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Carotid artery intima-media thickness in college students: race/ethnicity matters

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Abstract

Objective—Racial/ethnic differences in common carotid artery intima-media thickness (CIMT) and in risk factors associated with CIMT have been predominantly observed in middle-aged and older individuals. We aimed to characterize racial/ethnic differences CIMT and other cardiovascular risk factors in a healthy, young-adult population.

Methods—College students were recruited as part of a study to characterize determinants of atherogenesis. Students were eligible if they were lifetime non-smokers, lived in the United States since six months of age, and attended high school in the United States. Blood pressure, heart rate, height, and weight were measured, B-mode carotid ultrasound was performed, questionnaires were administered and a 12-hr fasting blood sample was collected. Associations between CIMT and other variables were assessed in 768 students aged 18 to 25 years using linear regression analysis.

Results—In models adjusted for common cardiovascular risk factors, sex exhibited the strongest influence on CIMT, with men having 15.4 μm larger CIMT compared to women (95%CI 6.6, 24.2). Race/ethnicity was also strongly associated with CIMT. African Americans had 17.3 μm greater CIMT (95% CI −0.3, 34.8) compared to non Hispanic Whites, whereas Asians and Hispanic Whites had 14.3 (95%CI −24.3, −4.4) and 15.4 (95%CI −26.2, −4.7) μm smaller CIMT, respectively. BMI and systolic blood pressure were positively associated with CIMT.

Conclusion—The risk factors associated with atherogenesis later in life are already present and observable in college-aged young adults, so targeted campaigns to reduce life-long cardiovascular disease burden should be initiated earlier in life to improve public health.

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Conflict of Interest
None declared.

Keywords

CIMT; SBP; race; ethnicity; young adults

Introduction

Atherogenesis is a life-long process beginning early in childhood.[1] The development and progression of atherosclerosis has been extensively studied in adults, and non-invasive measures of arterial structure such as carotid artery intima-media thickness (CIMT) have been associated with and are prognostic of cardiovascular events.[2–3] However, less is known about the atherogenic process in young adults or whether endpoints such as CIMT are useful prognostic measurements in younger age groups[4–5].

Research evaluating differences in CIMT in youth has mainly focused among groups with elevated levels of certain cardiovascular disease risk factors (obesity, diabetes, dislipidemia, and hypertension) and therefore believed to be at higher risk for cardiovascular disease.[6–7] Fewer studies have evaluated predictors of CIMT in healthy children or young adults. In such studies, CIMT has been associated with one or more of the commonly known adult cardiovascular risk factors, including age, male gender, family history of heart disease, smoking, blood pressure, body mass index, cholesterol, and triglycerides, but these reported associations are not consistent across studies.[8–10]

Information about ethnic differences in CIMT, particularly at young ages, is relatively scarce. Li *et al* observed differences in risk factors for CIMT between African-Americans and whites and ethnic differences in CIMT have been observed in older populations.[11–14] Moreover, many other atherogenic risk factors become more influential at older ages, and associations of these risk factors with CIMT can also vary by race/ethnicity.[15–16] Biomarkers such as CRP which have been associated with CIMT in high risk groups, particularly in diabetics and obese adults, have not been well- characterized in younger populations.[17–18]

We investigated the association between standard cardiovascular risk factors and CIMT in a population of non-smoking college students primarily 19–20 years old. In particular, we capitalized on the multi-ethnic composition of the study population to characterize racial and ethnic differences in CIMT. We also evaluated associations between circulating C-reactive protein (CRP), homocysteine, glucose and insulin levels and CIMT, and whether any of these associations varied by sex and race/ethnicity.

Methods

Study Design

The Testing Responses in Youth (TROY) study consists of 861 college students recruited from the University of Southern California (USC) in 2007–2009. The primary purpose of the TROY study is to characterize determinants of atherosclerosis and assess lifetime histories of air pollution exposure in relation to cardiovascular disease risk. Students were eligible for study inclusion if they were lifetime non-smokers, were born in the United States or moved to the United States within the first six months of life, attended high school in a large city in the United States, and provided written informed consent to participate.

