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Associations of Anger, Anxiety and Depressive symptoms with Carotid Arterial Wall Thickness: the Multi-Ethnic Study of Atherosclerosis

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Abstract

Objective—Carotid arterial wall thickness, measured as intima-media thickness (IMT), is an early subclinical indicator of cardiovascular disease. Few studies have investigated the association of psychological factors with IMT across multiple ethnic groups and by gender.

Methods—We included 6,561 men and women (2,541 whites, 1,790 African Americans, 1,436 Hispanics, and 794 Chinese) aged 45 to 84 years who took part in the first examination of the Multi-Ethnic Study of Atherosclerosis. Associations of trait anger, trait anxiety, and depressive symptoms with mean values of common carotid artery (CCA) and internal carotid artery (ICA) IMTs were investigated using multivariable regression and logistic models.

Results—In age, gender, race/ethnicity-adjusted analyses, the trait anger score was positively associated with CCA and ICA IMTs (mean differences per one SD increment of trait anger score were 0.014 (95% CI, 0.003–0.025, p=0.01) and 0.054 (0.017–0.090, p=0.004) for CCA and ICA IMTs respectively). Anger was also associated with the presence of carotid plaque (age, gender, and race/ethnicity-adjusted odds ratio per one SD increase in trait anger: 1.27 (95%CI, 1.06–1.52)). The associations of the trait anger score with thicker IMT was attenuated after adjustment for covariates, but remained statistically significant. Associations were stronger in men than in women and in whites than in other race/ethnic groups but heterogeneity was only marginally statistically significant by race/ethnicity. There was no association of depressive symptoms or trait anxiety with IMT.

Conclusions—Only one of the three measures examined was associated with IMT and the patterns appeared to be heterogeneous across race/ethnic groups.

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Keywords

Anger; Anxiety; Carotid artery wall thickness; Depressive symptoms; Intima-media thickness; Race/Ethnicity

It has long been hypothesized that psychological factors such as depressive symptoms, perceived stress, anger, hostility, and anxiety may play a role in the development of cardiovascular disease. A number of studies have found that these psychological characteristics are associated with increased risk of cardiovascular morbidity and mortality (1–5). However, questions remain regarding whether these effects are causal, and if so the mechanisms through which they may be mediated. An important question is whether psychological factors are related to the development of atherosclerosis, the triggering of clinical events in persons with underlying disease, or both (6).

Over the past few years, a number of standardized measures of subclinical atherosclerotic disease have been incorporated into epidemiologic studies (7). One such measure is carotid artery intima-media thickness (IMT). Previous epidemiological studies have reported that carotid IMT thickening predicts future stroke and myocardial infarction events (8–11). In addition, carotid IMT has been linked to cardiovascular risk factors in cross sectional and longitudinal studies (12, 13). The identification of associations of psychological factors with subclinical markers of atherosclerosis would be consistent with effects of psychological factors on the early development of atherosclerotic disease.

Although there is an extensive literature showing a link between psychological factors and clinical coronary heart disease and stroke, studies of psychological factors and subclinical carotid IMT are less common and have produced variable results. Several studies have reported associations of trait anger (14, 15), suppressed anger (16, 17), and hostility (16) with greater carotid IMT. Similarly, depressive symptoms have been linked to increased IMT in some studies (18, 19) but not in others (20, 21). Further, there were also inconsistent results in the association between anxiety and carotid IMT (16, 18, 22).

Prior studies of psychological factors and carotid IMT have varied widely in sample size (ranged from 200 to 3,781) (15–22). In addition, both levels and effects of psychosocial risk factors may vary by gender and race/ethnicity because of the presence of varying social exposures which may be antecedents or modulators of psychosocial characteristics (23–27). It is therefore important to examine these associations in diverse samples. However, few studies have had the sample size or gender and race/ethnic diversity necessary to examine these variations. We used data from the Multi-Ethnic Study of Atherosclerosis (MESA), a large multiethnic study of subclinical atherosclerosis and its predictors, to examine associations of depressive symptoms, anger and anxiety with state-of-the-art measures of carotid IMT in a large, population-based sample free of cardiovascular disease. We also investigated whether these associations differed by gender or race/ethnicity.

