Cynical hostility and carotid atherosclerosis in African American and white women: The Study of Women's Health Across the Nation (SWAN) Heart Study

Susan A. Everson-Rose, PhD, MPH,[a,b,c] Tené T. Lewis, PhD,[a] Kelly Karavolos, MA,[a] Karen A. Matthews, PhD,[d] Kim Sutton-Tyrrell, DrPH,[d] and Lynda H. Powell, PhD[a,c] ***Chicago, IL; and Pittsburgh, PA***

Background Hostility is associated with increased cardiovascular disease mortality and morbidity and may be related to subclinical atherosclerosis; less is known about this association among women and minority groups.

Methods We examined the association between hostility and intimal-medial thickening (IMT) as well as presence/absence of plaque in the carotid arteries in middle-aged white and African American women. Hostility was measured by a 13-item questionnaire and IMT and plaque were assessed by B-mode ultrasonography in 589 participants from the Chicago and Pittsburgh sites of the SWAN.

Results In age- and site-adjusted models, each 1-point increment in hostility score predicted a significant 0.0057-mm higher mean IMT ($P < .0001$) and 0.0081-mm higher maximum IMT ($P < .0001$)—effects that were identical in magnitude to each 1-year increment in age. Adjustments for race, education, body mass index, resting systolic blood pressure, and smoking diminished these associations, but they remained significant ($P \leq .01$). With hostility scores modeled in approximate tertiles, high scorers (≥6) had greater mean ($P = .0005$) and maximum ($P = .0004$) IMT than low scorers (0 or 1); moderate and low scorers did not differ (age-adjusted mean values for low, moderate, and high scorers were 0.657, 0.662, and 0.694 mm, respectively, for mean IMT; those for low, moderate, and high scorers were 0.855, 0.860, and 0.906 mm, respectively, for maximum IMT). Hostility was unrelated to presence of plaque and did not interact with race, education, smoking, and body mass index.

Conclusions Hostility is related to small but significantly greater subclinical atherosclerosis in middle-aged women; this association is not explained by traditional risk factors. (Am Heart J 2006;152:982.e7-e13.)

Historical anecdotes and clinical evidence have long suggested that the experience and expression of hostility—characterized by a suspicious and mistrustful attitude and considered to be an enduring trait—have important cardiovascular consequences. Empirical support for this hypothesis has grown in the past 25 years, although the literature and quality of studies are mixed (see Reference [1] for a review). A meta-analysis of 45 studies published in 1996 concluded that hostility was independently related to coronary heart disease and all-cause mortality.[2] Most studies published in the intervening 10 years have been positive, linking hostility to increased risk for hypertension and cardiovascular disease (CVD) morbidity and mortality.[3-6]

Research suggests that hostility may be related to subclinical markers of CVD. Among middle-aged Finnish men, high hostility scores were associated with greater 2-year progression of carotid atherosclerosis.[7] High levels of hostility, anger-in, and trait anger were related to severity and extent of carotid atherosclerosis 10 years later in healthy postmenopausal women.[8] In the National Heart, Lung, and Blood Institute Family Heart Study, high hostility was associated with increased odds of carotid lesions among women with high familial risk for coronary heart disease.[9] Hostile persons had greater odds of coronary calcification among a subsample of CARDIA

From the [a]Department of Preventive Medicine, Rush University Medical Center, Chicago, IL, [b]Institute for Healthy Aging, Rush University Medical Center, Chicago, IL, [c]Department of Behavioral Sciences, Rush University Medical Center, Chicago, IL and [d]Department of Epidemiology, University of Pittsburgh, Pittsburgh, PA.

The SWAN is supported by the National Institutes of Health, Department of Health and Human Services, through the National Institute on Aging, the National Institute of Nursing Research, and the National Institutes of Health Office of Research on Women's Health; the SWAN Heart Study is supported by the National Heart, Lung, and Blood Institute (grant nos. NR004061; AG012505, AG012535, AG012531, AG012539, AG012546, AG012553, AG012554, and AG012495; as well as HL065581 and HL065591). The Chicago site of the SWAN Heart Study is supported by the Charles J. and Margaret Roberts Trust.

Submitted June 21, 2006; accepted August 3, 2006.

