused on Mexican Americans³ whereas end-stage renal disease estimates have included a wider group of Hispanics.¹ Because Hispanic subgroups in the United States differ culturally, socially, and perhaps genetically,^{5,6} categorization of Hispanics into 1 homogeneous group could lead to spurious inferences that may not generalize.

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Differences between Hispanics and whites have also been reported for albuminuria, a marker of early kidney damage, and a known risk factor for CKD progression.⁷ Studies from the National Health and Nutrition Survey showed that albuminuria is more common among Hispanics of Mexican origin compared with whites.^{8,9} This has also been documented in other studies with representation from several Hispanic subgroups.^{4,10} However, none of these studies has evaluated whether the risk of albuminuria is uniform across Hispanic subgroups of differing ancestral origin or whether differences affect comparisons with non-Hispanic whites.

In addition, it is unknown whether the reported differences in albuminuria between Hispanics and whites are associated, at least in part, with genetic predisposition. Hispanics are known to be genetically admixed with European, Native American, and African ancestry, and the degree of admixture may vary by country of origin.^{5,11-13} Determination of individual genetic ancestry in an admixed population such as Hispanics can be obtained using a series of markers informative for ancestry by genetic admixture analysis.¹⁴⁻²⁰ This method quantifies the proportion of an individual's genome that is of a given ancestral origin. Admixture analysis may offer insights into whether genetic ancestry varies significantly by country of origin among Hispanics and whether genetic ancestry explains, at least in part, differences in kidney disease risk factors such as albuminuria. It may also allow the study of potential genetics versus sociodemographic contributions to these associations. In addition, genetic admixture analysis may provide a method for future gene identification by genetic admixture mapping.^{11,21,22}

We designed this study in the Multi-Ethnic Study of Atherosclerosis (MESA) to determine (1) whether genetically determined individual African, Native American, or European ancestry differs by country of origin among Hispanics; (2) whether country of origin is associated with differences in cardiovascular risk factors and albuminuria within Hispanic subgroups and when compared with whites; and (3) whether genetically determined individual ancestry is independently associated with albuminuria among Hispanics.

Methods

Subjects

The MESA is a large National Heart, Lung, and Blood Institute–sponsored study designed to understand subclinical cardiovascular disease and its progression in a multiethnic cohort. Details on recruitment and design have been previously published.²³ Briefly, MESA recruited 6814 men and women who were between 45 and 84 years old, were free of cardiovascular disease, and who self-identified as white, African American, Hispanic, or Chinese–American. Subjects were recruited from Baltimore City and Baltimore County, Md; Chicago, Ill; Forsyth County, NC; Los Angeles County, Calif; Northern Manhattan and the Bronx, NY; and St. Paul, Minn between July 2000 and August 2002. The institutional review boards at all participating centers approved the study, and all participants gave informed consent.

A subcohort of 2880 MESA subjects were selected for genetic studies from subjects who gave informed consent for DNA extraction and genetic substudy and had samples in the study DNA laboratory with sufficient DNA. Participants were balanced by ethnic group representation (≈ 720 per group) and equality by gender. For these analyses, we included self-identified Hispanics and non-Hispanic whites who were successfully genotyped and had a measure of albuminuria (705 Hispanics and 712 Whites) at baseline for a total $N=1417$.

Primary Predictors

Self-reported race/ethnicity and self-reported country of origin were assessed at baseline by questionnaire.²³ Individual genetic ancestry was determined by admixture analysis as described below.

Primary Outcome

The primary outcome of this study was albuminuria calculated as albumin to creatinine ratio in milligrams per gram from a spot collection at baseline. Albumin to creatinine ratio was log transformed because of the skewed distribution and used as a continuous variable. Urine albumin and creatinine were measured by nephelometry and the rate Jaffe reaction, respectively. Microalbuminuria was defined as ≥ 30 mg/g and macroalbuminuria was defined as ≥ 300 mg/g.