Methods

Study Population

The MESA cohort includes 3,213 men and 3,601 women aged 45 to 84 years who were free from cardiovascular disease (specifically diagnosed coronary heart disease, stroke, and atrial fibrillation). Participants were recruited between 2000 and 2002 in 6 US field centers: Baltimore, Maryland; Chicago, Illinois; Forsyth County, North Carolina; Los Angeles County, California; New York, New York; and St. Paul, Minnesota. The study design is described in detail elsewhere (28).

We excluded participants with missing or poor quality IMT data (n=196) or missing data for risk factors such as blood pressure (BP), anthropometrics, and psychological variables (n=57). The remaining 6,561 participants (2,541 whites, 1,790 African Americans, 1,436 Hispanics, and 794 Chinese) were included in the analyses. The study protocol was approved by the institutional review boards at each site and informed written consent was obtained from each participant.

Baseline Measurements

Three domains were selected as measure of psychological distress: anger, anxiety, and depressive symptoms. Questionnaires were administered as part of the baseline visit in English, Spanish, or Chinese to obtain information about anger and anxiety (the Spielberger Trait Anger and Anxiety Inventory; STAXI) (29) and depressive symptoms (the Center for Epidemiologic Studies Depression Scale; CES-D) (30). The trait anger scale was designed to assess an individual's disposition to feeling angry, and trait scales were chosen over state scales to better capture the relations that occur over longer periods of time. The trait anxiety scale captures differences between people in their disposition to respond to stressful situations with varying amounts of state anxiety. The STAXI and the CES-D have been used extensively in community samples and were scored according to standard criteria, with possible ranges of 10 to 40 for trait anger, 10 to 40 for trait anxiety, and 0 to 60 for CES-D; higher scores indicated higher levels of anger, anxiety, and depressive symptoms, respectively. There were weak to moderate correlations among the psychological variables; the Spearman correlation coefficients were 0.30 between depressive symptoms and anger, 0.61 between depressive symptoms and anxiety, and 0.40 between anger and anxiety.

During the examination, height, weight, and waist and hip circumferences were measured. Resting blood pressure was measured three times in the seated position using a Dinamap model Pro 100 automated oscillometric sphygmomanometer (Critikon, Tampa, Florida). The average of the last two measurements was used in analysis. Hypertension was defined as systolic blood pressure of ≥140 mmHg, diastolic blood pressure of ≥90 mmHg, and/or current treatment with antihypertensive medication. Body mass index (BMI) was calculated as weight (kg)/height $(m)^2$. Physical activity was measured by using a detailed, semi-quantitative questionnaire adapted from the Cross-Cultural Activity Participation Study (31). Minutes per week of all light, moderate, and vigorous activities were multiplied by their individual metabolic equivalent (MET) values to compute the total MET min/wk.

For carotid ultrasonography, images of the right and left common carotid and internal carotid arteries were captured, including images of the near and far wall, using high-resolution B-mode ultrasound (Logiq 700 ultrasound machine; General Electric Medical Systems) (32). The common carotid artery (CCA) IMT was assessed at the 1-cm segment proximal to the bifurcation) and the internal carotid artery (ICA) IMT was assessed at the carotid bifurcation and 1 cm distal to the bifurcation. IMT was characterized by the mean of all available maximum wall thicknesses across both left and right sides. The intra-class correlation coefficients for intra-reader and inter-reader reproducibility were 0.98 and 0.86, respectively, for CCA measurements, and 0.99 and 0.94 for ICA measurements. A standardized protocol with quality control procedures was used and interpretation was done at a centralized reading station, New England Medical Center, Boston, Massachusetts (32, 33). IMT was examined as a continuous variable and also dichotomized with “plaque” defined as wall thickness (CCA and/or ICA IMT) ≥1.5mm (34).

