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Trait Anger, Cynical Hostility and Inflammation in Latinas: Variations by Anger Type?

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Cardiovascular disease (CVD) is the leading cause of mortality within the U.S., and among Latinos, the largest U.S. ethnic minority group (American Heart Association, 2010). Mexican-American women in particular have rates of CVD only slightly lower than non-Latino white women (30.9% versus 33.8%), and rates of coronary heart disease (CHD) that are comparable [5.6% versus 5.8%; (Roger et al., 2011)]. Although inroads have been made [e.g., INTERHEART (Rosengren et al., 2004), Multi-Ethnic Study of Atherosclerosis (Bild et al., 2002)], knowledge about psychosocial factors related to CVD in Latinos remains limited. Anger and hostility appear to play a salient role in CVD risk and progression (Smith et al., in press; Smith et al., 2004); however, their association with CVD in Latinos remains relatively unexplored.

Anger has been defined as “an unpleasant emotion ranging in intensity from irritation or annoyance to fury or rage” (Smith et al., 2004), and can be conceptualized from a trait or state perspective. Hostility is a trait-like cognitive characteristic described as “an expectation that others are likely sources of wrong-doing, a relational view of opposition toward others” (Smith et al., 2004). Consequently, anger is viewed as being emotional in nature, whereas hostility, and in particular, cynical hostility, the dimension that involves mistrust of others and their perceived selfish motives, is considered to have a greater cognitive or attitudinal component (Sirois and Burg, 2003).

Reviews of the relationship between anger and CVD have found that anger is predictive of CVD morbidity in males and females (Sirois and Burg, 2003; Smith et al., 2004; Steptoe and Brydon, 2009), with one prospective 7- year study reporting a 2.66 times increased risk of coronary events in men reporting the greatest versus the lowest levels of anger (Kubzansky and Kawachi, 2000). Similarly, hostility has been shown to have a consistent association (r=.18) with coronary heart disease (CHD) outcomes in males and females (Miller et al., 1996a), with one study reporting twice the risk of a recurrent myocardial infarction (MI) in

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The current study provides evidence that specific aspects of anger and hostility relate to inflammatory pathways that potentially influence CVD risk in Latino women.

women with CHD in the highest versus the lowest quartile of hostility (Chaput et al., 2002), and has also been associated with greater CVD mortality (Matthews et al., 2004; Smith et al., 2004). Cynical hostility, specifically, has been linked to CVD risk markers including visceral adipose tissue, carotid intimal-medial thickness, and the metabolic syndrome, as well as CHD incidence and mortality (Lewis et al., 2009; Nelson et al., 2004; Raikkonen et al., 1999; Tindle et al., 2009).

It is unclear, however, whether these associations are similar across gender groups, as the majority of research has not disaggregated effects for men versus women (Kubzansky and Kawachi, 2000; Miller et al., 1996b). A recent meta-analysis (Chida and Steptoe, 2009) that evaluated anger and hostility in aggregate found that hostility-related elevated risk was 22% in healthy males but nonsignificant in healthy females at 7%; however, only 5 of the 21 studies reviewed contained information on females specifically, and the authors did not report results for research in diseased samples as too few studies evaluated gender differences. There is some evidence that differential relationships with CVD exist by gender, especially for anger and its dimensions (Culic et al., 2005; Davidson and Mostofsky, 2010; Harburg et al., 2003; Matthews et al., 1998; Player et al., 2007; Siegman et al., 2000; Williams et al., 2007).

Moreover, the majority of research has focused on non-Latino white samples (Miller et al., 1996a). A few studies examining relationships between anger, hostility and CVD outcomes have reported stronger effects among non-Latino blacks when compared with non-Latino whites (Magai et al., 2003; Markovitz et al., 1997; Tindle et al., 2009; Williams et al., 2007). To our knowledge only one study has evaluated anger and hostility as CVD risk factors among Latinos, and found that the dimension of “anger-out” was related to left ventricular mass, but only in females (Gleiberman et al., 2008). Consequently, additional work evaluating how these psychosocial factors and their dimensions relate to CVD risk in Latino women is greatly needed.

One mechanism by which anger and hostility may affect CVD risk is through the promulgation of systemic inflammation. It is believed that chronic anger and hostility lead to overstimulation of sympathetic-adrenal-medullary (SAM) and hypothalamicpituitary-adrenal (HPA) axis systems, triggering the frequent release of catecholamines and cortisol. Catecholamines are putative catalysts of systemic inflammatory processes (Kubzansky and Kawachi, 2000; Sirois and Burg, 2003; Smith and Ruiz, 2002), and cortisol, while acutely anti-inflammatory, is thought to result in increased inflammation with chronic secretion (Black, 2006). Continuous cortisol exposure is purported to lead to adrenal organ exhaustion and glucocorticoid receptor resistance, thereby causing disruption of the glucocorticoid anti-inflammatory feedback system, and increased inflammation; in addition, chronic cortisol secretion is believed to lead to increased susceptibility to secondary inflammations through down-regulation of the immune response (Hansel et al., 2010). Although causality is debated (e.g., Danesh and Pepys, 2009; Rattazzi et al., 2003), inflammation is thought to be involved in the creation of atherosclerotic plaques through promotion of smooth muscle proliferation at the site of endothelial dysfunction, and initiation of macrophage and lymphocyte accumulation, leading to plaque growth, instability and rupture (Lind, 2003; Ross, 1999). Intercellular adhesion molecule-1 (ICAM-1), which can be assessed in circulation in its soluble form (sICAM-1), promotes recruitment of inflammatory cells to the endothelium by facilitating firm leukocyte adhesion to the endothelial wall, leading to the initiation of pro-inflammatory signaling cascades (Keaney et al., 2004; Lawson and Wolf, 2009). Higher sICAM-1 has been linked to increased risk of CVD (Hajilooi et al., 2003; Jin et al., 2009; Luo et al., 2004; Shai et al., 2006; Tanne et al., 2002).

Previous studies of relationships between anger and/or hostility and inflammation (Coccaro, 2006; Graham et al., 2006; Marsland et al., 2008; Miller et al., 2003a; Stewart et al., 2008; Suarez et al., 2004) have focused on other inflammatory markers (e.g., interleukins, Tumor Necrosis Factor Alpha, C-Reactive Protein [CRP]), and not ICAMs, which represent a separate component of the inflammatory pathway and may be early indicators of endothelial damage (Blann et al., 2002; Kop, 1999). To our knowledge, only one study to date has considered anger in relation to sICAM-1 reactivity, and found no association; however, trait anger was not measured, and state anger was assumed to be provoked by a laboratory task (Kop et al., 2008).

The purpose of the current study was to investigate whether greater levels of cynical hostility and trait anger are associated with higher levels of sICAM-1 in Mexican-American women, an understudied population at high risk for CVD. As well as considering overall levels of trait anger, the study examined anger temperament and anger reaction subdimensions to determine whether their relationships with sICAM-1 differed. Given the conflicting nature of results thus far regarding the relationship of anger and cynical hostility to inflammation and the dearth of research in Latinos, we do not advance any *a priori* hypotheses regarding expected direction of effects.