Reprint requests: Susan A. Everson-Rose, PhD, MPH, Department of Preventive Medicine, Rush University Medical Center, 1700 West Van Buren St., Suite 470, Chicago, IL 60612-2391.

E-mail: susan_everson@rush.edu

0002-8703/$ - see front matter

doi:10.1016/j.ahj.2006.08.010

Study participants,[10] although power was limited to examine associations by sex and race. In a sample of untreated hypertensive men, anger-related personality traits were associated with greater carotid artery atherosclerosis.[11] In contrast, hostility was not associated with coronary calcification in a study on active-duty army personnel.[12] Nonetheless, because most studies on hostility and subclinical CVD have included predominantly white samples and several have included only male subjects, much remains to be learned about these associations in minority populations and among women.[1]

We examined the relationship between hostility and carotid artery atherosclerosis in a biracial sample of middle-aged women, hypothesizing that women reporting greater hostility would have greater carotid atherosclerosis than their less-hostile counterparts. Research suggests that certain groups (eg, members of ethnic minority groups or those of lower socioeconomic status) may be more susceptible to the adverse effects of psychosocial factors on CVD risk as a result of greater exposure to stress or other environmental factors.[1,13,14] Other work indicates that psychosocial risk factors may interact with established CVD risk factors or exacerbate risk in populations with prevalent CVD symptoms or conditions.[15,16] Thus, we also examined whether the effect of hostility on carotid artery atherosclerosis varied by race, education, smoking status, and body mass index (BMI).

Methods

Study population

The sample included 589 women (63.8% whites and 36.2% African Americans) from the Chicago and Pittsburgh sites of the SWAN who participated in an ancillary study examining the natural history of subclinical atherosclerosis as women transition through menopause (SWAN Heart Study). The SWAN, an ongoing community-based longitudinal study on menopausal transition conducted at 7 clinical sites (Chicago, IL; Pittsburgh, PA; Boston, MA; Detroit, MI; Newark, NJ; Oakland, CA; and Los Angeles, CA), enrolled 3302 women in 1996-1997, with annual examinations ongoing. Eligibility criteria were being between 42 and 52 years old, having an intact uterus, having at least one ovary, having reported menstrual bleeding and no use of reproductive hormones that affect ovarian or pituitary function within the past 3 months, not being currently pregnant or breast-feeding, and self-identifying as part of 1 of 5 prespecified racial/ethnic groups depending on site: non-Hispanic white (all sites); African American (Chicago, Pittsburgh, Boston, and Detroit); Hispanic (Newark); Chinese or Chinese-American (Oakland); and Japanese or Japanese-American (Los Angeles). Recruitment and study design details have been reported.[17]

Women enrolled into the SWAN Heart Study from 2001 to 2003, coincident with their 4th or 5th annual SWAN visit for 93.8% of participants; the rest of the subjects completed their first SWAN Heart Study visit coincident with their 6th or 7th annual SWAN visit. Women were eligible for the SWAN Heart Study if they had at least one ovary, had no evidence of clinical atherosclerosis (myocardial infarction, angina, intermittent claudication, cerebral ischemia, or revascularization), and did not currently use hormones or medications for hypertension, diabetes, or arrhythmias. The SWAN Heart Study enrolled 608 women (382 whites and 226 African Americans); 19 women had missing hostility data and were excluded from all analyses. This report is from the first (baseline) assessment of the SWAN Heart Study; the research protocol was approved by the institutional review boards of both sites and all women provided written informed consent.

Procedures

All participants completed a standard protocol upon entry into the parent SWAN study (1996-1997) and annually thereafter, including self- and interviewer-administered questionnaires, laboratory measures based on fasting blood and urine specimens, anthropometric measures, and blood pressure readings. Questionnaires assessed socioeconomic factors, medical history, medication use, psychosocial and lifestyle characteristics, behaviors, menstrual status, symptoms, quality of life, and sexual functioning. Laboratory measures assessed reproductive hormones, glucose and insulin levels, clotting factors, and lipid and lipoprotein profiles. Full details of the SWAN protocol are provided elsewhere.[17] Data collected as part of the annual SWAN assessment coincident with recruitment to the SWAN Heart Study provided information on the covariates of interest for the present analyses.

