



Published in final edited form as:

Psychosom Med. 2014 ; 76(6): 468–475. doi:10.1097/PSY.0000000000000069.

Associations of chronic stress burden, perceived stress, and traumatic stress with cardiovascular disease prevalence and risk factors in the HCHS/SOL Sociocultural Ancillary Study

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Abstract

Objective—The current study examined multiple stress indicators (chronic, perceived, traumatic) in relation to prevalent coronary heart disease (CHD), stroke, and major cardiovascular disease (CVD) risk factors (i.e., diabetes, dyslipidemia, hypertension, current smoking) in the multi-site Hispanic Community Health Study/Study of Latinos (HCHS/SOL) Sociocultural Ancillary Study (2010–2011).

Methods—Participants were 5313 men and women, 18–74 years old, representing diverse Hispanic/Latino ethnic backgrounds, who underwent a comprehensive baseline clinical exam and sociocultural exam with measures of stress.

Results—Chronic stress burden was related to a higher prevalence of CHD after adjusting for sociodemographic, behavioral and biological risk factors [OR (95% CI) = 1.22, (1.10–1.36)] and

related to stroke prevalence in the model adjusted for demographic and behavioral factors [OR (95% CI) = 1.26, (1.03–1.55)]. Chronic stress was also related to a higher prevalence of diabetes [OR=1.20, (1.11–1.31)] and hypertension [OR=1.10 (1.02–1.19)] in individuals free from CVD (N=4926). Perceived stress [OR=1.03 (1.01–1.05)] and traumatic stress [OR=1.15 (1.05–1.26)] were associated with a higher prevalence of smoking. Participants who reported a greater number of lifetime traumatic events also unexpectedly showed a *lower* prevalence of diabetes [OR=.89 (.83–.97)] and hypertension [OR=.88 (.82–.93)]. Effects were largely consistent across age and sex groups.

Conclusions—The study underscores the utility of examining multiple indicators of stress in relation to health, since the direction and consistency of associations may vary across distinct stress conceptualizations. In addition, the study suggests that chronic stress is related to higher CVD risk and prevalence in Hispanics/Latinos, the largest U.S. ethnic minority group.

Keywords

Cardiovascular Disease; Coronary Heart Disease; Hispanic; Latino; Stress

Several lines of research suggest that stress plays a role in the etiology and progression of cardiovascular disease (CVD) (1–3). Stress can hasten the atherosclerotic process, trigger acute events, and result in a less favorable prognostic picture in individuals with established disease (4, 5). The magnitude of the association between stress and coronary heart disease (CHD) risk is often in the range of that associated with established biobehavioral risk factors, such as hypertension, dyslipidemia or smoking (1, 6).

Both direct and indirect pathways may underlie the association between stress and CVD. Through neuroendocrine alterations associated with sympathetic adrenal medullary and hypothalamic adreno-corticol system activation, stress can influence cardiovascular, metabolic, and immune regulatory functions in a manner that hastens atherogenesis over time (7, 8). Acute stress-induced alterations in endothelial function and coagulation can foster coronary events in individuals with atherosclerotic disease (7, 8). Stress may also influence CVD risk indirectly, by encouraging atherogenic coping behaviors such as smoking (3), or increasing vulnerability to negative emotions and mood disorders (9), which are themselves putative risk factors (10). Major CVD risk factors, such as diabetes, hypertension, and dyslipidemia may represent an intermediate stage connecting stress, via these direct and indirect mechanisms, with CVD (5).

An important limitation of existing research is the focus on a single conceptualization of stress. However, stress is a complex multi-dimensional construct that is challenging to operationalize (11, 12). At the broadest level, measures differ according to whether they examine demanding environmental events (e.g., trauma; life events) or on the individual's appraisal of these events (e.g., perceived stress). Stress conceptualizations also vary along dimensions of time and severity. Differences in stress measures across studies may contribute to mixed findings in the literature (1, 13), since different dimensions of stress may show divergent relationships with CVD and could operate through different mechanisms. A more comprehensive approach to conceptualizing stress could lend a more accurate depiction of associations with health (13).