Participants attended one study visit, during which time systolic/diastolic blood pressure, heart rate, height, weight, and lung function were measured, B-mode carotid artery ultrasound was performed for assessment of CIMT. CIMT, heart rate, and blood pressure

were assessed by a single physician-imaging specialist from the USC Atherosclerosis Research Unit (ARU) Core Imaging and Reading Center (CIRC). Several self-administered questionnaires were completed during or prior to the office visit to gather information about health and socio-demographic characteristics (see online supplement for more details).

Participants provided a 12-hr fasting blood sample for lipid and biomarker analyses following completion of health testing. Of the 861 eligible participants, 14 were excluded for not meeting eligibility criteria and 78 did not provide blood samples or were missing lipid or biomarker measurements, leaving 768 participants in the study population.

The study protocol was approved by the institutional review board for human studies at the University of Southern California, and written consent was provided by the study subjects.

Health Measurements

High-resolution B-mode ultrasound images of the right common carotid artery (CCA) were obtained with a portable Biosound MyLab 25 ultrasound system attached to a 10-MHz linear array transducer and read by a single trained technician. As described previously (Patents 2005, 2006), [19–20] the jugular vein and carotid artery were imaged transversely with the jugular vein stacked above the carotid artery. All images contained internal anatomical landmarks for reproducing probe angulation and a single-limb lead electrocardiogram was recorded simultaneously with the B-mode image to ensure that CIMT was measured at the R-wave in the cardiac cycle. CCA far wall IMT was determined as the average of 70 to 100 measurements between the intima-lumen and media-adventitia interfaces along a 1-cm length just distal to the carotid artery bulb by automated computerized edge detection with an in-house developed software package (Patents 2005, 2006).[19–20] This method standardizes the timing, location and distance over which CIMT is measured, ensuring comparability across participants (see online supplement).[19–20]

Blood pressure and heart rate were measured immediately after the IMT scan by standard techniques after the subject was recumbent for at least 10 minutes. Blood pressure was measured three times in one-minute intervals, using an OMRON blood pressure monitor with automatic cuff inflation and deflation. Heart rate was measured using a 3 lead electrocardiogram as part of the Biosound MyLab 25 ultrasound system. Subject standing height was measured in stocking feet to the nearest centimeter, using a metal measuring tape placed perpendicularly to the floor through the use of a construction-type bubble level and a measurement block to properly align head orientation. Weight was measured to the nearest pound with a medical-grade scale calibrated prior to each day's testing using pre-determined calibration weights.

Biologic Measurements

Plasma and serum samples were aliquoted into 1 ml samples and stored at −80 degrees Celsius until analyzed. One ml of plasma from each subject was used to measure total cholesterol, triglyceride, and HDL cholesterol levels using an enzymatic method in conformance with the Standardization Program of the National Centers for Disease Control and Prevention. LDL-C was calculated using the Friedwald formula.[19]

One ml of serum from each subject was used to measure CRP, homocysteine, glucose and insulin. Insulin and high-sensitivity CRP were measured by a solid-phase chemiluminescent immunometric assay, while homocysteine was measured by a competitive chemiluminescent immunoassay using the Immulite 2000 analyzer (Siemens Medical Solutions Diagnostics, Malvern, PA). The sensitivities of the assays were 2 μIU/ml, 0.02 mg/dL, and 1.2 μmol/L, respectively. The inter-assay coefficients of variation were 3.6% for insulin, 7.0% for CRP and 11.5% for homocysteine. Glucose was measured by a standard procedure using the

Vitros Chemistry System. The analysis is based on the glucose oxidase-catalyzed reaction of glucose with molecular oxygen. The reaction sequence leads to the production of a highly colored red dye, whose color intensity is proportional to the amount of glucose in the sample.

Statistical Analysis

The median and inter-quartile range of subjects' health and anthropometric characteristics at study entry were calculated by gender and by race/ethnicity. Gender and race/ethnic differences were assessed using Wilcoxon rank-sum tests for continuous variables or Chi-squared tests for categorical data. The associations between CIMT and other variables were assessed using linear regression analysis. Variables initially evaluated for confounding and subsequently dropped for lack of evidence included family history of cardiovascular disease, physical activity, mother's education and second hand smoke exposure. Biomarkers of interest that were evaluated as correlates of CIMT included HDL and LDL cholesterol, total triglycerides, CRP, homocysteine, glucose and insulin. A squared term for Body Mass Index (BMI) was evaluated in the model and subsequently dropped given lack of evidence for non-linearity. BMI was divided into categories of underweight (<18.5), normal (18.5 – 25.0), overweight (25.0 – 30.0) and obese (≥30.0), and tested for linear trend in relation to CIMT. Similarly, an F-test was used to test whether ethnic categories (as a whole) were significantly associated with CIMT. A final parsimonious model included adjustments for age, sex, race/ethnicity, BMI and systolic blood pressure.