Measurement of hostility

Hostility was assessed only at the baseline SWAN visit (in 1996-1997) using 13 items derived from the Cook-Medley Hostility Scale,[18] which measures cynical attitudes and hostile feelings and behaviors rather than overt expressions of anger and aggression. Prior research characterized this 13-item subset as a measure of cynicism and found that high scores were associated with greater CVD morbidity and mortality.[19,20] All items have a true/false response format, with 1 point assigned for each "true" response. A summary score is created by summing across the 13 items; higher scores indicate greater hostility (range 0-13). Cronbach's α for the 13-item scale was .79, indicating good internal consistency. Hostility was assessed only once in the SWAN because this personality trait has been found to be relatively stable in both middle and later life[21-23]; for example, the test-retest reliability of the Cook-Medley Hostility Scale was 0.88 over 3 years in a sample of postmenopausal women.[23]

B-mode ultrasonography of the carotid arteries

Intimal-medial thickening (IMT) of the right and left carotid arteries was assessed by B-mode ultrasound using a Hewlett Packard (Hewlett Packard, Andover, MA) 5500 scanner for the Chicago site and a Toshiba (Toshiba American Medical Systems, Tustin, CA) SSA-270A scanner for the Pittsburgh site. These scanners produce images of comparable quality. B-mode images were obtained from 8 locations — 4 locations each from the left and right carotid arteries: the near and far walls of the distal common carotid artery (1 cm proximal to the carotid bulb), the far walls of the carotid bulb (the point in which the near and far walls of the common carotid are no longer parallel, extending to the flow divider), and the internal carotid artery

Table I. Baseline characteristics of the participants

	All participants (N = 589)	Whites (n = 376)	African Americans (n = 213)	*P*
Age [y, mean (SD)]	50.3 (2.8)	50.3 (2.9)	50.3 (2.8)	NS
Hostility [mean (SD)]	3.57 (2.9)	2.84 (2.4)	4.85 (3.1)	<.0001
Low (%)	29.5	35.9	18.3	
Moderate (%)	46.0	48.9	40.85	
High (%)	24.5	15.2	40.85	
Education (%)				
High school or less	15.3	14.9	16.0	<.02
Some college	32.1	27.9	39.4	
College degree	21.2	22.6	18.8	
Graduate school	31.4	34.6	25.8	
BMI [kg/m^2, mean (SD)]	29.2 (6.3)	28.2 (5.8)	31.1 (6.7)	<.0001
SBP [mm Hg, mean (SD)]	119.6 (16.7)	115.4 (14.4)	127.0 (18.0)	<.0001
Smokers (%)	15.3	14.6	16.4	NS
IMT [mm, mean (SD)]*				
Mean	0.668 (0.10)	0.656 (0.09)	0.689 (0.10)	<.0001
Maximum	0.870 (0.13)	0.854 (0.12)	0.896 (0.13)	.0002
Plaque prevalence (%)†	15.0	14.9	15.3	NS

P values are from *t* tests or χ^2 tests comparing whites and African Americans. *NS*, Nonsignificant.
*The number for both mean and maximum IMT was 576.
†The number for plaque prevalence was 582.

(from the flow divider to 1 cm distal to this point). Intimal-medial thickening measures were obtained by electronically tracing the lumen-intima interface and the media-adventitia interface across a 1-cm segment; one measurement was generated for each pixel over the area, for a total of approximately 140 measures for each segment. The average, standard deviation, minimum, and maximum values for these measures were recorded for all 8 locations. For analyses, the mean value of the average readings and that of the maximum readings at all 8 locations were used. Presence and extent of plaque also were evaluated in each segment and summarized as the presence or absence of any plaque in either the right or the left carotid artery. Readers were recertified annually against the same set of scans to guard against reader drift. All readings were conducted at the Ultrasound Research Laboratory of the Department of Epidemiology at the University of Pittsburgh under the direction of Dr Sutton-Tyrrell. Reproducibility of IMT measures was excellent. Replicate readings were performed on 20 scans, with an intraclass correlation of 0.98 for IMT values. Prior work at the Ultrasound Research Laboratory documented highly reproducible measures of plaque, with intraclass correlations ranging from 0.86 to 0.93.[24,25] Nearly all (95%) participants completed the carotid ultrasound assessments within 5 months (average 7 weeks) of their annual SWAN assessment.