Another notable limitation of the extant stress-CVD literature is the lack of studies within people of Hispanic/Latino descent [for an exception in Latin America, see (14)]. The Hispanic/Latino population in the U.S. increased by 43% between 2000 and 2010 alone (15), and Hispanics/Latinos are predicted to comprise 30% of the population by 2050 (16). Research suggests that Hispanics/Latinos show similar or higher rates of CVD risk factors (17, 18) and have poorer CVD risk factor control (19, 20) than non-Hispanic whites. However, paradoxically, many studies have observed lower CVD rates in Hispanics/Latinos when compared with non-Hispanic whites (17, 18). Very little is known about how stress or other psychosocial factors relate to CVD in this large and growing population.

To begin addressing these gaps in the literature, the current study examined associations of multiple dimensions of stress [chronic stress burden across important life domains, generalized appraisals of stress in the past 30 days (i.e., perceived stress), lifetime burden of traumatic stress] with CHD and stroke prevalence among participants in the Hispanic Community Health Study/Study of Latinos (HCHS/SOL), a multi-site cohort study of Hispanic/Latino adults from multiple ethnic background groups (21, 22) and the HCHS/SOL-Sociocultural Ancillary Study, a separate study of sociocultural factors in one third of the HCHS/SOL cohort. To provide (indirect) insight into whether stress has a role early in the atherogenic process in Hispanics/Latinos, additional analyses examined associations of stress with four major, readily ascertained CVD risk factors (17)—diabetes, hypertension, dyslipidemia, and current smoking—in individuals free from CHD or stroke. Although few studies have examined multiple dimensions of stress within a single methodological framework, the literature suggests that enduring stressors have a more salient health impact than other types of stress (4, 5). Therefore, we hypothesized that all types of stress would relate to CVD prevalence and risk factors, but that more consistent associations would be observed for chronic stress than other indicators.

Methods

Participants and Procedures

The HCHS/SOL—The HCHS/SOL is a prospective cohort study that seeks to establish prevalence, incidence, and risk and protective factors for CVD and other chronic conditions. Details concerning the study sample (22) and approach (21) have been reported. Briefly, 16,415 participants who self-identified as Hispanic/Latino [Central American (n=1,732); Cuban (n=2,348); Dominican (n=1,473); Mexican (n=6,472); Puerto-Rican (n=2,728); South American (n=1,072); another/more than one background (n=503); missing (n=87)] were recruited from the Bronx, NY, Chicago, IL, Miami, FL, and San Diego, CA. The target population was all non-institutionalized Hispanic/Latino adults, 18–74 years, residing in the defined community areas. A stratified two-stage area probability sample was applied at each field center. First, selected census block groups were randomly sampled with stratification on Hispanic/Latino and high/low socioeconomic status concentration. Second, households were randomly selected with stratification, from U.S. Postal Service registries that covered most census block groups. Both stages applied oversampling to increase the likelihood of Hispanic/Latino households. In addition, the 45–74 years age group (n=9,714) was oversampled. Screening and recruitment was conducted in-person or by telephone. Of

39,384 selected, screened, and eligible individuals, 41.7% were enrolled. Participants attended a baseline clinical exam with anthropometric assessment, fasting blood draw for assay of CVD risk markers, 2-hour oral glucose tolerance test (OGTT), electrocardiogram to identify evidence of past myocardial infarction (MI), self-report measures of sociodemographic factors and health, and medication review. For the current study, data to determine prevalence of CHD, stroke, diabetes, hypertension, dyslipidemia, smoking, as well as sociodemographic information, body mass index (BMI), alcohol use, and physical activity, were obtained from the HCHS/SOL baseline exam (2008–2011).

The HCHS/SOL Sociocultural Ancillary Study—The methods for the Sociocultural Ancillary Study have been described elsewhere (23). All HCHS/SOL participants who were willing and able to attend a separate visit within 9 months of their baseline exam were eligible (N= 7,321), and 5,313 (72.6% of those eligible) participated. Most participants (72.6%) completed the sociocultural assessment within 4 months of their baseline clinic exam. The Sociocultural Ancillary Study cohort is generally representative of the HCHS/SOL cohort, with the exception that participation was lower among some higher socioeconomic block groups (23). Participants completed a 1–2 hour assessment interview in their preferred language, which included the measures of stress examined in the current study. Participants were compensated \$60 for their time and effort. Institutional Review Board approval was obtained from all study sites for all HCHS/SOL and HCHS/SOL Sociocultural Ancillary Study procedures, and all participants provided written informed consent.