Total and HDL cholesterol, triglycerides, and glucose levels were measured from blood samples obtained after a 12-hour fast. We defined diabetes as fasting serum glucose ≥7.0 mmol/L (126 mg/dL) or pharmacological treatment for diabetes.

Statistical analysis

We examined associations of psychological variables with both CCA and ICA IMTs independently, as has been the approach in previous MESA analyses (35), because previous studies have shown that CCA and ICA IMTs may be differentially related to cardiovascular risk factors and outcomes (36). Analysis of covariance (ANCOVA) was used to examine differences in mean IMT across categories of demographic and cardiovascular risk factors. Linear regression analysis was used to investigate associations of psychological variables with cardiovascular risk factors and carotid artery IMTs. We estimated mean differences in IMT per one standardized deviation (SD) increase in continuous psychological predictors and changes in R squared values.

In order to evaluate their impact of psychological factors in having substantially thickened arteries, we also estimated adjusted odds ratios (OR) and 95% confidence intervals (CI) of the presence of carotid plaque per one SD increase in continuous psychological variables after adjustment for age, gender, and race/ethnicity, and after additional adjustment for cardiovascular risk factors using logistic regression. Since a CES-D score of 16 or higher is a well-accepted, standard cutoff for identification of clinically significant depression (30), CES-D scores were also examined dichotomously (CES-D <16 as referent). Covariates included age (years), gender, race/ethnicity (assessed based on US Census 2000 questions), systolic blood pressure (mmHg), use of antihypertensive medication (yes, no), smoking status (never/former, current smokers), BMI (kg/m^2), diabetes mellitus (yes, no), total/HDL cholesterol ratio, education (grade 8 or less, grades 9–11, complete high school/GED, technical school/associate degree/some college, bachelors degree, and graduate/professional school), and physical activities (MET min/wk). These variables were included because they may be confounders of the association between psychological variables and IMT thickness and were included as covariates in previous studies. Diet was not included as covariates in the present study because a dietary pattern based on variation in food group intake was not

significantly associated with carotid IMT in MESA (37). We also analyzed these associations stratified by gender, and ethnicity. All statistical analyses were conducted using SAS, version 9.1 (SAS Institute, Inc., Cary, NC, USA).

Results

Age- and race/ethnicity-adjusted mean values of carotid artery IMT were higher among men than among women (Table 1). African Americans had higher carotid IMT than Hispanics and Chinese but no differences were observed between African Americans and whites (p-value <0.01 for African Americans versus Hispanics and Chinese, and 0.23 versus whites). Age was positively associated with gender- and race/ethnicity-adjusted mean values of carotid IMT (p for trend <0.001). Age-, gender-, and race/ethnicity- adjusted mean values of carotid IMT were higher among participants with hypertension, high cholesterol levels (≥220mg/dl), diabetes mellitus, current smoking or obesity (BMI ≥30kg/m^2) than among participants without each of these cardiovascular risk factors.

Table 2 presents age, gender, and race/ethnicity-adjusted means or prevalences of cardiovascular risk factors according to quartiles of psychological variables. The trait anger, trait anxiety, and depressive symptoms scores were positively associated with the prevalence of current smoking and hypertension. The trait anger and depressive symptoms, but not trait anxiety, scores were positively associated with the prevalence of diabetes mellitus and mean values of BMI. There were no significant associations of any psychological variables with total cholesterol levels.

As shown in Table 3, the trait anger score was significantly positively associated with CCA and ICA IMTs after adjustment for age, gender, and race/ethnicity. Mean differences in IMT per one SD increase in trait anger were 0.014 (95% CI, 0.003 to 0.025, p=0.01) and 0.054 (95% CI, 0.017 to 0.090, p=0.004), for CCA and ICA IMTs respectively. Addition of trait anger scores to models including age, gender and race/ethnicity increased the R^2 by 0.1% for both CCA and ICA IMTs. The associations of the trait anger score with thicker IMT was attenuated after adjustment for covariates, but remained statistically significant for ICA IMT (p=0.03). The associations were virtually unchanged after further adjustment for depressive symptoms and anxiety (data not shown). Depressive symptoms and anxiety were not associated with CCA or ICA IMTs. Further, adjustment for use of antidepressant medications (n=424) had no impact on these results. There were no interactions between anger, anxiety, and depressive symptoms for CCA and ICA IMTs.