Methods

Participants and Recruitment

Participants were recruited using simple random sampling as part of a larger study on psychosocial factors and CVD risk in middle-aged Mexican-American women living near the San Diego, CA - Mexico border. Participants were identified from a list obtained through a commercial database that used public records to gather information such as names, addresses, and general demographics for a particular region. Potential participants were randomly selected from households with Latino surnames that had a female occupant in the targeted age range. They were subsequently contacted through targeted direct mailings and phone solicitation, with the goal of obtaining a sample representative across levels of socioeconomic status. Eligible participants were female, between the ages of 40–65, Mexican-American (selfidentified), residing in selected recruitment areas, and free of cardiovascular diseases, diabetes, kidney disease, chronic inflammatory conditions, or other major illnesses, pregnancy, or medications with autonomic nervous system effects. Six-hundred and fifty-six women were screened, 363 (55.3%) were deemed eligible, and 321 (88.4%) participated in some or all portions of the study. Of these, 17 did not complete the physical exam or blood draw and 10 had incomplete data on covariates, resulting in a final sample of $N=294$ for the current study.

Procedures

After enrollment, participants were scheduled for two home visits by study staff. Participants were given instructions over the phone to avoid the use of anti-inflammatory medications for at least 48 hours, strenuous exercise and alcohol consumption for at least 24 hours, and caffeine and tobacco consumption for at least 30 minutes prior to the physical exam (i.e., second visit). Participants were rescheduled if they reported any acute illness. During the first visit, participants were consented and given a battery of questionnaires to complete (in Spanish or English) assessing sociodemographic characteristics, behavioral risk factors, health history, and psychosocial variables. They were also given instructions for a 12-hour, overnight fast. During the second visit, a licensed phlebotomist obtained a fasting blood draw, and a research assistant performed physical measurements (blood pressure, height, weight, waist circumference). Research staff was trained in all study procedures. The SDSU and UCSD institutional review boards approved all study procedures.

Anger and Cynical hostility

Anger was assessed using the Spielberger Trait Anger Scale, a 10-item self-report measure of trait anger (Speilberger et al., 1983), comprised of two subscales, anger temperament and anger reaction. Anger temperament is the predisposition toward quick, unprovoked or minimally provoked anger. Anger reaction refers to anger aroused in response to frustration, criticism, or unfair treatment. For each item, participants marked a response ranging from (0) strongly disagree to (4) strongly agree. Cynical hostility was measured using an abbreviated 6-item version of the 13-item Cook-Medley cynicism subscale, a scale that measures negative beliefs and attitudes toward others (Barefoot et al., 1989). This abbreviated version has been used in other studies on mechanisms of CVD risk (Janicki-Deverts et al., 2010; Schulz et al., 2008). For each item, participants indicated whether they felt the item to be true of them (1) or false (0), resulting in a score from 0 to 6. Cronbach's alpha reliability coefficients were acceptable for the overall trait anger (.82), anger reaction (.67), and anger temperament (.81), as was the Kuder-Richardson 20 coefficient for the cynical hostility scale (.72). Previously validated Spanish versions of the cynical hostility (Butcher et al., 2007) and anger measures were utilized (Mind Garden, n.d.).

Soluble Inter-cellular Adhesion Molecule-1, Lipids, Leukocytes and HbA1c

Circulating levels of sICAM-1, lipids, and leukocytes were obtained from a venous blood draw using either EDTA as a preservative or serum. Samples for sICAM-1 were immediately put on ice and centrifuged and the plasma stored at −70 degree C until assay. Levels of sICAM-1 were determined at the University of California San Diego (UCSD) Clinical Biomarker Laboratory in non-freeze thawed plasma samples by commercial Enzyme-Linked Immunosorbent Assay (MSD, Gaithersburg, Maryland). Inter- and intra-assay coefficients of variation were <10%.

Serum low-density lipoprotein cholesterol, high-density lipoprotein cholesterol (HDL-c,) and triglycerides were measured by modified enzymatic methods (intra and inter assay CV <3%) on AU5400 random access analyzer, following standards set by the Lipid Standardization Program of the Centers for Disease Control and Prevention (Warnick, 2000). Hemoglobin A1c (HbA1c), a measure of average blood glucose concentration, was assayed using a Biorad Diomat high-pressure liquid chromatography analyzer. Complete blood count, including leukocytes, and differential counts were measured by flow Cytometry method (precision < 6% red blood cell count and varied in analytes) on a Beckman Coulter LH750 Hematology Analyzer, with improved cell enumeration accuracy, equipped with AccuCount technology.

Covariates

Analyses controlled for demographic, behavioral and biological factors associated with anger, hostility, or inflammation in prior research that could represent potential confounding influences or biobehavioral pathways from anger/hostility to inflammation, and that have been controlled for in studies of anger/hostility and inflammation (Fortmann et al., 2004; Graham et al., 2006; Marsland et al., 2008; Stewart et al., 2008; Suarez et al., 2004)1. Demographic covariates included age, nativity (U.S. or Mexico-born), and socioeconomic status (SES). SES was assessed by education (highest level of education achieved, from no education to a doctoral or professional degree, recoded into six categories for analysis) and yearly income (measured on an ordinal scale, ranging from less than $6000/year to more than $96000/year, recoded into six categories for analysis). Income was imputed using the

[1]Menopausal status was included in preliminary analyses; however, as this variable was highly correlated with age, and did not relate to sICAM-1 or alter model results when statistically controlled in addition to age, we excluded it from further analyses for parsimony.

expectation-maximization algorithm method (Dempster et al., 1977) for 5 participants with missing data using age and education as predictors. Behavioral covariates consisted of current smoking and alcohol use (both coded yes/no). Exercise frequency and intensity was assessed using the Leisure Time Exercise Questionnaire (Godin and Shephard, 1985), which asks respondents to state the number of times during a typical week they participated in strenuous or moderate exercise for at least 15 minutes, and provides an estimate of total Metabolic Equivalent of Task Units (METs) per week (an estimate of the intensity and energy expenditure of physical activity that is comparable across differing body sizes). For the Spanish language version of the scales, translation was conducted following a standard protocol which involved forward/back translation by trained bilingual staff, review for semantic equivalence by an expert committee, pilot-testing of items in bilingual and monolingual focus groups, and evaluation of equivalence of reliability and validity of scales by language version.