To examine whether the associations between risk variables and CIMT varied by sex or race/ethnicity, we included interaction terms in the regression models and used likelihood ratio tests to evaluate overall significance of the interactions. Regression procedures were conducted in SAS [21]. All statistical testing was conducted with a two-sided alpha level of 0.05.

We conducted a series of sensitivity analyses to evaluate the effects of excluding individuals who reported a family history of heart disease, current high cholesterol, hypertension, or use of heart medications. We also evaluated whether inclusion of individuals who reported having ever smoked non-tobacco products affected our results.

Results

Baseline characteristics of the 768 study participants are shown in Supplemental Tables S1 and S2. Little difference was observed between study participants and the 93 students who were excluded because of missing data or eligibility. All participants were college students who were on average 19 years of age and there were more females (59%) than males (41%). No participants had high blood pressure (SBP > 140mmHg) and family history of heart disease (5.5%) was rare in this population. Males tended to be more overweight, to more frequently smoke non-tobacco products (Supplement Table S3), and to have greater family history of heart disease compared to females.

Carotid intima-media thickness measurements were highly reproducible. The coefficient of variation between replicate scans (n=20) was 1.31% and the correlation coefficient was 0.97. Carotid intima-media thickness was normally distributed in this population, with a median (IQR) of 602 μm (79 μm) (Figure S1). Males (618.0 μm; IQR 91.5 μm) had significantly higher CIMT than females (592.0 μm; IQR 74.5 μm, p-value <0.001) (Figure S2). CIMT also differed by race/ethnicity; compared to non-Hispanic Whites (607.5 μm; IQR 79.0 μm), CIMT was higher in African Americans (631 μm; IQR 61.0 μm) and lower in Asians (587.5 μm; IQR 73.5 μm) and Hispanic Whites (588.5 μm; IQR 75.0 μm, p-value <0.0001) (Figure S3).

In this group of young adults, gender and ethnic variation in other common adult cardiovascular risks factors was observed. Males had higher systolic blood pressure (SBP) and lower HDL-C and LDL-C than female participants (Supplement Table S4). Race/ethnic differences in these characteristics were also observed (Tables 1 and 2). African-Americans and Hispanic Whites tended to have a larger percent of overweight and obese children and a lower level of parental education compared to non-Hispanic Whites. Mean CIMT, SBP, total cholesterol, triglycerides, HDL cholesterol and glucose also significantly differed across race/ethnicities. Levels of CRP, homocysteine, glucose and insulin were not highly correlated with one another nor with lipids (Suppl Table S5).

In univariate analyses, sex, race/ethnicity, BMI, SBP, smoking non-tobacco products and homocysteine were all significantly associated with CIMT (Table 3). Current and childhood exposure to second hand smoke was not associated with CIMT. In multivariable models, sex exhibited the strongest influence on CIMT, with men having on average 15.4 μm larger CIMT compared to women (95%CI 6.6, 24.2) (Table 4). Race/ethnicity was also strongly associated with CIMT. On average, African Americans had 17.3 μm greater CIMT (95% CI −0.3, 34.8) compared to non Hispanic Whites, whereas Asians and Hispanic Whites had 14.3 (95%CI −24.3, −4.4) and 15.4 (95%CI −26.2, −4.7) μm smaller CIMT, respectively. The observed differences in CIMT by sex and race/ethnicity were not explained by underlying cardiovascular risk factors across these groups.

Systolic blood pressure remained a significant predictor of CIMT after adjustment for various covariates (Table 4). A 1 mmHg increase in SBP was associated with a 0.5 μm increase in CIMT (95% CI 0.0, 1.0). When categorized into quartiles, CIMT was 12.6 μm (95%CI 0.3, 24.8) higher in the highest quartile of SBP (139 ≥ SBP >113mmHg) compared to the lowest quartile (81 ≤ SBP < 101 mmHg). BMI was significantly associated with CIMT in a multivariable model (β=1.4 μm, 95% CI 0.3, 2.4). When BMI was analyzed by underweight, normal, overweight and obese sub-categories, a significant trend was also observed ($p<0.02$).Overweight and obese students showed increases in CIMT (β=7.7, 95% CI: −2.5, 17.8 and β=17.6, 95% CI: −1.6, 36.8) compared to normal weight participants, whereas underweight students had a lower CIMT (β=−6.9 μm, 95%CI −25.7, 12.0).