DNA Extraction and Genotyping

DNA was extracted from peripheral leukocytes isolated from packed cells of anticoagulated blood by use of a commercially available DNA isolation kit (Puregene; Gentra Systems, Minneapolis, Minn). The DNA was quantified by determination of absorbance at 260 nm followed by PicoGreen analysis (Molecular Probes, Inc., Eugene, Ore). Two vials of DNA were stored per participant at -70°C .

Genotyping was performed by Illumina Genotyping Services (Illumina Inc., San Diego, Calif) using their proprietary GoldenGate assay. Details on quality control have been previously published.²⁴

Selection of Ancestry Informative Markers

Ancestry informative markers (AIMs) are single-nucleotide polymorphisms (SNPs) that are known to have different allele frequencies between ancestral populations. AIMs in MESA were selected to maximize differential allele frequency between 2 or more of the 4 ethnic groups and to distribute the SNPs as evenly across the genome as possible. AIMs in MESA were genotyped in 2 panels. In MESA panel 1, AIMs were selected from an Illumina proprietary SNP database to maximize the difference in allele frequencies between any pair of ethnic groups: Caucasian versus African American; Caucasian versus Chinese American; African versus Chinese American. For MESA CG panel 2, additional markers informative for Native American ancestry were selected from published lists.^{25,26} A total of 199 SNPs were successfully genotyped.

Determination of Individual Ancestry

We estimated individual ancestry using 171 AIMs in MESA. We excluded markers that had no ancestral information in HapMap (N=3) and markers in the X chromosome (N=25) to be able to look for gender interactions. We used individual level genotype data from 3 ancestral populations: 60 Yorubans, 60 European Ancestors from Hapmap (CEPH) from the HapMap genome project (<http://www.hapmap.org>), and 320 Native American ancestors (ancestral data provided by Drs Seldin and Choudhry).^{25,26} Determinations of deviations from Hardy-Weinberg Equilibrium were evaluated separately for the AIMs. Information on Hardy-Weinberg Equilibrium and on informativeness of the markers have been previously reported.²⁴

Individual admixture proportions were calculated using a Markov Chain-Monte Carlo (MCMC) method^{27,28} with the program STRUCTURE 2.1 with a burn-in length of 50 000 and 50 000 iterations after burn-in, assuming independent allele frequencies. We used $K=3$ (3 parental populations) based on prior studies that Hispanics are mainly admixed with Caucasian, Native American, and African ancestries.^{12,25} This was confirmed with several iterations using $K=2$, 4, and 5, and a $K=3$ was confirmed as the best fit. We also looked at the summary statistics plots of various key summary statistics including F_{st} , alpha, and likelihoods to make sure that they have come to equilibrium and the MCMC chain has converged.²⁴

Covariates

Information on age, self-reported race/ethnicity, level of education, annual household income, and smoking history was obtained using standardized questionnaires.²³ Blood pressure measurements were obtained using the Dinamap automated blood pressure device (Dinamap Pro 100 Monitor). Three sequential measures were obtained, and the average of the second and third measurements

was recorded. Hypertension was defined as systolic pressure ≥ 140 mm Hg, diastolic pressure ≥ 90 mm Hg, or current use of antihypertensive medication. Diabetes was defined as self-report of diabetes diagnosed by a physician, fasting glucose ≥ 126 mg/dL, or use of oral hypoglycemic medication or insulin. Cigarette smoking was defined as current/former or never. Height and weight were measured with participants wearing light clothing and no shoes. Body mass index was calculated as weight in kilograms per height in meter squared. Fasting blood was collected and stored at -70°F until needed for the appropriate assays, including total cholesterol, high-density lipoprotein cholesterol, triglycerides, and glucose. Low-density lipoprotein cholesterol was calculated using the Friedewald equation.²⁹

Statistical Analysis

We first compared participant characteristics by self-reported race/ethnicity (white versus all Hispanics) using t test or χ^2 where appropriate.