Biological covariates included body mass index (BMI), which was calculated using the standard algorithm [i.e., weight (kilograms)/height (meters)^2]. Systolic and diastolic blood pressure (SBP and DBP) readings were taken while the participant was in a seated position, with arm elevated to heart height, following 30 minutes of rest, using an automatic sphygmomanometer shown to be valid and reliable (i.e., Omron HEM 705-CP, Omron Corporation, Kyoto Japan). For parsimony, mean arterial pressure (MAP; 2/3*DBP + SBP/3) was used to indicate average blood pressure. Lipids, which have been associated with both hostility/anger and sICAM-1 (Bunde and Suls, 2006; Dujovne and Houston, 1991; Hsu et al., 2009; Shai et al., 2006; Suarez et al., 1998; Thorand et al., 2006), and HbA1c were also included as covariates. To account for the effects of acute infection on sICAM-1 levels, leukocyte count was included as a control variable, as in previous research (e.g., Alley et al., 2006).

Statistical Analyses

All data were analyzed using Predictive Analytics SoftWare, Version 17.0 for Macintosh (PASW, Chicago, Illinois, USA). Model assumptions about linearity, normality, independence, and homoscedasticity of errors were assessed graphically and analytically and were adequately satisfied. Ordinary Least Squares (OLS) hierarchical multiple linear regression was used to test the relationship between each predictor and sICAM-1. For each predictor (i.e., anger, anger temperament, anger reaction, cynical hostility), a model adjusting only for demographics (i.e., age, nativity, SES) was first estimated (Model 1). Biological variables (i.e., BMI, MAP, lipids, HbA1c, leukocyte count) that represented possible mediators or confounds of the relationship between the predictors and sICAM-1 were then added to Model 1 (Model 2). In addition to covariates included in Models 1 and 2, the final model (Model 3) included health behaviors that could fall in the causal pathway (i.e., smoking, alcohol use, exercise) between anger and CVD, to test the direct association of predictors with sICAM-1 independent of these intermediate mechanisms. These models were estimated for each predictor separately, and a final set of models was estimated entering all predictors simultaneously with the exception of the full anger scale, which was redundant with the anger subscales.

All non-categorical variables were centered about the sample mean to increase the interpretability of coefficients. Due to significant skew, exercise was categorized into quintiles for the purpose of analysis. Although the anger and cynical hostility scales were positively skewed, results using log-transformed versions were not appreciably different than those without transformation; consequently, for ease of interpretability and brevity, only analyses using non-transformed predictors are presented below.

Results

Table 1 displays means and standard deviations or frequencies and *N*'s, for all demographic, behavioral, and biological covariates as well as predictor and outcome variables. Correlations between anger scales, cynical hostility, sICAM-1 and all covariates are also listed. Means of anger (*M*= 15.63, *SD*= 4.54), anger temperament (*M*= 6.17, *SD*= 2.32), anger reaction (*M*= 6.76, *SD*= 2.30) and cynical hostility (*M*= 2.29, *SD*= 1.80) were below the mid-point of each scale. The means for the trait anger and anger reaction scales were also less than equivalent normative sample means for normal (i.e., non-psychiatric) women aged 30 years and older from a variety of occupational backgrounds ranging from professional and managerial to clerical as well as undergraduate and graduate students (17.01, 8.46, respectively; Speilberger, 1999), whereas the anger temperament mean was somewhat higher (5.71). Average age of the current sample was 49.66 years, and the majority had at least some college (53.39%). The mean value of sICAM-1 (274.97) was comparable to levels reported in non-Latino whites but higher than those reported for non-Latino blacks in other female-only (Miller et al., 2003b) and mixed gender samples (Bielinski et al., 2008; Wong et al., 2006). This trend is consistent with prior research that has shown Latinos to have sICAM-1 levels comparable to those of non-Latino whites, but higher than those of non-Latino blacks (Albert, 2007; Albert et al., 2007).

Of all covariates, only nativity and education were significantly related to any of the anger scales. Individuals who were born in the U.S. had lower trait anger (r=−0.13), anger temperament (r=−0.12), and anger reaction (r=−0.14) scores than those who were born in Mexico (all *ps* < .05). Higher education was related to lower anger temperament (r=−0.13), and was the only covariate significantly (inversely) associated with cynical hostility (r= −0.18). SICAM-1 was significantly related to all covariates with the exception of nativity, lipids, smoking, exercise, and leukocyte count. Relationships were in the expected direction with greater age, less education and income, and greater values on CVD risk factors (i.e., BMI, MAP, triglycerides, HbA1c) being associated with higher sICAM-1 levels. One exception was alcohol consumption, wherein lower sICAM-1 values were seen in drinkers (r=−0.12).

Anger, Cynical hostility and sICAM-1

Results of the primary analyses are presented in Table 2. As a set, covariates accounted for 19.2% of the variance in sICAM-1 (p<.001).

Neither overall trait anger nor anger temperament was significantly associated with sICAM-1 in any model (all *ps* >.05). Anger reaction was not related to sICAM-1 in Model 1 (model adjusted for demographics only), but the association approached statistical significance in Model 2 (model adjusted for demographic and biological factors) and Model 3 (fully adjusted model). Those who endorsed greater levels of anger reaction tended to have higher sICAM-1 levels (β =4.77, p=.06, in Model 3). Cynical hostility was significantly related to sICAM-1 in all models (all *ps* <.05), with greater cynical hostility associated with higher sICAM-1 (β = 5.89, p=.04, in Model 3). The significant linear trend for cynical hostility is depicted in Figure 1. When both anger dimensions and cynical hostility were entered simultaneously, the associations for cynical hostility were attenuated in all models, and became marginally significant (β =5.50, p=.07, in Model 3), indicating that the anger and cynical hostility constructs overlap to some extent in their relationship with sICAM-1. No other associations notably differed from models that considered each variable individually. In aggregate, the cynical hostility, anger reaction, and anger temperament scores accounted for 2.20% of the variance explained in sICAM-1 (p=.05), after accounting for all covariates.

Discussion

Mexican-Americans, and Mexican-American women in particular, have been identified in some studies as having risks for CVD mortality higher or equivalent to their non-Latino white counterparts (Hunt et al., 2003; Pandey et al., 2001). The current study is, to our knowledge, the first to examine the association between overall trait anger, anger dimensions and cynical hostility with sICAM-1, in this subpopulation. As a set, anger and cynical hostility accounted for just over 2% of the variance in sICAM-1. Although not large, this effect is comparable to those found in other studies of anger/hostility and markers of inflammation in non-Latino samples (e.g., Miller et al., 2003a; Stewart et al., 2008).

Interestingly, although overall trait anger was not associated with sICAM-1 in the current sample, cynical hostility was. Associations between overall hostility and inflammation appear to vary according to which marker or combination of markers is examined, with various studies reporting positive, negative, and nonsignificant relationships between hostility and IL-6, CRP, and TNF-a (Brummett et al., 2010; Graham et al., 2006; Kiecolt-Glaser et al., 2005; Mommersteeg et al., 2008; Suarez et al., 2004; Suarez et al., 2002). With the exception of the current study there was no prior research to our knowledge demonstrating associations between any dimension of hostility and sICAM-1. Additional work examining other inflammatory markers representing various stages in the inflammatory process (e.g., serum amyloid A, interferon-gamma) would further bolster evidence of an overall link between hostility and inflammation.