Measures

CVD Prevalence—Participants who self-reported that a doctor told them they had a heart attack, and/or who reported having a cardiac procedure (stent placement, angioplasty, or bypass surgery), and/or for whom electrocardiographic evidence of possible MI was observed at baseline, were categorized as having CHD. Given the correlation between stress and self-reported cardiovascular symptoms in the absence of underlying disease (24, 25), self-reported angina alone was excluded from the CHD definition. Stroke prevalence was evaluated by self-report only (a doctor has told the participant he/she had a stroke) (18).

CVD Risk Factors—Participants who were taking a glycemic regulating medication and/or who met current criteria for diabetes according to physiological information (fasting plasma glucose ≥ 126 mg/dL, 2-h plasma glucose >200 mg/dL during OGTT, glycosylated hemoglobin $\geq 6.5\%$) (26) were classified as having diabetes. Hypertension was defined as systolic and/or diastolic blood pressure $\geq 140/90$ mmHg, respectively, and/or if the participant was currently taking an antihypertensive medication. Participants with triglycerides ≥ 200 mg/dL, low-density lipoprotein cholesterol ≥ 160 mg/dL, or high-density lipoprotein cholesterol < 40 mg/dL, or taking a lipid lowering medication were characterized as dyslipidemic (27). Participants who reported smoking at least 100 cigarettes during their lifetime and currently smoking some or all days were characterized as current smokers (18, 28).

Stress—Chronic stress burden was evaluated with an 8-item scale that assesses the number of current ongoing problems of at least 6 months duration in major life domains (i.e., financial, work, relationship, health problems in self or close other, drug or alcohol problems in close other, caregiving, other chronic stressor). This measure has been used in prior multi-ethnic cohort studies (29, 30). The 10-item Perceived Stress Scale (PSS10) was used to assess appraisals of recent stress (e.g., In the past month, how often have you found that you could not cope with all the things that you had to do?; how often have you felt that you were unable to control the important things in your life?; responses on a five-point likert scale). Scores on the 10 items are summed, with higher scores indicating greater perceived stress. This measure has demonstrated high internal consistency and evidence of construct validity in prior research (31), and showed good internal consistency, support for the one factor structure, and factorial invariance across language versions in the current cohort (English $\alpha = .86$; Spanish $\alpha = .84$). The number of lifetime traumatic life stressors experienced was measured with the Traumatic Stress Screener [TSS; (32)], a brief screening instrument developed to assess traumatic stress exposure in the general population. Participants were asked to indicate the occurrence, frequency, and timing of 10 relatively common traumatic events (e.g., physical or sexual assault; combat exposure; natural disaster) (33). The current study examined the total number of distinct lifetime events, which was thought to be most relevant to understanding the protracted disease process of atherosclerosis (i.e., as opposed to recent events).

Sociodemographic Characteristics—Sociodemographic variables included age, sex self-identified Hispanic/Latino background, study site, education [$<$ high school (HS) diploma/general education degree (GED), HS diploma/GED only, $>$ HS diploma/GED), and household yearly income (10 categories, ranging from $<$ \$10,000 to $>$ \$100,000). Language of interview (Spanish or English) and nativity/immigration status (immigrated $<$ 10 years ago, immigrated 10 years ago, born in US mainland) were examined as proxy indicators of acculturation (34).

Behavioral Health Indicators—BMI was calculated as weight in kg/height in m^2 , and examined continuously. The Global Physical Activity Questionnaire (35) measured physical activity in leisure, work, and transportation contexts. Participants were categorized as having high, moderate, or low levels of physical activity based on duration, frequency, and intensity of activity (35). Participants self-reported quantity and frequency of alcohol consumption, and were grouped as *non-drinkers* (no alcohol in past year), *former drinkers* (stopped using alcohol), *low-risk drinkers* ($<$ 7 and $<$ 14 drinks per week for women and men, respectively), or *at-risk drinkers* ($>$ 7 or $>$ 14 drinks per week for women and men, respectively) (28).