The univariate association of homocysteine with CIMT was not found with adjustment for sex or systolic blood pressure. Similarly, the increase in CIMT seen in participants who smoked non-tobacco products was no longer statistically significant after adjustment for sex, race/ethnicity or systolic blood pressure. CRP, glucose and insulin were not associated with CIMT in multivariable models. Male and female students did not have significantly different associations between known cardiovascular risk factors and IMT nor did risk factor associations with CIMT vary by race/ethnicity.

Sensitivity analyses were conducted to evaluate whether exclusion of 54 students who reported having high cholesterol or hypertension or 42 students who reported a family history of heart disease materially affected our results. The effect of SBP on CIMT was slightly reduced when individuals with a family history of heart disease were excluded (β=0.4, p=0.12) but the effects of sex, BMI and race/ethnicity on CIMT remained unaltered. Exclusion of the 176 individuals who reported ever smoking non-tobacco products did not materially alter our conclusions, although the magnitude of the effect of being African-American on IMT was strengthened.

Discussion

To assess the cardiovascular health of young college students, we recruited and tested 768 individuals. We observed that sex, race/ethnicity, BMI and SBP were observable

atherogenic risk factors even among a select group of healthy college students. The observed differences in CIMT could not be explained by other cardiovascular risk factors that typically manifest at older ages.

Consistent with other studies both in children and in young adults, male college students tended to have thicker carotid arterial walls than female students of the same age, and BMI and SBP were positively associated with CIMT.[7–10] Differences in the distributions of CVD risk factors such as SBP, lipid levels, and BMI were also observed between men and women.[22]

Notable ethnic differences in CIMT were present in this young adult population. African-Americans tended to have marginally significantly thicker carotid arterial walls, though this may be due to the small number of African-Americans in the study. Asians and Hispanic Whites had significantly thinner arterial walls compared to non-Hispanic Whites. While our results demonstrate similar findings as in other studies for young adult African-Americans, [12, 23] we present some of the first findings for Hispanic Whites at a young age. Our racial/ethnic findings are generally consistent with data reported in older adults for CIMT [14, 24–25] and for ethnic differences in carotid plaques.[26]

We observed that racial/ethnic differences in CIMT seen in older populations are already evident at an early age, even in a select group of healthy, non-smoking college students which may not be representative of the general young adult population. The differences we observed are also consistent with trends in ethnic variation in CVD morbidity and mortality in the United States. Generally, African Americans have the highest age-adjusted death rate from CVD, followed by Whites, Hispanics, and Asians, with death rates in African Americans 2–3 times those in Asians.[27]

Racial/ethnic-specific differences in the associations between other cardiovascular risk factors and CIMT are less consistent. Differences in associations between BMI, LDL-C, triglycerides and SBP with CIMT have been observed between African-Americans and Whites,[12] and for total cholesterol, HDL-C, and diabetes with CIMT between Asian Indians and Whites.[28] Application of the Framingham Risk Score to a young adult population also demonstrated a positive association with CIMT in young adults, however, no difference in this association was observed between African-Americans and whites [11]. Race/ethnic variation in the association of biomarkers such as CRP and homocysteine with CIMT in young populations is inconsistent, with some studies observing associations[18] and others not [29]. In our population, we found no strong evidence to suggest risk factors associated with CIMT differ across race/ethnic groups.

Racial/ethnic differences in CIMT may occur for a number of reasons, both environmental and genetic. Underlying genetic differences across racial/ethnic groups provide some explanation for the observed differences in CIMT and associated risk factors. However, racial/ethnic categories used in statistical analyses are artificial constructs, since a large amount of genetic heterogeneity remains. Advances in use of ancestry information markers may help to shed light in further refining risk differences by race/ethnicity.[30]

This study was cross-sectional in nature, making temporal separation of cause and effect difficult. CIMT and biomarkers were measured at only one point in time. The study population included non-cigarette-smoking college students, which removed potential confounding by cigarette smoking, one of the greatest risk factors for cardiovascular disease. However, 23% of students reported ever having smoked non-tobacco products. While we do not have detailed information about types of products smoked, collectively the main effects of these non-cigarette smokers on CIMT were not apparent after accounting for sex, race

and blood pressure differences, and a sensitivity analysis in which these individuals were removed did not materially change our results.