Anger scores were also associated with the odds of having carotid plaque: the age, gender, and race/ethnicity-adjusted OR of having carotid artery plaque (18.7% of the sample had plaque) per one SD increase in trait anger was 1.27 (95%CI, 1.06, 1.52). After further adjustment for cardiovascular risk factors, the association was 1.22 (95%CI, 1.01, 1.47). There was no association of depressive symptoms or trait anxiety with carotid artery plaques. Age, gender, and race/ethnicity-adjusted OR of having carotid artery plaque for persons with depression (CES-D ≥16) vs. those without depression (CES-D <16) was 1.08 (95%CI, 0.88, 1.32).

Table 4 and 5 present multivariable-adjusted mean differences in CCA and ICA IMTs per one SD increase in trait anger scores and 95% CIs stratified by gender and race/ethnicity. Associations of trait anger with CCA and ICA IMTs were stronger in men than women (Table 4), but tests for heterogeneity by gender were not statistically significant ($p>0.10$).

Trait anger was positively associated with CCA and ICA IMTs in whites and associations in a similar direction were observed in Hispanics although they were not statistically significant. No associations were observed in African Americans or Chinese (Table 5). There was borderline statistical evidence that associations of trait anger with ICA IMTs varied by race/ethnicity (P value for heterogeneity by race/ethnicity =0.09). Anger was positively associated with the presence of plaque in whites but weaker and non-statistically significant associations were observed in Hispanics and African Americans and no associations were observed in Chinese (P value for heterogeneity by race/ethnicity =0.05). There were no significant associations of depressive symptoms and anxiety with CCA and ICA IMTs in either gender or in any race/ethnicity group (data not shown).

Discussion

Only one of the three measures of psychological distress that we examined was associated with subclinical atherosclerosis as indexed by carotid artery IMT. In the full sample, trait anger measured by the STAXI was positively associated with carotid artery IMT. Associations were present for both ICA and CCA, and were consistent for continuous versions of wall thickness as well as for the presence of plaque. There was some evidence of heterogeneity in this relation across population subgroups. Associations of anger with IMT were stronger in men than in women although this heterogeneity was not statistically significant. It also varied somewhat across race/ethnic groups with the clearest associations being present in whites. Weaker and non significant associations were observed in Hispanics, and African Americans (only for plaque), and no associations of anger with IMT were present in Chinese. Although an association of psychological factors with incidence of cardiovascular diseases is well established, the mechanisms underlying this association remain unclear. Our findings are consistent with the hypothesis that trait anger may be associated with incidence of cardiovascular diseases at least in part through the development of carotid atherosclerosis at least in some population subgroups (15). However, there was no evidence that psychological distress generally is associated with carotid artery IMT since we found no associations for depressive symptoms or trait anxiety.

Prior work on the relationship between anger and IMT has had mixed results. A cross-sectional study of 14,098 US men and women showed that trait anger was associated with carotid IMT in black men, but not whites or black women (14). In contrast, a longitudinal study of 209 women, predominantly white, reported a positive association between trait anger and progression of carotid IMT (15). Another cross-sectional study of 200 US men and women reported a positive association of suppressed anger with carotid IMT, but no associations were observed for anger-out or trait anger measures (17). In our analyses, trait anger was associated with carotid IMT thickening in the full sample. Stratified analyses showed that associations were largely confined to whites although there was a suggestion of weak associations in African Americans and Hispanics for some measures.

Specific mechanisms for possible chronic effects of trait anger on carotid artery wall thickness have not been fully elucidated. In the present study, the trait anger scores were positively associated with the prevalence of hypertension and diabetes. Anger scores have been shown to predict later hypertension incidence (38), therefore, it is possible that trait anger may be associated with progression of carotid artery wall atherosclerosis through the development of hypertension. Another study also showed that the association between trait anger and carotid IMT may be mediated via metabolic syndrome (15). Other putative mechanisms linking anger to the development of atherosclerosis include inappropriate health behaviors such as smoking and heavy drinking, hypothalamo-pituitary-adrenal (HPA) axis, sympathetic nervous system activation, or neuroimmune modulation of inflammatory processes (39–41). The associations that we observed persisted after adjustment for established cardiovascular risk factors, but these cross-sectional analyses are limited in their ability to draw conclusions regarding mediating mechanisms.