Relative to hostility, trait anger has received less attention in the literature as it relates to inflammation. A few studies have found relationships between anger and CRP (Hapuarachchi et al., 2003; Suarez, 2004). It is possible that differences in anger associations with inflammation across studies may be due to unrecognized moderating effects, as interactions have been identified with factors as variant as hypertension status (Williams et al., 2001), multivitamin supplement use (Suarez, 2003), and level of insulin resistance (Suarez et al., 2006). Findings in the literature are equivocal, and may indicate the need for further identification of potential moderating factors.

Whereas overall trait anger did not relate to inflammation in the current study, there was a trend for higher levels of anger reaction, but not anger temperament, to relate to elevated sICAM-1 levels. Anger reaction trends were not present in demographic-only controlled models, but became evident in models including biobehavioral covariates, which is likely the result of a reduction in error variance in sICAM-1 with the addition of these controls. Previous work on dimensions of anger or hostility in relation to inflammation has produced varying results based on dimension type. For example, anger-out but not anger-in was positively related to CRP levels in an Australian sample, and anger control (tendency to control expressions of anger) was associated with lower levels of CRP (Hapuarachchi et al., 2003). A study of the relationship between the cognitive, affective, and behavioral components of hostility and IL-6 and CRP found that only the behavioral component was significantly related to inflammatory markers (Marsland et al., 2008); however only the cognitive component was associated with TNF-α and interferon gamma in another study (Janicki-Deverts et al., 2010). Thus, it is apparent that differences across dimensions exist, although no clear pattern has emerged. With further research, identification of “high-risk dimensions” could help inform interventional efforts toward anger reduction for patients with known heightened CVD vulnerability.

The possible role of cultural influences in the context of the current findings warrants additional comment. A Latino cultural belief, especially prevalent in rural populations, is that the frequent expression of strong emotions like anger can predispose an individual to

diseases such as diabetes (Coronado et al., 2004), or psychological distress, such as the culturally bound syndrome “ataque de nervios” (nerve attack; Arce and Torres-Matrullo, 1982; de Snyder et al., 2000). Thus, although they may be angry, women in the study may have been less likely to endorse these feelings, as they may view this as being culturally non-sanctioned or harmful, resulting in possible confounding of the relationship between trait anger, anger temperament and sICAM-1. However, the measurement of specific cultural beliefs and attitudes and their possible moderating effects should be further explored.

The current study possesses several strengths, including the examination of a novel marker of inflammation, the exploration of associations by subdimensions of anger, and an ethnic minority sample in which the relationship between anger, hostility and inflammation has been understudied. Nonetheless, several limitations should also be noted. The study is cross-sectional in nature, and causality cannot be determined. It is possible that inflammation can promote anger, as studies have reported increased anger, aggression and hostility in patients given cytokine therapy to treat medical conditions (Zalcman and Siegel, 2006). In addition, the measures of anger and cynical hostility were self-report, and may be affected by social desirability or recall bias; the employment of multi-method measures (e.g., significant-other report, relative-report, child-report) will be useful for future research. Finally, the current sample consisted only of Mexican American women, from selected communities in a border region, and thus, results may not generalize to males or other Latino subgroups.

The current study provides additional evidence that negative emotions and cognitions, particularly, specific aspects of anger and hostility, relate to physiological pathways that have the potential to influence health over the long term. Several questions remain to be answered regarding specific physiological pathways, differences that may exist by other anger subdimensions, specific populations studied (e.g., healthy vs. post-coronary event, young vs. elderly, white vs. ethnic minority, etc.), and inflammatory biomarker type and function. Answers to these questions can eventually inform and tailor interventions to promote CVD risk reduction.

References

Albert MA. Inflammatory biomarkers, race/ethnicity and cardiovascular disease. Nutr Rev. 2007; 65:S234–S238. [PubMed: 18240555]

Albert MA, Glynn RJ, Buring JE, Ridker PM. Relation between soluble intercellular adhesion molecule-1, homocysteine, and fibrinogen levels and race/ethnicity in women without cardiovascular disease. The American Journal Of Cardiology. 2007; 99:1246–1251. [PubMed: 17478152]

Alley DE, Seeman TE, Ki Kim J, Karlamangla A, Hu P, Crimmins EM. Socioeconomic status and C-reactive protein levels in the US population: NHANES IV. Brain Behav Immun. 2006; 20:498–504. [PubMed: 16330181]

Arce AA, Torres-Matrullo C. Application of cognitive behavioral techniques in the treatment of Hispanic patients. Psychiatric Quarterly. 1982; 54:230–236. [PubMed: 7187511]

American Heart Association. Heart Disease and Stroke Statistics - 2010 Update. Dallas, Texas: 2010.

Barefoot JC, Dodge KA, Peterson BL, Dahlstrom WG, Williams RB Jr. The Cook-Medley hostility scale: item content and ability to predict survival. Psychosom Med. 1989; 51:46–57. [PubMed: 2928460]

Bielinski SJ, Pankow JS, Li N, Hsu FC, Adar SD, Jenny NS, Bowden DW, Wasserman BA, Arnett D. ICAM1 and VCAM1 polymorphisms, coronary artery calcium, and circulating levels of soluble ICAM-1: the multi-ethnic study of atherosclerosis (MESA). Atherosclerosis. 2008; 201:339–344. [PubMed: 18420209]

Bild DE, Bluemke DA, Burke GL, Detrano R, Diez Roux AV, Folsom AR, Greenland P, Jacob DR Jr, Kronmal R, Liu K, Nelson JC, O’Leary D, Saad MF, Shea S, Szklo M, Tracy RP. Multi-ethnic

study of atherosclerosis: objectives and design. Am J Epidemiol. 2002; 156:871–881. [PubMed: 12397006]

Black PH. The inflammatory consequences of psychologic stress: relationship to insulin resistance, obesity, atherosclerosis and diabetes mellitus, type II. Med Hypotheses. 2006; 67:879–891. [PubMed: 16781084]

Blann AD, Ridker PM, Lip GYH. Inflammation, cell adhesion molecules, and stroke: tools in pathophysiology and epidemiology? Stroke; A Journal Of Cerebral Circulation. 2002; 33:2141–2143.

Brummett BH, Boyle SH, Ortel TL, Becker RC, Siegler IC, Williams RB. Associations of depressive symptoms, trait hostility, and gender with C-reactive protein and interleukin-6 response after emotion recall. Psychosom Med. 2010; 72:333–339. [PubMed: 20190126]

Bunde J, Suls J. A quantitative analysis of the relationship between the Cook-Medley Hostility Scale and traditional coronary artery disease risk factors. Health Psychology: Official Journal Of The Division Of Health Psychology, American Psychological Association. 2006; 25:493–500.