Measurement of covariates

Age was self-reported and modeled continuously. Women self-identified themselves as African American or white (referent). Education was modeled categorically (high school or less, some college, college degree, or graduate school [referent]). Systolic blood pressure (SBP), the average of 2 manual readings obtained after a 5-minute rest in a seated position, was modeled continuously. Body mass index, calculated as weight in kilograms divided by height in meters squared, was modeled continuously. Weight and height were measured to the nearest 0.01 kg and 0.01 cm, respectively, using standard protocols and equipment. Current smoking status was a binary variable, with nonsmokers as the referent.

Data analyses

We characterized our sample on age, hostility scores, education, BMI, SBP, smoking status, mean and maximum IMT, and prevalence of plaque with descriptive statistics and used *t* tests and χ^2 tests to compare whites and African Americans on these characteristics. Linear regression models estimated the associations between hostility and mean as well as maximum IMT. Logistic regression models evaluated the association between hostility and presence of plaque. Hostility was modeled continuously and categorically in approximate tertiles. Initial models were age adjusted; additional models included covariates for age, race, and education (model 2) as well as BMI, SBP, and smoking status (model 3). All models included study site as a covariate. Analyses were conducted in PC-SAS (version 8.2, SAS Institute, Cary, NC) using a GLM, LOGISTIC, UNIVARIATE, or FREQ procedure. Because of missing or invalid data on the outcomes, the numbers for analyses of mean and maximum IMT (n = 557) and plaque (n = 560) varied slightly.

Results

Participant characteristics

Table I shows the participants' characteristics, measured at the SWAN Heart Study baseline (hostility scores were assessed at the parent SWAN baseline), for all women and separately by race. Women were approximately 50 years old; most were well educated; 15%

Table II. Cynical hostility and mean as well as maximum carotid artery IMT: SWAN Heart Study

	Model 1*				Model 2†				Model 3‡			
	Estimate	**SE**	***t***	***P***	**Estimate**	**SE**	***t***	***P***	**Estimate**	**SE**	***t***	***P***
Mean IMT												
Cynical hostility	0.0057	0.001	4.26	<.0001	0.0038	0.001	2.70	.007	0.0033	0.001	2.46	.014
Age	0.0057	0.001	4.25	<.0001	0.0057	0.001	4.24	<.0001	0.0041	0.001	3.12	.002
African American					0.0307	0.008	3.64	.0003	0.0145	0.008	1.75	.081
White					Referent				Referent			
Education												
High school or less					0.0142	0.012	1.18	.24	0.0140	0.012	1.20	.23
Some college					0.0068	0.009	0.72	.47	−0.0008	0.009	−0.09	.93
College degree					0.0028	0.011	0.26	.79	0.0021	0.01	0.21	.83
Graduate school					Referent				Referent			
BMI									0.0029	0.0006	4.65	<.0001
SBP									0.0010	0.0002	4.11	<.0001
Current smoker									−0.0147	0.01	−1.45	.15
Nonsmoker									Referent			
Maximum IMT												
Cynical hostility	0.0081	0.002	4.40	<.0001	0.0061	0.002	3.13	.002	0.0054	0.002	2.91	.004
Age	0.0080	0.002	4.38	<.0001	0.0080	0.002	4.34	<.0001	0.0061	0.002	3.37	.0008
African American					0.0318	0.012	2.74	.006	0.0116	0.011	1.00	.32
White					Referent				Referent			
Education												
High school or less					0.0220	0.017	1.32	.19	0.0207	0.016	1.28	.20
Some college					0.0056	0.013	0.43	.67	−0.0045	0.013	−0.35	.72
College degree					0.0039	0.015	0.26	.79	0.0029	0.014	0.20	.84
Graduate school					Referent				Referent			
BMI									0.0039	0.0009	4.43	<.0001
SBP									0.0012	0.0003	3.52	.0005
Current smoker									−0.0111	0.014	−0.78	.43
Nonsmoker									Referent			

Results are from linear regression models, with hostility scores modeled continuously; all models also included a covariate for site.
*For mean IMT, $R^2 = 0.11$; for maximum IMT, $R^2 = 0.07$.
†For mean IMT, $R^2 = 0.14$; for maximum IMT, $R^2 = 0.09$.
‡For mean IMT, $R^2 = 0.22$; for maximum IMT, $R^2 = 0.17$.