Several studies have reported that depressive symptoms and anxiety appear to be associated with carotid artery wall thickness (18, 19, 22). The Pittsburgh Healthy Heart Project of 324 men and women aged 50–70 years and the Cardiovascular Health Study of 3,781 men and women aged ≥65 years reported positive associations of depressive symptoms with progression of carotid IMT (18, 19), but the Cardiovascular Risk in Young Finns (CRYF) Study of 1,126 young adults and the Baltimore Longitudinal Study of Aging of 556 men and women aged 20–93 years showed no longitudinal association between depressive symptoms and carotid IMT in both men and women (20, 21), while a positive association between depressive symptoms and IMT was observed among men in cross-sectional analyses in the CRYF Study. The Etude sur le Vieillissiment Artériel (EVA) Study of 726 men and women aged 59–71 years reported a positive association of sustained anxiety with progression of carotid IMT in both men and women (22), but the Pittsburgh Healthy Heart Project did not find the association (18). We found no evidence of associations of depressive symptoms or anxiety with IMT in the full sample or in any race/ethnic group.

Although in general we found that trends in the association between trait anger and carotid IMT were similar in most groups, the positive association between trait anger scores and carotid IMT was stronger in men than in women, and in whites than in other race/ethnic groups. Although we have no clear explanation for these differences, mean values of carotid IMT varied widely by gender and race/ethnicity in this study. For example, Chinese participants in the present study had the lowest carotid IMT and prevalence of cardiovascular risk factors (23–27), which may affect the ability to detect associations with trait anger. It is also possible that the presence of other risk factors modulates or overwhelms relatively small effects of anger in non-white race/ethnic groups. In addition the relevance of a particular psychosocial factor to health may vary depending on other features of the social context. For example other psychosocial factors may be more relevant to subclinical atherosclerosis in African Americans than in other groups (42). However, due to limited data on differential effects of psychosocial factors across race/ethnic groups, additional data is needed before firm conclusions about differential effect by race/ethnicity can be drawn.

Although we found a positive association of trait anger scores with carotid IMT, prior MESA analyses failed to detect an association of psychosocial factors with coronary

calcification, another measure of subclinical atherosclerosis (23). If differential association of psychosocial factors with atherosclerosis in different vascular beds is confirmed, further study of the reasons for these differences could shed light in the causal processes involved.

The strength of the present study is that we analyzed the associations of psychological factors with carotid artery IMT using population-based data, including various race/ethnic groups, with a relatively large numbers of participants. Most participants in previous studies examining the associations of psychological factors with carotid artery IMT were whites and the studies have varied widely in sample size (ranged from 200 to 3,781) (15–22). Other important strengths in MESA, include the state-of-the-art measurement techniques used for all assessments.

Cross-sectional studies of psychological factors and clinical outcomes may be biased because of reverse causation, ie that disease status could have adverse psychological consequences. However, in our analyses all participants were free of clinical cardiovascular disease. It is unlikely that asymptomatic IMT thickening could have affected psychological characteristics. Although significant positive associations of trait anger with carotid IMT were observed in this study, the magnitudes of the effects were small and the addition of psychological factors had very marginal effects on the variance explained. Therefore, it is not clear how relevant the observed differences are to overall risk of cardiovascular disease. However, because the associations of carotid artery wall thickness with incident of cardiovascular events appear to be approximately liner (8–11), small differences in IMT could affect risk at a population level. As a point of comparison, the difference in IMT associated with a one SD increase in trait anger was approximately similar to that observed for an increase of 3 years in age in our data.