Butcher, JN.; Cabiya, J.; Lucio, EM.; Garrido, M. Assessing Hispanic Clients Using the MMPI-2 and MMPI-A. American Psychological Association; Washington, D.C: 2007.

Chaput LA, Adams SH, Simon JA, Blumenthal RS, Vittinghoff E, Lin F, Loh E, Matthews KA. Hostility predicts recurrent events among postmenopausal women with coronary heart disease. Am J Epidemiol. 2002; 156:1092–1099. [PubMed: 12480653]

Chida Y, Steptoe A. The association of anger and hostility with future coronary heart disease: a meta-analytic review of prospective evidence. J Am Coll Cardiol. 2009; 53:936–946. [PubMed: 19281923]

Coccaro EF. Association of C-reactive protein elevation with trait aggression and hostility in personality disordered subjects: a pilot study. J Psychiatr Res. 2006; 40:460–465. [PubMed: 15993896]

Coronado GD, Thompson B, Tejeda S, Godina R. Attitudes and Beliefs among Mexican Americans about Type 2 Diabetes. J Health Care Poor Underserved. 2004; 15:576–588. [PubMed: 15531816]

Culic V, Eterovic D, Miric D. Meta-analysis of possible external triggers of acute myocardial infarction. Int J Cardiol. 2005; 99:1–8. [PubMed: 15721492]

Danesh J, Pepys MB. C-reactive protein and coronary disease: is there a causal link? Circulation. 2009; 120:2036–2039. [PubMed: 19901186]

Davidson KW, Mostofsky E. Anger expression and risk of coronary heart disease: evidence from the Nova Scotia Health Survey. Am Heart J. 2010; 159:199–206. [PubMed: 20152217]

de Snyder VNS, Diaz-Perez MDJ, Ojeda VD. The prevalence of nervios and associated symptomatology among inhabitants of Mexican rural communities. Cult Med Psychiatry. 2000; 24:453–470. [PubMed: 11128627]

Dempster AP, Laird NM, Rubin DB. Maximum Likelihood from Incomplete Data Via Em Algorithm. Journal of the Royal Statistical Society Series B-Methodological. 1977; 39:1–38.

Dujovne VF, Houston BK. Hostility-related variables and plasma lipid levels. J Behav Med. 1991; 14:555–565. [PubMed: 1791620]

Fortmann SP, Ford E, Criqui MH, Folsom AR, Harris TB, Hong Y, Pearson TA, Siscovick D, Vinicor F, Wilson PF. CDC/AHA Workshop on Markers of Inflammation and Cardiovascular Disease: Application to Clinical and Public Health Practice: report from the population science discussion group. Circulation. 2004; 110:e554–e559. [PubMed: 15611381]

Gleiberman L, Greenwood TA, Luke A, Delgado MC, Weder AB. Anger types: heritability and relation to blood pressure, body mass index, and left ventricular mass. Journal Of Clinical Hypertension (Greenwich, Conn). 2008; 10:700–706.

Godin GF, Shephard RJ. A simple method to assess exercise behavior in the communty. Canadian Journal of Applied Sports Science. 1985; 10:141–146.

Graham JE, Robles TF, Kiecolt-Glaser JK, Malarkey WB, Bissell MG, Glaser R. Hostility and pain are related to inflammation in older adults. Brain Behav Immun. 2006; 20:389–400. [PubMed: 16376518]

Hajilooi M, Sanati A, Ahmadieh A, Ghofraniha A, Massoud A. Circulating ICAM-1, VCAM-1, E-selectin, P-selectin, and TNFalphaRII in patients with coronary artery disease. Immunol Invest. 2003; 32:245–257. [PubMed: 14603993]

Hansel A, Hong S, Camara RJA, von Kanel R. Inflammation as a psychophysiological biomarker in chronic psychosocial stress. Neurosci Biobehav Rev. 2010; 35:115–121. [PubMed: 20026349]

Hapuarachchi JR, Chalmers AH, Winefield AH, Blake-Mortimer JS. Changes in clinically relevant metabolites with psychological stress parameters. Behavioral Medicine (Washington, DC). 2003; 29:52–59.

Harburg E, Julius M, Kaciroti N, Gleiberman L, Schork MA. Expressive/suppressive anger-coping responses, gender, and types of mortality: a 17-year follow-up (Tecumseh, Michigan, 1971–1988). Psychosom Med. 2003; 65:588–597. [PubMed: 12883109]

Hsu L-A, Ko Y-L, Wu S, Teng M-S, Chou H-H, Chang C-J, Chang P-Y. Association of soluble intercellular adhesion molecule-1 with insulin resistance and metabolic syndrome in Taiwanese. Metabolism. 2009; 58:983–988. [PubMed: 19394054]

Hunt KJ, Resendez RG, Williams K, Haffner SM, Stern MP, Hazuda HP. All-cause and cardiovascular mortality among Mexican-American and non-Hispanic White older participants in the San Antonio Heart Study- evidence against the “Hispanic paradox”. Am J Epidemiol. 2003; 158:1048–1057. [PubMed: 14630600]

Janicki-Deverts D, Cohen S, Doyle WJ. Cynical hostility and stimulated Th1 and Th2 cytokine production. Brain Behav Immun. 2010; 24:58–63. [PubMed: 19647069]

Jin C, Lu L, Zhang RY, Zhang Q, Ding FH, Chen QJ, Shen WF. Association of serum glycated albumin, C-reactive protein and ICAM-1 levels with diffuse coronary artery disease in patients with type 2 diabetes mellitus. Clinica Chimica Acta; International Journal Of Clinical Chemistry. 2009; 408:45–49.

Keaney JF Jr, Massaro JM, Larson MG, Vasan RS, Wilson PWF, Lipinska I, Corey D, Sutherland P, Vita JA, Benjamin EJ. Heritability and correlates of intercellular adhesion molecule-1 in the Framingham Offspring Study. J Am Coll Cardiol. 2004; 44:168–173. [PubMed: 15234428]

Kiecolt-Glaser JK, Loving TJ, Stowell JR, Malarkey WB, Lemeshow S, Dickinson SL, Glaser R. Hostile marital interactions, proinflammatory cytokine production, and wound healing. Arch Gen Psychiatry. 2005; 62:1377–1384. [PubMed: 16330726]

Kop WJ. Chronic and acute psychological risk factors for clinical manifestations of coronary artery disease. Psychosom Med. 1999; 61:476–487. [PubMed: 10443756]

Kop WJ, Weissman NJ, Zhu J, Bonsall RW, Doyle M, Stretch MR, Glaes SB, Krantz DS, Gottdiener JS, Tracy RP. Effects of acute mental stress and exercise on inflammatory markers in patients with coronary artery disease and healthy controls. The American Journal Of Cardiology. 2008; 101:767–773. [PubMed: 18328837]

Kubzansky LD, Kawachi I. Going to the heart of the matter: do negative emotions cause coronary heart disease? J Psychosom Res. 2000; 48:323–337. [PubMed: 10880655]

Lawson C, Wolf S. ICAM-1 signaling in endothelial cells. Pharmacological Reports: PR. 2009; 61:22–32. [PubMed: 19307690]

Lewis TT, Everson-Rose SA, Karavolos K, Janssen I, Wesley D, Powell LH. Hostility is associated with visceral, but not subcutaneous, fat in middle-aged African American and white women. Psychosom Med. 2009; 71:733–740. [PubMed: 19592520]

Lind L. Circulating markers of inflammation and atherosclerosis. Atherosclerosis. 2003; 169:203–214. [PubMed: 12921971]

Luo Y, Xie XM, Liu HX. [Related inflammation markers among different types of coronary heart disease]. Zhong Nan Da Xue Xue Bao. Yi Xue Ban = Journal Of Central South University. Medical Sciences. 2004; 29:227–229.