In a second step, we determined individual percent African, Native American, and European ancestry for each participant. We have previously reported evidence of population substructure among Hispanics (with 61 AIMS deviating from Hardy-Weinberg Equilibrium and 14 pairs of AIMS for Hispanics having pairwise linkage disequilibrium ≥ 0.20 [r^2]).²⁴ We investigated whether ancestral estimates varied significantly by country of origin using the Kruskal-Wallis test, and we present individual admixture estimates stratified by country of origin.

We then categorized Hispanics into 3 main subgroups by country of origin as follows: Mexicans and Central Americans, Puerto Ricans, and Dominicans. We grouped Mexicans with Central Americans because they were genetically similar and had similar risk factor profiles. For these subgroup analyses, we excluded those who reported being from Cuba, South America, or other because of the small numbers in each group. In addition, for South Americans, the numbers for each individual country were too small, and South American countries differ socially and culturally and perhaps in the prevalence of kidney disease risk factors.³⁰ We then compared participant characteristics by Hispanic subgroups using t test or χ^2 where appropriate.

In a third step, we tested the association of Hispanic ethnicity with log-transformed ratio of albumin to creatinine as a continuous variable in a series of nested models using linear regression using baseline examination data. Beta coefficients were back-transformed into relative difference (RD) in percent. We first adjusted for age and sex only and then by income and education. Finally, we added adjustment for systolic blood pressure, body mass index, high-density lipoprotein and total cholesterol, triglycerides, hypertension, diabetes, smoking, and fasting glucose. We first conducted these analyses within Hispanics only to assess for differences by Hispanic subgroup, with Mexicans/Central Americans as the referent group. We then repeated the analyses including whites and tested the association of Hispanic ethnicity (all Hispanics) with albumin to creatinine ratio

compared with whites as referent. To understand whether differences within Hispanic subgroups would also be apparent when assessing differences in albuminuria between Hispanics and whites, we tested for an interaction by country of origin and stratified by Hispanic subgroup (Mexican and Central American, Puerto Rican, and Dominican).

Finally, we studied the association of European and Native American ancestry and albuminuria within all Hispanics together and then within Hispanic subgroups. We tested for interactions by country of origin. Because only $K-1$ ancestral populations are needed to describe ancestral contributions, we used European and Native American ancestry for the main analyses. We used linear regression and studied the associations per 1SD increase. We also conducted separate analyses with African ancestry because it is unknown, a priori, which ancestral component will be most associated with the outcome, and there is a potential for the associations to vary by Hispanic subgroup.

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Results

Of the 1417 participants (mean age 61 years), 40% had hypertension, 12% were diabetic, and 52% were smokers. Based on self-reported race/ethnicity, compared with whites, Hispanics had a higher body mass index, larger waist circumference, lower high-density lipoprotein cholesterol, higher triglycerides, and were more likely to be in the lower income and educational categories (Table 1). Overall, 134 (9.5%) participants had albumin to creatinine ratio >30 mg/g and 31 (2.2%) had macroalbuminuria (albumin to creatinine ratio >300 mg/g). Characteristics of Hispanic and white MESA participants in this study, who were randomly selected, did not significantly differ from those of the overall MESA cohort.

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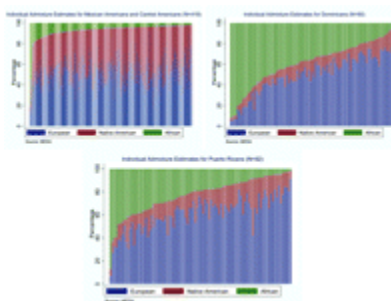
Table 1.

Baseline Characteristics of Self-Identified Whites and Hispanics in MESA

Genetic Ancestry and Country of Origin

Among all Hispanics, average Native American ancestry was $32\pm 17\%$ and mean African ancestry was $15\pm 18\%$. However, these proportions varied significantly by country of origin ($P<0.0001$; Figure 1). Within the major Hispanic subgroups in MESA, Mexican and Central Americans had a very low African ancestral component (mean $7\pm 9\%$), with the remainder of their genome mostly comprising European (mean $52\pm 13\%$) and Native American ancestry ($41\pm 13\%$). Puerto Ricans had $23\pm 16\%$ African, $61\pm 15\%$ European, and $16\pm 8\%$ Native American ancestry, whereas Dominicans had the highest

proportion of African Ancestry ($39 \pm 21\%$), with $49 \pm 19\%$ European and only $11 \pm 5\%$ Native American ancestry. Among South Americans ($N=46$), $34 \pm 19\%$ was Native American ancestry, $56 \pm 20\%$ was European and $10 \pm 15\%$ ancestry was of African origin, and the South Americans reported being from several different countries.