Statistical Analyses

All analyses accounted for design effects and sample weights (22). The sampling weights were non-response adjusted, trimmed to reduce the variability of the weights, calibrated to the 2010 U.S. Census Population according to age, sex and Hispanic/Latino background, and normalized to the overall HCHS/SOL cohort sample size. Descriptive statistics were calculated in IBM SPSS Statistics 20.0 (IBM, Inc., Armonk, NY) using complex survey procedures. The maximum likelihood robust (MLR) estimation procedure in MPlus (36) was

used to estimate model parameters for all remaining analyses. This procedure allowed cases with missing data on at least one study variable to be included in analyses ($n=821$; see Table 1). MLR, as implemented in MPlus, is a full-information maximum likelihood (FIML) approach to missing data in which model parameters (e.g., regression coefficients) and standard errors are estimated using all observed data. Thus, data from both complete cases and partial cases are used to estimate target model parameters. This procedure has been shown to produce unbiased parameter estimates and standard errors under various missing data conditions (37).

Because participants were nested within primary sampling units, multilevel logistic regression models were used to examine associations of stress with CHD prevalence, stroke prevalence, and (in individuals without prevalent CVD; $n= 4926$), diabetes, hypertension, dyslipidemia, and current smoking. The three stress indicators were examined simultaneously to provide information about their unique, and relative, associations with CVD prevalence and risk. All stress variables were normally distributed (skewness and kurtosis statistics < 1.0) and were modeled continuously. Model 1 adjusted for conceptually relevant sociodemographic covariates including age, sex, education, income, language of interview, nativity/immigration, and Hispanic/Latino background, and was calculated for all outcomes. Model 2 added adjustment for behavioral health indicators including BMI, physical activity, alcohol, and smoking, and was conducted for outcomes other than smoking (for which these variables are not viewed as possible confounds or indirect pathways). Finally, Model 3 was conducted for CHD and stroke prevalence and added adjustment for biological risk factors diabetes, hypertension, and dyslipidemia.

Sensitivity analyses were conducted to examine the consistency of the stress-CVD prevalence and risk factor associations across sex and age (18–44 versus 45 and older) groups. These analyses included all age by stress and sex by stress 2-way interaction effects (6 interaction terms), following entry of the stress main effects and control for demographic covariates (Model 1).

Results

Descriptive Analyses

Bivariate associations among the stress indicators were statistically significant and positive: chronic stress, traumatic stress, $r = 0.38$, $p < .001$; chronic stress, perceived stress, $r = 0.38$, $p < .001$; and traumatic stress, perceived stress, $r = 0.19$, $p < .001$. As shown in Table 1, the majority of the sample was at least 45 years old (61.4%) and female (62.1%), with individuals of Mexican descent representing the largest ethnic group (39.2%). Most had a household income of less than \$30,000 (66.2%), were not born in the U.S. (82.6%), and completed their interview in Spanish (80.7%). In the target population, CHD (4.2%) and stroke prevalence (1.3%) were both low, whereas prevalences of CVD risk factors were higher.¹ Approximately 75.6% and 80.7% of individuals in the target population reported at least one chronic and traumatic stressor, respectively. Perceived stress was moderate on average (weighted mean = 14.86).

¹Prevalence of CVD and risk factors in the total HCHS/SOL cohort has been reported elsewhere (18).

Associations of Stress Indicators with CHD and Stroke Prevalence

As shown in Table 2, only chronic stress burden was significantly related to CHD and stroke prevalence. In the fully adjusted model, each additional chronic stressor was associated with a 22% increased odds of CHD; for stroke prevalence, each additional chronic stressor was associated with a 26% increased odds of stroke after controlling for sociodemographic characteristics and behavioral health factors. This effect became non-significant ($p = .065$) with control for biobehavioral risk factors.

Associations of Stress Indicators with CVD Risk Factors

Associations of stress indicators with major CVD risk factors are shown in Table 3. In models that adjusted for demographic and behavioral indicators, each additional chronic stressor reported was associated with increased odds of diabetes and hypertension of 20% and 14%, respectively. Perceived stress was associated with a small increase in odds of current smoking (3%), but did not relate to other CVD risk factors. For traumatic stress, the expected positive association was found with smoking, so that each additional traumatic stressor reported was associated with a 15% increased odds of current smoking. However, in models that adjusted for demographic and behavioral indicators, each additional traumatic stressor reported *decreased* the odds of diabetes and hypertension by 11% and 12%, respectively.