Magai C, Kerns MD, Gillespie M, Huang B. Anger experience and anger inhibition in sub-populations of African American and European American older adults and relation to circulatory disease. Journal Of Health Psychology. 2003; 8:413–432. [PubMed: 19127709]

Markovitz JH, Smith D, Raczynski JM, Oberman A, Williams OD, Knox S, Jacobs DR Jr. Lack of relations of hostility, negative affect, and high-risk behavior with low plasma lipid levels in the

Coronary Artery Risk Development in Young Adults Study. Arch Intern Med. 1997; 157:1953–1959. [PubMed: 9308507]

Marsland AL, Prather AA, Petersen KL, Cohen S, Manuck SB. Antagonistic characteristics are positively associated with inflammatory markers independently of trait negative emotionality. Brain Behav Immun. 2008; 22:753–761. [PubMed: 18226879]

Matthews KA, Gump BB, Harris KF, Haney TL, Barefoot JC. Hostile behaviors predict cardiovascular mortality among men enrolled in the Multiple Risk Factor Intervention Trial. Circulation. 2004; 109:66–70. [PubMed: 14662707]

Matthews KA, Owens JF, Kuller LH, Sutton-Tyrrell K, Jansen-McWilliams L. Are hostility and anxiety associated with carotid atherosclerosis in healthy postmenopausal women? Psychosom. Med. 1998; 60:633–638.

Miller GE, Freedland KE, Carney RM, Stetler CA, Banks WA. Cynical hostility, depressive symptoms, and the expression of inflammatory risk markers for coronary heart disease. J Behav Med. 2003a; 26:501–515. [PubMed: 14677209]

Miller MA, Sagnella GA, Kerry SM, Strazzullo P, Cook DG, Cappuccio FP. Ethnic differences in circulating soluble adhesion molecules: the Wandsworth Heart and Stroke Study. Clinical Science (London, England: 1979). 2003b; 104:591–598.

Miller TQ, Smith TW, Turner CW, Guijarro ML, Hallet AJ. A meta-analytic review of research on hostility and physical health. Psychol Bull. 1996a; 119:322–348. [PubMed: 8851276]

Miller TQ, Smith TW, Turner CW, Guijarro ML, Hallet AJ. Meta-analytic review of research on hostility and physical health. Psychol Bull. 1996b; 119:322–348. [PubMed: 8851276]

Mind Garden. n.d. http://www.mindgarden.com/products/staisad.htm

Mommersteeg PMC, Vermetten E, Kavelaars A, Geuze E, Heijnen CJ. Hostility is related to clusters of T-cell cytokines and chemokines in healthy men. Psychoneuroendocrinology. 2008; 33:1041–1050. [PubMed: 18640786]

Nelson TL, Palmer RF, Pedersen NL. The metabolic syndrome mediates the relationship between cynical hostility and cardiovascular disease. Exp Aging Res. 2004; 30:163–177. [PubMed: 15204630]

Pandey DK, Labarthe DR, Goff DC, Chan W, Nichaman MZ. Community-wide coronary heart disease mortality in Mexican Americans equals or exceeds that in non-Hispanic whites: the Corpus Christi Heart Project. The American Journal Of Medicine. 2001; 110:81–87. [PubMed: 11165547]

Player MS, King DE, Mainous AG 3rd, Geesey ME. Psychosocial factors and progression from prehypertension to hypertension or coronary heart disease. Annals Of Family Medicine. 2007; 5:403–411. [PubMed: 17893381]

Raikkonen K, Matthews KA, Kuller LH, Reiber C, Bunker CH. Anger, hostility, and visceral adipose tissue in healthy postmenopausal women. Metabolism. 1999; 48:1146–1151. [PubMed: 10484055]

Rattazzi M, Puato M, Faggin E, Bertipaglia B, Zambon A, Pauletto P. C-reactive protein and interleukin-6 in vascular disease: culprits or passive bystanders? J Hypertens. 2003; 21:1787–1803. [PubMed: 14508181]

Roger VL, Go AS, Lloyd-Jones DM, Adams RJ, Berry JD, Brown TM, Carnethon MR, Dai S, de Simone G, Ford ES, Fox CS, Fullerton HJ, Gillespie C, Greenlund KJ, Hailpern SM, Heit JA, Ho PM, Howard VJ, Kissela BM, Kittner SJ, Lackland DT, Lichtman JH, Lisabeth LD, Makuc DM, Marcus GM, Marelli A, Matchar DB, McDermott MM, Meigs JB, Moy CS, Mozaffarian D, Mussolino ME, Nichol G, Paynter NP, Rosamond WD, Sorlie PD, Stafford RS, Turan TN, Turner MB, Wong ND, Wylie-Rosett J. Committee, o.b.o.t.A.H.A.S., Stroke Statistics Subcommittee, Disease, O.b.o.t.A.H.A.H., Stroke Statistics Writing Group. Heart Disease and Stroke Statistics--2011 Update: A Report From the American Heart Association. Circulation. 2011; 123:e18–209. [PubMed: 21160056]

Rosengren A, Hawken S, Ounpuu S, Sliwa K, Zubaid M, Almahmeed WA, Blackett KN, Sitthi-amorn C, Sato H, Yusuf S. Association of psychosocial risk factors with risk of acute myocardial infarction in 11119 cases and 13648 controls from 52 countries (the INTERHEART study): case-control study. Lancet. 2004; 364:953–962. [PubMed: 15364186]

Ross R. Atherosclerosis--an inflammatory disease. The New England Journal Of Medicine. 1999; 340:115–126. [PubMed: 9887164]

Schulz, R.; Zdaniuk, B.; Greenhouse, J.; Seltman, H. A Pittsburgh Mind-Body center white paper [White Paper]. 2008.

Shai I, Pischon T, Hu FB, Ascherio A, Rifai N, Rimm EB. Soluble intercellular adhesion molecules, soluble vascular cell adhesion molecules, and risk of coronary heart disease. Obesity (Silver Spring, Md). 2006; 14:2099–2106.