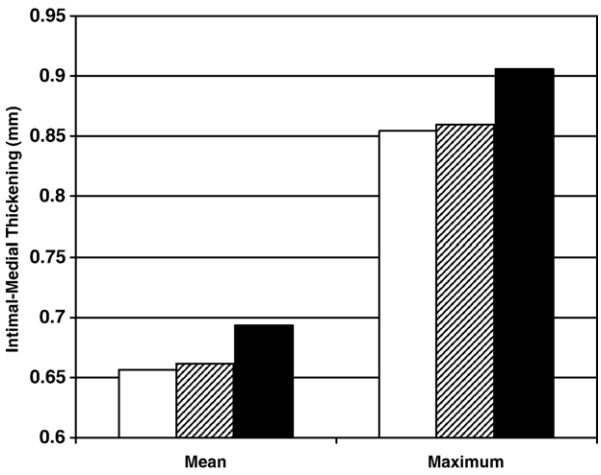
currently smoked; their average SBP was 119 mm Hg; and they tended to be overweight, with a mean BMI >29 kg/m^2. Compared with whites, African Americans were less educated ($P < .02$) and had significantly higher hostility scores and BMI, resting SBP, as well as mean and maximum IMT values (all $P < .0001$).

Associations between hostility and IMT

Table II shows findings from 3 sets of linear regression models assessing the relation between hostility and mean as well as maximum IMT. In age- and site-adjusted models, each 1-point increment in hostility score predicted a significant 0.0057-mm higher mean IMT and 0.0081-mm higher maximum IMT. For both IMT outcomes, the effect of hostility was identical in magnitude to each 1-year increment in age (see columns under "Model 1"). With race and education included as covariates ("Model 2"), the effect of hostility decreased but remained significant for mean and maximum IMT. Similar results were noted with further adjustments for BMI, SBP, and current smoking status ("Model 3"). Age remained a significant covariate in all models. Race, although significant in model 2, became nonsignificant in model 3 after adjustments were made for BMI, SBP, and smoking as a result of the highly significant effects of BMI and SBP on the IMT outcomes.

With hostility scores modeled categorically in approximate tertiles in which the bottom and top categories represented a 2-SD difference in hostility scores, women with a hostility score ≥6 (black bars in Figure 1) had significantly greater mean ($P = .0005$) and maximum ($P = .0004$) IMT than women in the bottom third of the distribution (ie, a hostility score of 1 or 0) (white bars in Figure 1). Women with hostility scores in the middle range of 2 to 5 (hatched bars in Figure 1) did not differ from those with low hostility scores. Average age- and site-adjusted IMT values for low, middle, and high scorers, respectively, were 0.657, 0.662, and 0.694 mm for mean IMT and 0.855, 0.860, and 0.906 mm for maximum IMT. Additional adjustments for race, education, BMI, SBP, and

Figure 1



Mean and maximum IMT by approximate tertiles of cynical hostility scores. White bars represent women with low hostility scores (0-1); hatched bars, women with moderate hostility scores (2-5); black bars, women with high hostility scores (≥6).

smoking status did little to diminish the associations (data not shown).

Associations between hostility and plaque

Hostility scores were not related to presence of plaque (OR 1.02, 95% CI 0.94-1.10) in an age- and site-adjusted logistic regression model. However, only 15% of our sample had measurable plaque in their carotid arteries (Table II), limiting our ability to adequately test this hypothesis.

Do associations vary as a function of race, education, smoking status, or BMI?

We observed no significant interaction between hostility scores (modeled continuously) and race, education, smoking status, and BMI for either the mean or the maximum IMT (all $P > .4$). Similarly, hostility did not interact with race, education, smoking, and BMI with presence of plaque as the outcome (all $P > .2$).

Discussion

This study supports the hypothesis that higher levels of hostility are associated with greater carotid IMT in middle-aged women. Associations were consistent for mean and maximum IMT and remained significant in multivariable models. Effect sizes for hostility were small but similar in magnitude to effect sizes observed for age and BMI (Table II), both of which were highly significant covariates. Several studies have shown that carotid artery IMT independently predicts incident CVD and stroke,[26-28] with small and incremental differences in IMT associated with increased risk for coronary events.[29]

African American women had higher mean hostility scores and higher (unadjusted) mean levels of IMT than white women in our sample. The association between hostility and IMT did not differ by race, suggesting that hostility likely plays a similar role in the atherosclerotic disease process for African American and white women. However, this conclusion must be tempered by the fact that almost no published study has had an adequate sample size or enough power to reliably assess the effects of hostility on subclinical CVD by race or by sex; thus, our findings need to be replicated.