Although we used well-validated questionnaires to measure psychological factors, the use of single assessment of psychological factors in this study could have resulted in misclassification of psychological variables biasing our results towards the null. Variations on the measurement properties of the scales by race/ethnicity and gender could also have affected our results, although in general measurement properties were acceptable in all groups; Cronbach’s alphas ranged from a low of 0.72 for depressive symptoms in Chinese persons and white men to a high of 0.84 for anger in Hispanic men (23). In addition, other measures of psychological distress may be more relevant than the ones we investigated, especially in some population subgroups.

In conclusion, our data provides some evidence that trait anger may be associated with subclinical carotid atherosclerosis, which increases the risk of cardiovascular disease. But the association was weak and this pattern was only observed in whites and to some extent in African Americans and Hispanics and was not present on Chinese. No associations were observed for depressive symptoms or anxiety. Further work is needed to confirm heterogeneous effects of anger and to better understand how and why other factors linked to gender or race/ethnicity may modulate these effects.

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Glossary

ANCOVA	analysis of covariance
BMI	body mass index
BP	blood pressure
CCA	common carotid artery
CES-D	the Center for Epidemiologic Studies Depression Scale
ECG	electrocardiogram
ICA	internal carotid artery
HPA	hypothalamo-pituitary-adrenal
IMT	intima-media thickness
MESA	the Multi-Ethnic Study of Atherosclerosis
MET	metabolic equivalent
STAXI	the Spielberger Trait Anger and Anxiety Inventory

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Table 1

Age, gender, race/ethnicity-adjusted means (SD) of common and internal carotid artery IMTs according to demographic and cardiovascular risk factors, MESA

Characteristics	N	Common carotid artery IMT (mm)	Internal carotid artery IMT (mm)
Gender			
Women	3,463	0.85 (0.18)	1.01 (0.59)
Men	3,098	0.89 (0.20)	1.14 (0.63)
Age-ethnicity adjusted overall *P*		<0.001	<0.001
Race/ethnicity			
White	2,541	0.86 (0.20)	1.12 (0.63)
African American	1,790	0.91 (0.19)	1.11 (0.61)
Hispanic	1,436	0.86 (0.19)	1.05 (0.59)
Chinese	794	0.82 (0.17)	0.87 (0.47)
Age-gender adjusted overall *P*		<0.001	<0.001
Age			
44–49	851	0.75 (0.15)	0.80 (0.27)
50–59	1,968	0.80 (0.15)	0.92 (0.45)
60–69	1,996	0.89 (0.17)	1.10 (0.59)
70–	1,746	0.98 (0.21)	1.35 (0.76)
Gender-ethnicity adjusted overall *P*		<0.001	<0.001
Hypertension *			
Yes	2,924	0.90 (0.21)	1.16 (0.70)
No	3,637	0.85 (0.17)	1.00 (0.49)
P		<0.001	<0.001
Antihypertensive medication			
Yes	2,155	0.89 (0.20)	1.16 (0.70)
No	4,406	0.86 (0.18)	1.03 (0.53)
P		<0.001	<0.001
Total cholesterol			
>=220 mg/dl	1,416	0.89 (0.21)	1.12 (0.70)
<220 mg/dl	5,145	0.86 (0.17)	1.06 (0.49)
P		<0.001	<0.001
Diabetes mellitus **			
Yes	900	0.91 (0.20)	1.22 (0.69)
No	5,661	0.86 (0.19)	1.05 (0.58)
P		<0.001	<0.001
Smoking			
Current	861	0.88 (0.19)	1.17 (0.60)
Never, former	5,700	0.87 (0.18)	1.06 (0.60)
P		0.27	<0.001
Body mass index			
>=30 kg/m^2	2,058	0.90 (0.21)	1.13 (0.70)

Characteristics	N	Common carotid artery IMT (mm)	Internal carotid artery IMT (mm)
<30 kg/m^2	4,503	0.86 (0.17)	1.04 (0.49)
P		<0.001	<0.001

*Systolic blood pressure>=140mmHg, diastolic blood pressure>=90mmHg, and/or use of antihypertensive medication.