Sensitivity Analyses

Sensitivity analyses identified no statistically significant interaction effects for prevalence of CHD, stroke, diabetes, or dyslipidemia (all $ps > .05$). The analysis for hypertension identified a significant age by chronic stress interaction (OR = 0.706, 95% CI .60–0.82, $p < .001$), but no other interaction effects. Age stratified analyses showed that the association of chronic stress with hypertension prevalence was statistically significant in the 18–44 year-old age group (OR=1.40, 95% CI 1.21–1.61, $p < .001$), but not in the 45 and older group (OR=0.98, 95% CI 0.88–1.10).

Discussion

The current study is among the first to examine the association of stress with CVD prevalence and risk factors in U.S. Hispanics/Latinos. The study also adds to the literature concerning stress and CVD by examining several common indicators of stress within a single methodological framework. As expected, chronic stress was consistently associated with prevalence of CHD and stroke, and major CVD risk factors including diabetes, hypertension, and smoking. These findings concur with theoretical models suggesting that it is protracted exposure to stress over time that is most harmful to health (38). In contrast, perceived stress related to current smoking only. Most surprisingly, traumatic stress showed an *inverse* relationship with diabetes and hypertension, yet was associated with higher odds of smoking. Associations of stress with CVD prevalence and risk factors were generally consistent across age and sex groups, with the exception of a weaker (and non-significant) association of chronic stress with hypertension in older than in younger participants. Observed associations were generally small in magnitude, but robust to adjustment for potential confounding influences (e.g., sociodemographic factors) and indirect pathways

(e.g., health behaviors; traditional risk factors) through which stress may relate to CVD. For example, each additional chronic stressor reported predicted a 22% and 23% higher odds of CHD and stroke, respectively, in fully controlled models. By way of comparison, in the overall HCHS/SOL cohort, ORs for the association of traditional risk factors with CHD after controlling for demographic, behavioral, and other biological risk factors ranged from 1.3 (95% CI 1.0–1.8, NS) for obesity, to 1.6 (95% CI, 1.2–2.2) for dyslipidemia, 1.7 (95% CI 1.3–2.3) for diabetes, 1.7 (95% CI 1.2–2.5) for smoking, and 1.9 (95% CI 1.4–2.6) for hypertension. For stroke, risk factor effects were smaller, and significant only for hypertension, OR=1.9 (95% CI 1.2–2.8) and diabetes OR=1.9 (95% CI 1.3–2.8) (18). The additional predictive utility of chronic stress is modest, though likely to be clinically significant at the population level.

Prior reviews have concluded that chronic stress in important life domains predicts a moderate (40–60%) elevation in risk of CHD over time, though measures (work stress, social isolation) and control for potential confounding or intervening factors has varied widely (4, 5). The research connecting stress with stroke has produced mixed findings, with some studies identifying a relationship [e.g., (39, 40)] and others reporting inconclusive findings (41). Some studies have also linked stress (variously defined) with traditional risk factors such as diabetes (42–44), hypertension (45, 46), dyslipidemia (47–49), and smoking (50, 51). Well-established risk factors may represent an intermediate stage through which stress increases CVD morbidity and mortality, although the association of chronic stress with CHD remained statistically significant after adjustment for these risk factors. Likewise, prior studies suggest that established risk factors do not account completely for the stress-CVD association (5). It is also notable that control for intermediate behavioral health indicators resulted in only small changes in the strength of associations of chronic stress with CHD, stroke, and traditional risk factors in the current study. The pathways connecting stress with CVD are complex and involve a variety of interrelated physiological, behavioral, and emotional pathways (1, 2, 52).

Given the cross-sectional design, observed associations of chronic stress with CHD and stroke are likely to be bidirectional. Stress could contribute to the development of these conditions, but individuals with CVD may also experience increased stress due to health problems. Chronic stress was associated with CVD risk factors in participants free from prevalent CVD; however, having a complex condition such as diabetes, and to some extent hypertension, could foster chronic stress. Thus, even at subclinical stages, atherosclerosis and major CVD risk factors could contribute to a reverse causation bias that inflates observed effects of chronic stress (53). It will be important to examine the association of chronic stress with incident CVD when follow-up data for the HCHS/SOL cohort become available.