We did not observe an interaction between hostility and education level, BMI, and smoking status in predicting IMT or plaque. Less education, overweight and obesity, and cigarette smoking confer increased cardiovascular risk[30]; prior research has found that each of these factors is related to higher hostility levels.[31,32] Also, the effects of psychosocial factors—including hostility—on CVD risk may be stronger in populations deemed vulnerable because of their greater exposure to stress or other environmental factors[1,13,14] or those at risk because of the presence of established coronary risk factors.[15,16] Therefore, we reasoned that IMT levels would be greater among women with at least one CVD risk factor and who reported higher hostility scores. Our data did not support this. As noted, the effects of hostility on IMT are comparable in magnitude with the effects of age and BMI. Perhaps, somewhat surprisingly, race, education, and smoking status were weak or nonsignificant correlates of IMT in our adjusted models. This pattern of findings suggests that hostility has a consistent independent effect on subclinical CVD even among relatively healthy middle-aged women—effects that are stronger than some traditional risk factors.

The extent of subclinical atherosclerosis in our sample likely is limited by the relatively young age of the participants. Perhaps larger effect sizes and interactions between hostility and CVD risk factors would be observed in older samples with more diseases. The average mean IMT in our sample is lower than that reported in a study on primarily middle-aged white women using a similar IMT assessment protocol (0.668 vs 0.76 mm),[8] although participants in that study were, on average, 7 years older than our participants. Prevalence of any plaque (15%) was somewhat lower in this study than as previously reported from other studies on middle-age women[33,34] and may have contributed to our finding of a lack of association between hostility and plaque. However, plaque prevalence was related to history of recurrent depression in the participants from the Pittsburgh SWAN site in previous analyses.[35]

What mechanisms may mediate the influence of hostility on subclinical CVD? Higher levels of hostility are related to poor health behaviors, socioeconomic disad-

vantage, greater stress, lower social support, higher levels of blood pressure, poor lipid profiles, platelet reactivity, activation of the hypothalamic-pituitary-adrenal axis, and greater prevalence of the metabolic syndrome (MBS).[13,31,32,36,37] Studies have found that the effects of hostility on CVD are mediated through behavioral risk factors[3] or the MBS or its components[38]; others have shown that hostility increases CVD risk independent of biologic, behavioral, or social factors.[39] We found that the effects of hostility were independent of age, race, education, BMI, blood pressure, and smoking. We also examined whether prevalence of the MBS influenced our findings in a subsequent analysis on 70% of our sample for whom we had available data on prevalence of the MBS (data not shown); 18.4% of the participants met MBS criteria,[40] but the MBS was not associated with hostility and did not attenuate the hostility-IMT association. We did not have adequate data to explore whether individual MBS components or other biologic factors (eg, clotting factors) mediate the observed associations. Because hostility likely operates through multiple pathways to influence CVD risk, further research that more fully explores potential behavioral, psychosocial, and biologic mechanisms is needed.

This cross-sectional study does not provide data on atherosclerotic progression; it is important to determine whether hostility influences rates of progression. Research show that IMT values increase by 0.01 to 0.03 mm per year[29] and that the rate of progression is significantly influenced by established coronary risk factors.[41,42] Data from the Kuopio Study show that psychosocial factors, including hostility, hopelessness, and stress-induced reactivity, adversely influence IMT progression in middle-aged white men.[7,43-45] It remains to be seen whether these same factors contribute to progression of IMT in women.

In summary, the SWAN Heart Study provides evidence that middle-aged African American and white women with higher reported levels of hostility have greater carotid artery IMT, an association that is independent of important CVD risk factors. Mechanisms underlying the observed relationships remain to be elucidated, and further research examining the impact of personality factors such as hostility on atherosclerotic progression is warranted.

We thank the study staff at each site and all the women who participated in the SWAN.