High cholesterol and hypertension were also infrequent in this population, and sensitivity analyses showed little change in our conclusions when those individuals with prior report of high cholesterol or hypertension were excluded. Thus, important factors that often complicate investigations of cardiovascular risk factors in older populations were addressed. However, it is possible that differences in other factors, such as diet and physical activity, between racial/ethnic groups could explain the observed associations. Diet was not assessed and remains a limitation of this study. Physical activity was evaluated via questionnaire self-report and not found to be associated with IMT in this population.

Conclusion

Notable differences in CIMT were observed within different racial/ethnic groups, between men and women, and in relation to BMI and SBP in a population of young, healthy college students. The risk factor burden observed in college students is concordant with those risk factors found later in life, indicating that the atherogenic process has important determinants early in life even among healthy young adults and could be emphasized in public health campaigns to potentially reduce atherosclerotic burden and its consequences. The causes of the observed variation in CIMT in this population may be driven by underlying genetic components and/or environmental differences. Future investigations in the TROY study will investigate whether environmental exposures, such as lifetime exposure to air pollution, are related to CIMT.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Table 1

Demographic characteristics of TROY participants by race/ethnicity

	NHW*		African-American		Asian		HW*		Other		P-value**
	N	%	N	%	N	%	N	%	N	%	
Sex											0.15
Female	189	54.8	25	65.8	99	61.5	75	57.3	63	67.7	
Male	156	45.2	13	34.2	62	38.5	56	42.8	30	32.3	
Obesity‡											<.0001
Underweight	9	2.6	1	2.6	14	8.7	6	4.6	2	2.2	
Normal	273	79.1	23	60.5	127	78.9	77	58.8	73	78.5	
Overweight	55	15.9	12	31.6	17	10.6	34	26.0	13	14.0	
Obese	8	2.3	2	5.3	3	1.9	14	10.7	5	5.4	
Current exposure to Second-hand smoke§											0.10
No	219	63.5	19	50.0	89	55.3	89	67.9	56	60.2	
Yes	126	36.5	19	50.0	72	44.7	42	32.1	37	39.8	
Second-hand smoke exposure during childhood											0.07
No	322	93.3	31	81.6	152	94.4	118	90.1	84	90.3	
Yes	23	6.7	7	18.4	9	5.6	13	9.9	9	9.7	
Mother's Education											<.0001
High School or less	9	2.6	1	2.6	21	13.0	45	34.4	7	7.5	
Some College	62	18.0	15	39.5	31	19.3	43	32.8	26	28.0	
College grad/some grad school	274	79.4	22	57.9	107	66.5	40	30.5	60	64.5	
Unknown	.	.	.	.	2	1.2	3	2.3	.	.	
Family history of heart attack, heart failure or stroke											0.15
No	325	94.2	29	76.3	149	92.6	117	89.3	80	86.0	
Yes	15	4.4	5	13.2	8	5.0	8	6.1	6	6.5	
Don't know	5	1.5	4	10.5	4	2.5	6	4.6	7	7.5	

NHW=non-Hispanic White; HW=Hispanic White

Table 2

Descriptive statistics of baseline characteristics and biomarker measurements in Troy participants, by race/ethnicity (N=768)