Siegman AW, Townsend ST, Civelek AC, Blumenthal RS. Antagonistic behavior, dominance, hostility, and coronary heart disease. Psychosom Med. 2000; 62:248–257. [PubMed: 10772405]

Sirois BC, Burg MM. Negative emotion and coronary heart disease. A review Behav Modif. 2003; 27:83–102.

Smith, TW.; Gallo, LC.; Shivpuri, S.; Brewer, AL. Personality and health: Current issues and emerging perspectives. In: Baum, A.; Revension, T., editors. The Handbook of Health Psychology. 2. Lawrence Erlbaum; Hillsdale, NJ: in press

Smith TW, Glazer K, Ruiz JM, Gallo LC. Hostility, anger, aggressiveness, and coronary heart disease: an interpersonal perspective on personality, emotion, and health. J Pers. 2004; 72:1217–1270. [PubMed: 15509282]

Smith TW, Ruiz JM. Psychosocial influences on the development and course of coronary heart disease: current status and implications for research and practice. J Consult Clin Psychol. 2002; 70:548–568. [PubMed: 12090369]

Speilberger, CD. STAXI-2 State-trait anger expression inventory-2: Professional manual. Psychological Assessment Resources, Inc; Lutz, FL: 1999.

Speilberger, CD.; Jacobs, GA.; Russell, S.; Cranes, RS. Assessment of anger: The trait anger scale. In: Butcher, JN.; Speilberger, CD., editors. Advances in personality assessment. Lawrence Erlbaum; Hillsdale, N.J: 1983.

Steptoe A, Brydon L. Emotional triggering of cardiac events. Neurosci Biobehav Rev. 2009; 33:63–70. [PubMed: 18534677]

Stewart JC, Janicki-Deverts D, Muldoon MF, Kamarck TW. Depressive symptoms moderate the influence of hostility on serum interleukin-6 and C-reactive protein. Psychosom Med. 2008; 70:197–204. [PubMed: 18256345]

Suarez EC. Plasma interleukin-6 is associated with psychological coronary risk factors: Moderation by use of multivitamin supplements. Brain Behav Immun. 2003; 17:296–303. [PubMed: 12831832]

Suarez EC. C-reactive protein is associated with psychological risk factors of cardiovascular disease in apparently healthy adults. Psychosom Med. 2004; 66:684–691. [PubMed: 15385692]

Suarez EC, Bates MP, Harralson TL. The relation of hostility to lipids and lipoproteins in women: evidence for the role of antagonistic hostility. Annals Of Behavioral Medicine: A Publication Of The Society Of Behavioral Medicine. 1998; 20:59–63. [PubMed: 9989309]

Suarez EC, Boyle SH, Lewis JG, Hall RP, Young KH. Increases in stimulated secretion of proinflammatory cytokines by blood monocytes following arousal of negative affect: The role of insulin resistance as moderator. Brain Behav Immun. 2006; 20:331–338. [PubMed: 16288846]

Suarez EC, Lewis JG, Krishnan RR, Young KH. Enhanced expression of cytokines and chemokines by blood monocytes to in vitro lipopolysaccharide stimulation are associated with hostility and severity of depressive symptoms in healthy women. Psychoneuroendocrinology. 2004; 29:1119–1128. [PubMed: 15219635]

Suarez EC, Lewis JG, Kuhn C. The relation of aggression, hostility, and anger to lipopolysaccharide-stimulated tumor necrosis factor (TNF)-alpha by blood monocytes from normal men. Brain Behav Immun. 2002; 16:675–684. [PubMed: 12480498]

Tanne D, Haim M, Boyko V, Goldbourt U, Reshef T, Matetzky S, Adler Y, Mekori YA, Behar S. Soluble intercellular adhesion molecule-1 and risk of future ischemic stroke: a nested case-control study from the Bezafibrate Infarction Prevention (BIP) study cohort. Stroke; A Journal Of Cerebral Circulation. 2002; 33:2182–2186.

Thorand B, Baumert J, Döring A, Schneider A, Chambless L, Löwel H, Kolb H, Koenig W. Association of cardiovascular risk factors with markers of endothelial dysfunction in middle-aged men and women. Results from the MONICA/KORA Augsburg Study. Thromb Haemost. 2006; 95:134–141. [PubMed: 16543972]

Tindle HA, Chang YF, Kuller LH, Manson JE, Robinson JG, Rosal MC, Siegle GJ, Matthews KA. Optimism, cynical hostility, and incident coronary heart disease and mortality in the Women's Health Initiative. Circulation. 2009; 120:656–662. [PubMed: 19667234]

Warnick GR. Measurement of cholesterol and other lipoprotein constituents in the clinical laboratory. Clinical Chemistry And Laboratory Medicine: CCLM/FESCC. 2000; 38:287–300. [PubMed: 10928647]

Williams JE, Couper DJ, Din-Dzietham R, Nieto FJ, Folsom AR. Race-gender differences in the association of trait anger with subclinical carotid artery atherosclerosis: the Atherosclerosis Risk in Communities Study. Am J Epidemiol. 2007; 165:1296–1304. [PubMed: 17363362]

Williams JE, Nieto FJ, Sanford CP, Tyroler HA. Effects of an angry temperament on coronary heart disease risk: The Atherosclerosis Risk in Communities Study. Am J Epidemiol. 2001; 154:230–235. [PubMed: 11479187]

Wong TY, Islam FMA, Klein R, Klein BEK, Cotch MF, Castro C, Sharrett AR, Shahar E. Retinal vascular caliber, cardiovascular risk factors, and inflammation: the multi-ethnic study of atherosclerosis (MESA). Invest Ophthalmol Vis Sci. 2006; 47:2341–2350. [PubMed: 16723443]

Zalcman SS, Siegel A. The neurobiology of aggression and rage: role of cytokines. Brain Behav Immun. 2006; 20:507–514. [PubMed: 16938427]

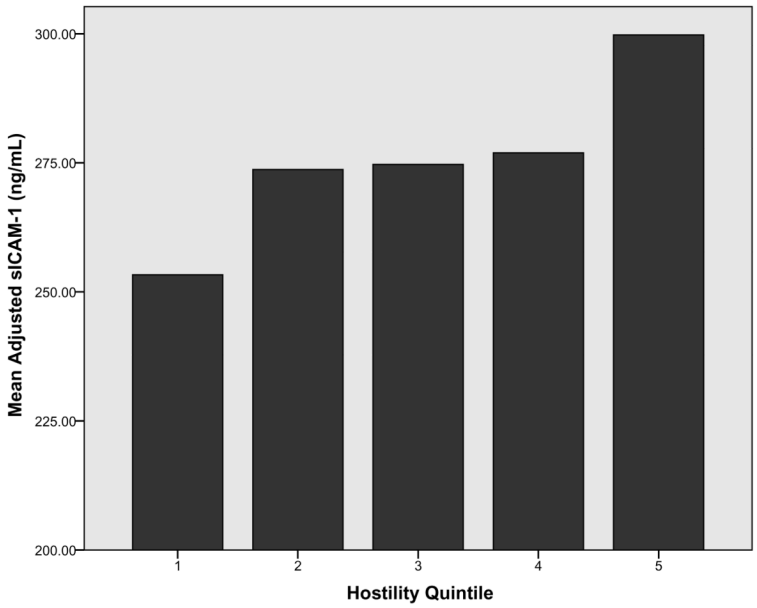

Figure 1.
Mean adjusted values of sICAM-1 by cynical hostility quintile level (quintiles displayed for purposes of illustration; full continuum presented in analyses).