**Fasting glucose>=126mmg/dL, and/or use of antidiabetic medication.

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Table 2

Age, gender, and race/ethnicity-adjusted means or prevalences of cardiovascular risk factors according to quartile of psychosocial variables, MESA

Characteristics	range	n	Age, year	Men, %	Whites/African Americans/Hispanics, %	Hypertension*, %	Diabetes Mellitus**, %	Total cholesterol, mmg/dL	Current smoking, %	Body mass index, mg/dL
Trait anger										
Q1	10–12	1,927	65	49	32/33/25	44	13	195	12	28.0
Q2	13–14	1,644	62	47	43/28/18	43	12	193	13	28.3
Q3	15–16	1,273	61	47	42/26/20	44	14	193	14	28.3
Q4	17–40	1,710	59	46	39/21/23	48	15	196	15	28.4
P for trend			<0.001	0.17	<0.001	0.007	0.02	0.17	<0.001	0.03
Trait anxiety										
Q1	10–12	1,700	64	55	35/34/22	43	14	193	12	28.3
Q2	13–15	1,736	62	51	39/28/22	43	13	196	12	28.2
Q3	16–18	1,376	61	44	39/25/22	44	13	194	14	28.0
Q4	19–37	1,738	61	39	42/22/23	49	14	194	15	28.4
P for trend			<0.001	<0.001	<0.001	<0.001	0.75	0.95	0.004	0.61
Depression										
Q1	0–2	1,772	63	54	40/28/18	43	13	194	11	27.9
Q2	3–5	1,506	62	52	42/27/18	44	13	193	12	28.3
Q3	6–10	1,645	62	47	38/30/21	44	13	195	14	28.3
Q4	11–53	1,638	62	36	35/25/31	47	16	194	16	28.4
P for trend			0.002	<0.001	0.12	0.02	0.02	0.89	<0.001	0.02

*Systolic blood pressure>=140mmHg, diastolic blood pressure>=90mmHg, and/or use of antihypertensive medication.

**Fasting glucose>=126mmg/dL, and/or use of antidiabetic medication.

Table 3

Age, gender, and race/ethnicity-adjusted and multivariable-adjusted mean differences in common and internal carotid artery IMTs and odds ratios for carotid plaque per one SD increase in psychological scores, MESA

Characteristics	Age, gender, and race-adjusted		Multivariable-adjusted*	
	Common carotid artery IMT (mm)	Internal carotid artery IMT (mm)	Common carotid artery IMT (mm)	Internal carotid artery IMT (mm)
Trait anger				
Mean difference per one SD increase** (95% CI)	0.014 (0.023, 0.025)	0.054 (0.017, 0.090)	0.011 (0.000, 0.022)	0.041 (0.004, 0.078)
P	0.01	0.004	0.06	0.03
OR*** for carotid plaque per one SD increase (95%CI)	1.27 (1.06, 1.52)		1.22 (1.01, 1.47)	
P	0.008		0.04	
Trait anxiety				
Mean difference per one SD increase** (95% CI)	0.010 (0.000, 0.021)	0.018 (−0.017, 0.053)	0.010 (−0.001, 0.021)	0.006 (−0.003, 0.042)
P	0.06	0.32	0.07	0.73
OR*** for carotid plaque per one SD increase (95%CI)	1.03 (0.87, 1.22)		0.95 (0.79, 1.14)	
P	0.75		0.60	
Depression				
Mean difference per one SD increase** (95% CI)	0.001 (−0.003, 0.005)	0.006 (−0.008, 0.021)	−0.001 (−0.006, 0.003)	−0.001 (−0.016, 0.014)
P	0.80	0.40	0.53	0.91
OR*** for carotid plaque per one SD increase (95%CI)	1.02 (0.94, 1.10)		0.97 (0.89, 1.06)	
P	0.64		0.49	

* Adjusted for age, ethnicity, systolic blood pressure, use of antihypertensive medication, smoking status, body mass index, diabetes mellitus, total/HDL cholesterol ratio, education, and physical activity.