A recent review and meta-analysis examining effects of perceived stress identified a modest increase in incident CHD (OR = 1.27, 95% CI 1.12, 1.45) across six studies that primarily examined European or Asian populations (6). Several of these studies measured perceived stress using only 1–2 general items and none controlled for other types of stress. The current study is the first, to our knowledge, to examine perceived stress and CVD prevalence and risk among Hispanics/Latinos. Overall, the proximal model of stress captured by the

perceived stress scale (past month) may be less clearly related to a protracted disease process such as CVD. In contrast, effects of perceived stress on an unhealthy coping behavior (smoking) could be more easily ascertained in the short term.

The observed “protective” associations of traumatic stress with biological CVD risk factors—which persisted with control for demographic factors and behavioral pathways—were unexpected. Several prior studies have shown that individuals reporting past traumatic events evidenced an increased risk of incident CHD (54–56); however, other studies suggest that health risks occur only when post-traumatic stress disorder follows trauma (57). Nonetheless, inverse effects of stressful life events with health have been observed in other studies. For example, being placed into care outside the home as a child, an indicator of household dysfunction, related to a reduced incidence of CHD in men, but not women, in the Copenhagen City Heart Study (58). Melamad and colleagues (59) found that a greater occurrence of (mostly negative) life events predicted lower levels on physiological risk indicators (blood pressure, triglycerides, uric acid) in Israeli men, whereas life events predicted an increased risk of smoking and other behavioral risks. It is possible that individuals who successfully manage traumatic stress develop adaptive strategies for coping with future stressors that protect against related psychological and physical health problems (60, 61). In addition, personal and interpersonal resources could modify the association between major life events and CHD (13). Nonetheless, the positive link between traumatic stress and smoking and the expected positive associations among all types of stress suggest that the impact of personal robustness may be modest. A final factor that may have contributed to these unexpected findings is limitations of the checklist assessment approach, which may include error due to recall and response biases, and differences in event interpretation (11, 62). Further, the traumatic stress scale (32) examines event occurrence, but not stress reactions, which are likely to vary significantly across individuals and time. Thus, inaccuracies in conceptualizing the trauma or a failure to consider stress responses and their duration may have contributed to unexpected findings, though such errors would be more likely to bias the findings toward the null. It should also be noted that analyses were repeated examining effects of traumatic stress alone, and results were consistent with those from models that included all stress indicators. Pending availability of follow-up data on the HCHS/SOL cohort (with a second exam planned for 2015–2018), future research in the cohort examining associations of traumatic events with incident disease outcomes, and with risk factor progression, may help shed light on these surprising results.

The current study has several strengths, including the large, diverse Hispanic sample, which is representative of the Hispanic/Latino population in the recruitment communities (22). The clearest limitation of the current study is the cross-sectional framework, which prohibits conclusions regarding temporality or causality. Thus, results should be interpreted cautiously, and as noted, it is likely that stress-CVD prevalence and risk factor associations are bidirectional. The inclusion of multiple measures of stress is a strength of the study since – as observed here – the different approaches exhibit only small-moderate associations and each may show distinct associations with CVD risk factors and events. However, all stress types were assessed by self-report and the checklist approach to assessing trauma is not optimal. In addition, this study did not address culturally driven forms of stress (e.g., discrimination, acculturation stress), which could be particularly relevant in this cohort. The

self-report assessment of stroke and some aspects of CHD could result in under-reporting and biases related to healthcare access, which could in turn underestimate effect sizes for stress variables (18). Finally, the study adopted a relatively thorough approach to controlling for potential sociodemographic confounds, to better isolate the effects of stress beyond these known correlates of stress and CVD. However, this approach may have underestimated effects of chronic stress relative to other studies that did not include such controls. Indeed, low socioeconomic adversity has been used as a proxy for stress exposure in some studies (5).