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Figure 1.

Individual ancestry estimates by country of origin. For Mexican/Central Americans: Mexicans=368, El Salvador=19, Guatemala=11, Nicaragua=7, and Panama=6.

Participant Characteristics by Country of Origin

We compared anthropomorphic characteristics and cardiovascular risk factors among Hispanics subgroups (Table 2). Hispanics from Mexico and Central America had significantly larger waist to hip ratios and higher triglyceride levels compared with Puerto Ricans and Dominicans. Mexicans/Central Americans had higher levels of fasting glucose compared with Puerto Ricans. Interestingly, Mexican/Central Americans had much lower rates of smoking compared with Dominicans or Puerto Ricans.

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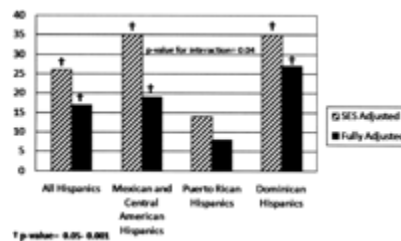
Table 2.

Characteristics of Hispanics by Country of Origin in MESA

Country of Origin and Albuminuria

Prevalence of albuminuria (>30 mg/g) varied by country of origin, with 14.1% among Mexican/Central Americans, 5.4% among Puerto Ricans, and 16.7% among Dominicans ($P=0.05$). Compared with Mexicans/Central Americans, Puerto Ricans had significantly lower levels of albuminuria, with a RD of -26% ($P=0.03$). This association persisted after adjustment for income and education (RD -23% , $P=0.05$). Although the association was attenuated after adjustment for comorbidities, it was still in the same direction of lower albuminuria for Puerto Ricans compared with Mexican/Central Americans (RD -18% , $P=0.12$). Dominicans also had lower levels of albuminuria compared with Mexican/Central Americans, but this difference was not statistically significant in unadjusted or adjusted models (RD -13% , $P=0.31$ and RD -6% , $P=0.64$, respectively).

We then studied the association of Hispanic ethnicity and albumin to creatinine ratio compared with whites. Overall, Hispanics had higher albumin to creatinine ratios compared with whites, with 12.3% Hispanics having albumin/creatinine ratio >30 mg/g versus 6.7% among whites ($P<0.001$). Even after adjustment for sociodemographic factors and comorbidities, Hispanic ethnicity was associated with higher albumin to creatinine ratio. However, these associations were not uniform across country of origin subgroups (adjusted P value for interaction= 0.04). Mexicans/Central Americans and Dominicans had higher albumin to creatinine ratio compared with whites after adjustment. However, Puerto Ricans did not have significant differences in albumin to creatinine ratio compared with whites (Figure 2).



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Figure 2.

The association between self-reported Hispanic ethnicity (all Hispanics and by country of origin) and albuminuria compared with whites in MESA.

Genetic Ancestry and Albuminuria

Increasing Native American ancestry was associated with higher levels of albuminuria in age- and sex-adjusted analyses among all Hispanics (Table 3). This association was attenuated after adjustment for income and education and was further attenuated after full adjustment, but the

direction of the association remained the same. There was no statistically significant interaction by country of origin in the association of Native American ancestry with albumin to creatinine ratio ($P=0.13$). Among Mexicans/Central Americans and Puerto Ricans, Native American ancestry was directionally associated with increased albumin to creatinine ratio, although not statistically significant. In contrast, the estimates were in the opposite direction for Dominicans, although not statistically significant ([Table 3](#)).

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Table 3.