** One SD: depression, 8 points; trait anger, 4 points; trait anxiety, 5 points.

*** Odds ratio.

Table 4

Age and race/ethnicity-adjusted and multivariable-adjusted * mean differences in common and internal carotid artery IMTs and odds ratios for carotid plaque per one SD increase in trait anger, stratified by gender, MESA

Characteristics	Age and race-adjusted		Multivariable-adjusted*	
	Common carotid artery IMT (mm)	Internal carotid artery IMT (mm)	Common carotid artery IMT (mm)	Internal carotid artery IMT (mm)
Men				
Mean difference per one SD increase ** (95% CI)	0.021 (0.004, 0.038)	0.075 (0.020, 0.130)	0.016 (−0.001, 0.032)	0.054 (−0.002, 0.110)
P	0.01	0.008	0.07	0.06
OR *** for carotid plaque per one SD increase (95%CI)	1.44 (1.13, 1.84)		1.36 (1.05, 1.75)	
P	0.003		0.02	
Women				
Mean difference per one SD increase ** (95% CI)	0.008 (−0.007, 0.021)	0.034 (−0.015, 0.082)	0.006 (−0.009, 0.021)	0.027 (−0.022, 0.076)
P	0.29	0.17	0.42	0.29
OR *** for carotid plaque per one SD increase (95%CI)	1.10 (0.84, 1.42)		1.07 (0.81, 1.41)	
P	0.49		0.64	

* Adjusted for age, ethnicity, systolic blood pressure, use of antihypertensive medication, smoking status, body mass index, diabetes mellitus, total/HDL cholesterol ratio, education, and physical activity.

** One SD: 4 points of trait anger score.

*** Odds ratio.

Table 5

Age and gender-adjusted and multivariable-adjusted* mean differences in carotid artery IMT and odds ratios for carotid plaque per one SD increase in trait anger, stratified by race/ethnicity, MESA

Characteristics	Age and gender-adjusted		Multivariable-adjusted*	
	Common carotid artery IMT (mm)	Internal carotid artery IMT (mm)	Common carotid artery IMT (mm)	Internal carotid artery IMT (mm)
White				
Mean difference per one SD increase** (95% CI)	0.025 (0.006, 0.044)	0.100 (0.036, 0.164)	0.020 (0.001, 0.038)	0.096 (0.033, 0.160)
P	0.009	0.002	0.04	0.003
OR for carotid plaque (95%CI)***	1.51 (1.14, 2.01)		1.53 (1.13, 2.08)	
P	0.004		0.007	
African American				
Mean difference per one SD increase** (95% CI)	0.003 (−0.020, 0.025)	0.033 (−0.042, 0.107)	−0.002 (−0.024, 0.021)	0.002 (−0.074, 0.078)
P	0.82	0.55	0.88	0.96
OR for carotid plaque (95%CI)***	1.25 (0.90, 1.75)		1.14 (0.78, 1.67)	
P	0.39		0.50	
Hispanic				
Mean difference per one SD increase** (95% CI)	0.015 (−0.006, 0.036)	0.034 (−0.034, 0.102)	0.014 (−0.007, 0.036)	0.026 (−0.045, 0.097)
P	0.15	0.32	0.19	0.47
OR for carotid plaque (95%CI)***	1.25 (0.90, 1.74)		1.14 (0.80, 1.62)	
P	0.18		0.47	
Chinese				
Mean difference per one SD increase** (95% CI)	−0.003 (−0.033, 0.028)	−0.029 (−0.115, 0.058)	−0.007 (−0.038, 0.023)	−0.003 (−0.120, 0.056)
P	0.87	0.52	0.64	0.48
OR for carotid plaque (95%CI)***	0.74 (0.38, 1.42)		0.75 (0.38, 1.50)	
P	0.36		0.42	

*Adjusted for age, gender, systolic blood pressure, use of antihypertensive medication, smoking status, body mass index, diabetes mellitus, total/HDL cholesterol ratio, education, and physical activity.

** One SD: 4 points of trait anger score.

*** Odds ratio for carotid plaque per one SD increase (95%CI).