	Asian (N=161)		Black (N=38)		Hispanic White (N=131)		Non-Hispanic White (N=345)		Other (N=93)		p-value
	Median	IQR	Median	IQR	Median	IQR	Median	IQR	Median	IQR	
Age	19.2	1.7	19.7	2.3	19.2	1.9	19.8	2.2	19.5	1.9	<0.0001
CIMT, µm	587.5	73.5	631.0	61.0	588.5	75.0	607.5	79.0	604.5	95.0	<0.0001
Height, cm.	165.0	14.0	170.5	14.0	168.0	13.0	172.0	13.0	169.0	14.0	<0.0001
Weight, lbs.	59.9	16.3	68.7	20.0	66.7	17.2	67.1	16.3	64.9	15.9	<0.0001
BMI, kg/m^2	21.4	3.6	23.0	5.3	23.2	5.0	22.4	3.4	22.5	3.8	<0.0001
SBP, mmHg	104.0	13.0	109.0	12.0	107.0	14.0	109.0	11.0	107.0	12.0	0.0002
DBP, mmHg	58.0	7.0	60.5	11.0	57.0	8.0	57.0	9.0	58.0	7.0	0.08
CHOL (mg/dL)	165.0	37.0	148.5	35.0	158.0	40.0	156.0	38.0	153.0	33.0	0.02
TRIG (mg/dL)	72.0	39.0	51.0	21.0	72.0	48.0	74.0	44.0	67.0	36.0	<0.0001
HDL (mg/dL)	54.0	17.0	58.5	12.0	50.0	18.0	50.0	17.0	55.0	20.0	<0.0001
LDL (mg/dL)	93.0	30.0	81.5	32.0	90.0	31.0	89.0	34.0	83.0	32.0	0.08
CRP, mg/L	0.3	0.7	0.3	0.6	0.7	1.6	0.6	1.6	0.4	1.3	<0.0001
Insulin, mIU/mL	4.3	5.9	3.9	5.2	3.7	5.5	3.6	4.7	3.1	4.6	0.27
Homocysteine, mmol/L	6.1	2.3	6.2	2.4	5.9	2.3	6.1	2.1	5.8	2.1	0.31
Glucose, mg/dL	79.0	7.0	76.0	5.0	78.0	7.0	77.0	7.0	77.0	8.0	0.03

* p-values are calculated from Kruskal-Wallis Test.

Min=Minimum value, Max=Maximum value, IQR=Interquartile range.

Table 3

The effects of adult cardiovascular risk factors on IMT in college students, using univariate regression models (N=768)

	Difference in IMT, μm	95%CI		p-value
Race/ethnicity				<.001
Non-hispanic White	*ref*			
Asian	−18.4	28.5	−8.4	<.001
African-American	17.5	−0.5	35.4	0.06
Hispanic White	−14.4	25.2	−3.6	0.01
Other	−6.2	18.5	6.0	0.32
Male sex	21.9	14.2	29.5	<.001
Age, yrs	1.5	−1.0	4.0	0.24
BMI	1.9	0.9	3.0	<.001
Systolic Blood Pressure	1.2	0.8	1.6	<.001
Mother's education				0.02
High school or less	*ref*			
Some college	13.2	−0.9	27.4	0.07
College grad/some grad school	12.2	−0.4	24.8	0.06
Unknown	−9.8	−58.8	39.2	0.69
Current SHS†	5.0	−2.9	12.9	0.21
Childhood SHS†	2.8	−11.5	17.0	0.70
HDL-C, mg/dL	−0.2	−0.5	0.1	0.11
LDL-C, mg/dL	0.0	−0.2	0.1	0.76
Triglyceride, mm/dL	0.0	−0.1	0.1	0.80
CRP, mg/L	0.2	−0.8	1.1	0.72
Homocysteine, mmol/L	2.7	0.5	4.8	0.02
Glucose, mg/dL	−0.5	−1.2	0.2	0.14
Insulin, mIU/mL	0.3	−0.5	1.2	0.46
Other-Smoker*	9.1	−0.1	18.2	0.05
Family history of heart disease§				0.64
No	*ref*			
Yes	6.6	−10.4	23.5	0.45
Don't know	−5.9	−27.2	15.4	0.59

*One subject with missing value was excluded from the analysis.

†SHS = second hand smoke

Table 4

Associations of cardiovascular risk factors with CIMT in college students: multivariable linear regression (N=768)

	Difference in IMT, μm*	95%CI		p-value
Race/ethnicity				0.001
Non-hispanic White	*ref*			
Asian		-		
	−14.3	24.3	−4.4	0.005
African-American	17.3	−0.3	34.8	0.05
Hispanic White		-		
	−15.4	26.2	−4.7	0.005
Other		-		
	−3.8	15.8	8.2	0.53
Male sex	15.4	6.6	24.2	0.001
Age, yrs	0.4	−2.0	2.9	0.72
BMI	1.4	0.3	2.4	0.01
Systolic Blood Pressure	0.5	0.0	1.0	0.04

*adjusted for age, sex, ethnicity, BMI, and systolic blood pressure

CIMT=carotid intima-media thickness, BMI = body mass index