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Table 1

Descriptive statistics (means, standard deviations or frequencies, percentiles) for all variables, and bivariate correlations between anger, anger subscales, cynical hostility, sICAM-1, and covariates

Variable	*M(SD)/N(%)*	Anger *r*	Temp[a] *r*	React[b] *r*	Hostility[c] *r*	sICAM-1 *r*
Age (years)	49.66 (6.53)	−0.07	−0.08	−0.02	0.01	0.20**
Education[d,e]		−0.07	−0.13*	−0.02	−0.18**	−0.18**
≤8th grade	47 (15.99)					
Some high school	54 (18.37)					
GED/high school diploma	36 (12.24)					
Some college	93 (31.63)					
4 year degree	46 (15.64)					
Graduate degree	18 (6.12)					
Yearly Income[d,e]		−0.10	−0.10	−0.04	−0.09	−0.25**
<$15000	30 (10.20)					
$15000–$25000	40 (13.60)					
$25000–$34999	50 (17.01)					
$35000–$49999	55 (18.71)					
$50000–$74999	56 (19.05)					
≥$75000	63 (21.43)					
Nativity[d]						
% Born in U.S.	76 (25.85)	−0.13*	−0.12*	−0.14*	−0.09	0.02
Body Mass Index	29.03 (6.39)	0.01	0.04	−0.02	0.01	0.21**
Mean Arterial Pressure (mmHg)	87.85 (9.82)	−0.02	−0.02	−0.00	0.02	0.22**
HDL-c (mg/dL)	56.85 (15.30)	0.02	0.04	−0.01	0.05	−0.07
LDL-c (mg/dL)	121.33 (32.12)	0.02	−0.01	0.05	0.03	−0.00
Triglycerides (mg/dL)	130.45 (61.84)	−0.06	−0.08	−0.03	−0.01	0.16**
Hemoglobin A1c (% of hemoglobin)	5.73 (0.66)	−0.03	−0.02	−0.03	0.09	0.25**
Leukocyte Count (×10^3/L)	5.94 (1.57)	−0.06	−0.11	0.00	0.06	0.04

Variable	*M(SD)/N(%)*	Anger *r*	Temp[a] *r*	React[b] *r*	Hostility[c] *r*	sICAM-1 *r*
Current Smoker[e]	25 (8.50)	−0.05	−0.05	−0.03	−0.03	0.09
Current Drinker[e]	169 (57.48)	0.09	0.05	0.07	−0.10	−0.12*
Exercise (METs/week)	19.70 (20.57)	−0.03	−0.03	−0.03	−0.08	−0.05
Anger	15.63 (4.54)	-	-	-	-	-
Temperament	6.17 (2.32)	0.86**	-	-	-	-
Reaction	6.76 (2.30)	0.84**	0.49**	-	-	-
Cynical hostility	2.29 (1.80)	0.26**	0.25**	0.21**	-	-
sICAM-1 (ng/mL)	274.97 (93.84)	0.04	−0.01	0.07	0.14*	-

*$p<.05$,

**$p<.01$

[a] Anger Temperament

[b] Anger Reaction

[c] Cynical Hostility

[d] *N (%)*

[e] Spearman rank-order correlation

Table 2

Results of multiple linear regression analyses regressing sICAM-1 separately on anger, anger subscales, and cynical hostility, and simultaneously on anger subscales and cynical hostility, adjusting for demographic (Model 1), biological (Model 2), and behavioral (Model 3) covariates

	Model 1		Model 2		Model 3	
Covariates	**B (S.E.)**		**B (S.E.)**		**B (S.E.)**	
Demographic						
Age	2.86** (0.80)	0.20	2.66** (0.86)	0.18	2.65** (0.86)	0.18
Education	−7.09§ (3.99)	−0.11	−5.56 (4.00)	−0.09	−5.48 (4.05)	−0.09
Income	−12.83** (3.65)	−0.22	−10.21** (3.65)	−0.18	−9.48* (3.72)	−0.16
Nativity[a]	28.95* (12.53)	0.13	24.49* (12.40)	0.11	23.81§ (12.46)	0.11
Biological						
Body Mass Index			1.29 (0.92)	0.09	1.31 (0.92)	0.09
Mean Arterial Pressure			0.89 (0.58)	0.09	0.93 (0.59)	0.10
HDL-c			0.10 (0.39)	0.02	0.08 (0.40)	0.01
LDL-c			−0.34* (0.17)	−0.12	−0.33§ (0.17)	−0.11
Triglycerides			0.10 (0.10)	0.07	0.10 (0.10)	0.06
Hemoglobin A1c			19.60* (8.28)	0.14	19.31* (8.32)	0.14
Lymphocyte Count			0.48 (3.38)	0.01	−0.31 (3.43)	−0.01
Behavioral						
Current Smoker[b]					28.25 (18.52)	0.08
Current Drinker[b]					−6.91 (10.82)	−0.04
Exercise					0.90 (3.68)	0.01
Predictors						
Anger[c]	0.92 (1.16)	0.04	1.24 (1.14)	0.06	1.42 (1.15)	0.07
Anger Temperament[c]	−0.41 (2.28)	−0.01	−0.01 (2.26)	0.00	0.26 (2.27)	0.01
Anger Reaction[c]	3.33 (2.26)	0.08	3.94§ (2.21)	0.10	4.20§ (2.22)	0.10
Cynical Hostility[c]	5.94* (2.91)	0.11	5.72* (2.87)	0.11	5.89* (2.89)	0.11
Anger	−3.39 (2.61)	−0.08	−3.28 (2.59)	−0.08	−3.07	−0.08

	Model 1		Model 2		Model 3	
Covariates	**B (S.E.)**		**B (S.E.)**		**B (S.E.)**	
Temperament[d]	4.05 (2.59)	0.10	4.65[§] (2.54)	0.11	(2.60)	0.12
Anger Reaction[d]	5.85[§] (3.00)	0.11	5.43[§] (2.96)	0.10	4.77[§] (2.54)	0.11
Cynical Hostility[d]					5.50[§] (2.98)	

[§] p< .10

[*] p< .05,

[**] p<.01

[a] Mexico Born (0), U.S. Born (1),

[b] No (0), Yes (1),

[c] Predictors entered in separate models,

[d] Predictors entered simultaneously