References

1. Everson-Rose SA, Lewis TT. Psychosocial factors and cardiovascular diseases. Annu Rev Public Health 2005;26:469-500.
2. Miller TQ, Smith TW, Turner CW, et al. A meta-analytic review of research on hostility and physical health. Psychol Bull 1996;119:322-48.
3. Everson SA, Kauhanen J, Kaplan GA, et al. Hostility and increased risk of mortality and acute myocardial infarction: the mediating role of behavioral risk factors. Am J Epidemiol 1997;146:142-52.
4. Niaura R, Todaro JF, Stroud L, et al. Hostility, the metabolic syndrome, and incident coronary heart disease. Health Psychol 2002;21:588-93.
5. Yan LL, Liu K, Matthews KA, et al. Psychosocial factors and risk of hypertension: the Coronary Artery Risk Development in Young Adults (CARDIA) Study. JAMA 2003;290:2138-48.
6. Matthews KA, Gump BB, Harris KF, et al. Hostile behaviors predict cardiovascular mortality among men enrolled in the Multiple Risk Factor Intervention Trial. Circulation 2004;109:66-70.
7. Julkunen J, Salonen R, Kaplan GA, et al. Hostility and the progression of carotid atherosclerosis. Psychosom Med 1994;56:519-25.
8. Matthews KA, Owens JF, Kuller LH, et al. Are hostility and anxiety associated with carotid atherosclerosis in healthy postmenopausal women? Psychosom Med 1998;60:633-8.
9. Knox SS, Adelman A, Ellison RC, et al. Hostility, social support, and carotid artery atherosclerosis in the National Heart, Lung, and Blood Institute Family Heart Study. Am J Cardiol 2000;86:1086-9.
10. Iribarren C, Sidney S, Bild DE, et al. Association of hostility with coronary artery calcification in young adults—the CARDIA Study. JAMA 2000;283:2546-51.
11. Bleil ME, McCaffery JM, Muldoon MF, et al. Anger-related personality traits and carotid artery atherosclerosis in untreated hypertensive men. Psychosom Med 2004;66:633-9.
12. O'Malley PG, Jones DL, Feuerstein IM, et al. Lack of correlation between psychological factors and subclinical coronary artery disease. N Engl J Med 2000;343:1298-304.
13. Smith TW, Glazer K, Ruiz JM, et al. Hostility, anger, aggressiveness, and coronary heart disease: an interpersonal perspective on personality, emotion, and health. J Pers 2004;72:1217-70.
14. Gallo LC, Bogart LM, Vranceanu AM, et al. Socioeconomic status, resources, psychological experiences, and emotional responses: a test of the reserve capacity model. J Pers Soc Psychol 2005;88:386-99.
15. Barefoot JC, Patterson JC, Haney TL, et al. Hostility in asymptomatic men with angiographically confirmed coronary artery disease. Am J Cardiol 1994;74:439-42.
16. Olson MB, Krantz DS, Kelsey SF, et al. Hostility scores are associated with increased risk of cardiovascular events in women undergoing coronary angiography: a report from the NHLBI-sponsored WISE Study. Psychosom Med 2005;67:546-52.
17. Sowers MF, Crawford S, Sternfeld B, et al. SWAN: a multicenter, multiethnic, community-based cohort study of women and the menopausal transition. In: Lobo RA, Kelsey J, Marcus R, editors. Menopause: biology and pathobiology. San Diego: Academic Press; 2000. p. 175-88.
18. Cook WW, Medley DM. Proposed hostility and pharisaic-virtue scales for the MMPI. J Appl Psychol 1954;38:414-8.
19. Barefoot JC, Dodge KA, Peterson BL, et al. The Cook-Medley Hostility Scale: item content and ability to predict survival. Psychosom Med 1989;51:46-57.
20. Barefoot J, Larsen S, von der Leith L, et al. Hostility, incidence of acute myocardial infarction, and mortality in a sample of older Danish men and women. Am J Epidemiol 1995;142:477-84.
21. Shekelle RB, Gale M, Ostfeld AM, et al. Hostility, risk of coronary heart disease, and mortality. Psychosom Med 1983;45:109-14.
22. Barefoot JC, Beckham JC, Haney TL, et al. Age differences in hostility among middle-aged and older adults. Psychol Aging 1993;8:3-9.