The Association of Native American and European Ancestry (per 10% Increase) and Albuminuria Among Hispanics

Higher European ancestry was associated with lower albumin to creatinine ratio for all Hispanics. Although the effect was attenuated by further adjustment, the direction of the estimates remained consistent. Although there was no statistically significant interaction by country of origin ($P=0.89$), higher European ancestry was most strikingly protective among Puerto Ricans, where higher European ancestry was associated with lower albumin to creatinine ratio, even after full adjustment. The association of European ancestry and albumin to creatinine ratio was not significant after adjustment among Mexican/Central Americans and Dominicans, but the direction of the association was consistent across groups ([Table 3](#)).

As a confirmatory analysis, we repeated our analyses using African ancestry as our predictor. There was no significant association between African ancestry and albuminuria among all Hispanics in unadjusted or unadjusted analyses (adjusted RD -0.5 [95% CI -5% to 4%]). Interestingly, higher African ancestry was associated with higher albuminuria among Puerto Ricans, RD 11% (0.1% to 21%) after full adjustment. There were no associations between African ancestry and albuminuria among Dominicans (RD 3% [-8% to 14%]) or Mexican/Central Americans (RD -5% [-19% to 7%]).

There were no significant interactions by gender for the association of Native American or European ancestry and albuminuria ($P=0.31$ and 0.83 , respectively).

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Discussion

Using genetic admixture analysis, we found that Hispanics in MESA are admixed with European, African, and Native American ancestries and that the proportion of each ancestral group varies strikingly by country of origin, with Mexicans/Central Americans having the largest Native American

component, Dominicans having the largest African component, and Puerto Ricans having the highest European component. Moreover, we found that cardiovascular and renal risk factor profiles differ by country of origin among Hispanic subgroups, with Mexicans/Central Americans having more truncal obesity, worse lipid profile, and the highest levels of albuminuria but lower prevalence of smoking compared with Dominicans or Puerto Ricans. Differences observed among Hispanic subgroups greatly impact the association of Hispanic ethnicity and urinary albumin to creatinine ratio compared with whites, where Hispanic ethnicity was associated with significantly higher levels of albumin to creatinine ratio among Mexican/Central Americans and Dominican Hispanics but not Puerto Ricans. Most interestingly, we found that higher European ancestry may be associated with lower albuminuria, particularly among Puerto Ricans, and that Native American ancestry may, at least in part, be associated with higher levels of albuminuria among Hispanics.

Differences in the genetic ancestral component by country of origin have been documented in prior studies of Mexican Americans and Puerto Ricans.^{13,25,31,32} Our study extends these findings to other Hispanic subgroups in a large, community-based cohort in the United States. We found that the differences by country of origin are also apparent in risk factor profiles in MESA. Whether the differences in genetic ancestral component among Hispanic subgroups account for these observed differences is still unclear.^{30,33} It is also possible that social, cultural, and environmental factors may account for the differences observed in risk factor profile. Our findings have important implications for future epidemiological and genetic studies among Hispanics. Future analyses should take into account country of origin when studying Hispanic populations.

Our observation that Hispanics have higher levels of albuminuria than whites is consistent with prior reports.^{8,9,34} However, most national studies have focused on Mexican Americans.^{8,9} Our study is the first to show that levels of albuminuria among Hispanics may vary by country of origin, and that these observed differences significantly affect the association of Hispanic ethnicity and albuminuria when compared with whites. Because albuminuria is a known important risk factor for adverse cardiovascular events and kidney disease progression,⁷ our findings highlight the importance of recognizing the heterogeneity of Hispanic subgroups. That is, future studies should be aimed at understanding whether different Hispanic subgroups have different risk of kidney disease onset, progression, and whether the mediators may vary by country of origin. Accurately describing Hispanic subgroups may aid in the understanding of the conundrum of lower CKD prevalence but higher end-stage renal disease incidence among Hispanics in the United States.