23. Raikkonen K, Matthews KA, Kuller LH. Trajectory of psychological risk and incident hypertension in middle-aged women. Hypertension 2001;38:798-802.
24. Sutton-Tyrrell K, Wolfson Jr SK, Thompson T, et al. Measurement variability in duplex scan assessment of carotid atherosclerosis. Stroke 1992;23:215-20.
25. Thompson T, Sutton-Tyrrell K, Wildman R. Continuous quality assessment programs can improve carotid duplex scan quality. J Vasc Technol 2001;25:33-9.
26. Salonen R, Salonen JT. Intima-media changes in a population study: KIHD. In: Boccalon H, editor. Amsterdam: Elsevier Science Publishers; 1993. p. 301-4.
27. Bots ML, Hoes AW, Koudstaal PJ, et al. Common carotid intima-media thickness and risk of stroke and myocardial infarction: the Rotterdam Study. Circulation 1997;96:1432-7.
28. Touboul PJ, Labreuche J, Vicaut E, et al. Carotid intima-media thickness, plaques, and Framingham risk score as independent determinants of stroke risk. Stroke 2005;36:1741-5.
29. O'Leary DH, Polak JF. Intima-media thickness: a tool for atherosclerosis imaging and event prediction. Am J Cardiol 2002;90:18L-21L.
30. Thom T, Haase N, Rosamond W, et al. Heart disease and stroke statistics—2006 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2006;113:e85-e151.
31. Siegler IC, Peterson BL, Barefoot JC, et al. Hostility during late adolescence predicts coronary risk factors at mid-life. Am J Epidemiol 1992;136:146-54.
32. Scherwitz LW, Perkins LL, Chesney MA, et al. Hostility and health behaviors in young adults: the CARDIA Study. Coronary Artery Risk Development in Young Adults Study. Am J Epidemiol 1992; 136:136-45.
33. Fabris F, Zanocchi M, Bo M, et al. Carotid plaque, aging, and risk factors. A study of 457 subjects. Stroke 1994;25:1133-40.
34. Sutton-Tyrrell K, Lassila HC, Meilahn E, et al. Carotid atherosclerosis in premenopausal and postmenopausal women and its association with risk factors measured after menopause. Stroke 1998;29:1116-21.
35. Jones DJ, Bromberger JT, Sutton-Tyrrell K, et al. Lifetime history of depression and carotid atherosclerosis in middle-aged women. Arch Gen Psychiatry 2003;60:153-60.
36. Barefoot JC, Peterson BL, Dahlstrom WG, et al. Hostility patterns and health implications: correlates of Cook-Medley Hostility Scale scores in a national survey. Health Psychol 1991;10:18-24.
37. Markovitz JH. Hostility is associated with increased platelet activation in coronary heart disease. Psychosom Med 1998;60: 586-91.
38. Nelson TL, Palmer RF, Pedersen NL. The metabolic syndrome mediates the relationship between cynical hostility and cardiovascular disease. Exp Aging Res 2004;30:163-77.
39. Chaput LA, Adams SH, Simon JA, et al. Hostility predicts recurrent events among postmenopausal women with coronary heart disease. Am J Epidemiol 2002;156:1092-9.
40. Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults. Executive summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA 2001;285:2486-97.
41. Howard G, Wagenknecht LE, Burke GL, et al. Cigarette smoking and progression of atherosclerosis: the Atherosclerosis Risk in Communities (ARIC) Study. JAMA 1998;279:119-24.
42. Chambless LE, Folsom AR, Davis V, et al. Risk factors for progression of common carotid atherosclerosis: the Atherosclerosis Risk in Communities Study, 1987-1998. Am J Epidemiol 2002; 155:38-47.
43. Everson SA, Kaplan GA, Goldberg DE, et al. Hopelessness and 4-year progression of carotid atherosclerosis. The Kuopio Ischemic Heart Disease Risk Factor Study. Arterioscler Thromb Vasc Biol 1997;17:1490-5.
44. Everson SA, Lynch JW, Chesney MA, et al. Interaction of workplace demands and cardiovascular reactivity in progression of carotid atherosclerosis: population based study. BMJ 1997;314:553-8.
45. Lynch JW, Everson SA, Kaplan GA, et al. Does low socioeconomic status potentiate the effects of heightened cardiovascular responses to stress on the progression of carotid atherosclerosis? Am J Public Health 1998;88:389-94.