Our study is the first to report the association of genetic ancestry and albuminuria among Hispanics. The observation that European ancestry may be protective for albuminuria, particularly among Puerto Ricans, may suggest that alleles more commonly found in Europeans reduce the risk of albuminuria. One study of Hispanic patients with systemic lupus erythematosus has suggested that differences in the frequency of lupus nephritis correlated with the relative proportion of non-

European admixture.³⁵ However, these findings may also be due to unmeasured environmental factors. For example, the fact that European ancestry was significantly associated with lower albumin to creatinine ratio among Puerto Ricans, but not among other groups, could be explained by differences in social or environmental factors unique to Puerto Ricans with higher European ancestry (ie, diet, socioeconomic status, neighborhood, and acculturation). Our findings that Native American ancestry may, at least in part, be associated with higher levels of albuminuria are also noteworthy. Native Americans in the United States have been reported to have >20% prevalence of albuminuria.³⁶ If higher Native American ancestry is associated with higher albuminuria among Hispanics, it is possible that genetic factors due to a common Native American ancestry may play some role in explaining this observation. The attenuation by measures of socioeconomics and comorbidities could suggest mediation of the pathway by these factors confounding or an important gene-environment interaction that was not elucidated in this study. The heritability of albuminuria among Mexican Americans with high degree of Native American ancestry has been shown in prior studies,³⁷ and certain loci have been associated with albuminuria³⁸ and other risk factors among Mexican Americans.³⁹ These suggest that albuminuria may be a phenotype amenable to admixture mapping among Hispanics, a method that has proven fruitful among African Americans.^{21,22,40} However, these maps need to take into account the 3 ancestral populations among Hispanics.^{11,41}

The strengths of our study include a large, community-based, well-characterized, multiethnic cohort. Participants were mostly healthy, had a wide age range, low rates of CKD, and had no known cardiovascular disease at study entry. Our study used 171 AIMS well selected for allele frequency differences between populations. Some error may still be present in the estimate, but prior studies have shown that accurate estimates may be obtained with 100 AIMS.⁴² However, our findings should be interpreted with caution given that we were likely limited by power in our subgroup analyses. In particular, Dominican and Puerto Rican subgroups were much smaller than Mexican/Central American, thus potentially biasing results toward null. There may have also been some misclassification because we used self-reported country of origin for categorization into genetic groups. Although our study represents the major Hispanic subgroups in the United States (<http://www.census.gov/population/www/socdemo/hispanic/reports.html>), most Hispanics were recruited from only 3 sites. Most Dominicans and Puerto Ricans were recruited from NY, whereas Mexicans and Central Americans were recruited from Los Angeles and Minn. Thus, their characteristics may not be representative of the whole country. It is also possible that some Hispanics born in United States may have parents from different countries of origin. However, the majority of MESA participants were born outside United States or were generation 1 immigrants.

In addition, we did not account for other possible factors that may differ across Hispanic groups such as acculturation,⁴³ access to health care, discrimination, and poverty.⁴⁴ We used only 1 spot measure of albuminuria rather a 24-hour collection that may lead to some misclassification, but studies have shown that 1 spot ratio is highly associated with 24-hour excretion.^{7,45}

In summary, we found that Hispanic subgroups differ significantly in their genetic ancestral components as well as in their risk factor profile by country of origin. We also found that levels of albuminuria vary significantly by country of origin among Hispanics and when compared with whites. Moreover, genetic ancestry may explain, at least in part, differences observed between Hispanics and whites in albuminuria. Our findings have important implications for future epidemiological and genetic studies, which should take into account Hispanic country of origin. In addition, albuminuria may be a phenotype amenable to admixture mapping among Hispanics.

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Footnotes

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Disclosures

None.

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Clinical Perspective

Previous Section

We investigated whether Hispanic subgroups (identified by country of origin) differ in both genetic component and risk for albumin in the urine, which is an important risk factor for cardiovascular events and kidney disease. We observed that Hispanics differ in their genetic ancestral component by country of origin. In addition, their risk of albuminuria differs by country of origin when compared with whites. Our findings suggest that country of origin should be included as a covariate when studying differences between Hispanics and non-Hispanic whites for select cardiovascular risk factors.