Although Hispanics/Latinos are the largest and fastest growing ethnic minority population in the U.S. (15), very little research has examined the extent to which stress and other psychosocial factors relate to their health (63). Thus, the current study addressing the associations of chronic, perceived, and traumatic stress with CVD prevalence and risk factors provides an important—albeit preliminary—contribution to the literature. The indication that Hispanics/Latinos with greater chronic stress evidence higher rates of CVD and related risk factors, while both perceived and traumatic stress relate to smoking, suggests that stress may be a potential target for intervention strategies in this population. A review of multifactorial CVD risk reduction interventions with a stress management component found evidence of a reduced cardiac event rate, and a decrease in composite CVD risk scores; effects on individual intermediate markers (e.g., blood pressure, lipids) were less clear (64). Recent meta-analyses have concluded that transcendental meditation is an effective stress management approach for reducing blood pressure (65) and decreasing mortality in persons with hypertension (66). Although studies examining stress reduction programs in Hispanics/Latinos are limited, a recent study found that a culturally relevant, peer-led coping skills training program reduced stress and depression among recent Hispanic/Latino immigrants (67). Another found that a culturally contextualized cognitive behavioral stress management intervention improved quality of life in Hispanic/Latino prostate cancer survivors (68). These stress interventions were adapted to incorporate a strong emphasis on traditional cultural values of social interaction and family relationships. Additional research is needed to determine the most effective and culturally appropriate approaches to reducing stress in Hispanics/Latinos and their utility in decreasing CVD risk. Future prospective research that substantiates the current findings will provide the necessary impetus for such efforts.

Acknowledgments

The Hispanic Community Health Study/Study of Latinos was carried out as a collaborative study supported by contracts from the National Heart, Lung, and Blood Institute (NHLBI) to the University of North Carolina (N01-HC65233), University of Miami (N01-HC65234), Albert Einstein College of Medicine (N01-HC65235), Northwestern University (N01-HC65236), and San Diego State University (N01-HC65237). The following Institutes/Centers/Offices contribute to the HCHS/SOL through a transfer of funds to the NHLBI: National Institute on Minority Health and Health Disparities, National Institute on Deafness and Other Communication Disorders, National Institute of Dental and Craniofacial Research, National Institute of Diabetes and Digestive and Kidney Diseases, National Institute of Neurological Disorders and Stroke, Office of Dietary Supplements. The HCHS/SOL Sociocultural Ancillary Study was supported by grant 1 RC2 HL101649 from the NIH/NHLBI (Gallo/Penedo PIs). The authors thank the staff and participants of HCHS/SOL and the HCHS/SOL Sociocultural Ancillary Study for their important contributions.

Abbreviations

BMI	Body Mass Index
CHD	Coronary Heart Disease
CVD	Cardiovascular Disease
GED	General Education Degree
HCHS/SOL	Hispanic Community Health Study/Study of Latinos
HS	High School
OGTT	Oral Glucose Tolerance Test

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Table 1

Descriptive statistics for sample demographic characteristics, cardiovascular disease and risk factor prevalence, and stress variables: HCHS/SOL Sociocultural Ancillary Study (overall N=5313)

Variable ^I	N	%	Weighted % (95% CI)
Demographic Characteristics			
Age (N=5313)			
18–44 years	2035	38.3	56.5 (54.2–58.7)
45–74 years	3278	61.7	43.5 (41.3–45.8)
Female (N=5313)	3299	62.1	52.9 (54.9–56.8)
Hispanic/Latino background (N=5309)			
Central American	553	10.4	7.6 (6.2–9.2)
Cuban	775	14.6	20.3 (16.3–25.1)
Dominican	534	10.1	11.7 (9.9–13.8)
Mexican	2080	39.2	36.5 (32.6–40.6)
Puerto Rican	880	16.6	15.8 (13.8–18.0)
South American	350	6.6	4.8 (4.0–5.8)
More than one	137	2.6	3.3 (2.4–4.5)
Study Site (N=5313)			
Bronx, NY	1342	25.3	30.2 (26.4–34.4)
Chicago, IL	1329	25.0	15.6 (13.2–18.4)
Miami, FL	1315	24.8	29.1 (23.8–35.1)
San Diego, CA	1327	25.0	25.0 (21.2–29.2)
Education: < HS diploma or GED (N=5129)	1862	35.0	32.6 (30.4–34.9)
Household income: < \$30K/yr (N=4799)	3515	66.2	70.1 (67.3–72.8)
Nativity/immigration: Born in U.S. Mainland (N=5230)	909	17.4	22.0 (19.7–24.5)
Language of Interview: Spanish (N=5233)	4225	80.7	75.3 (72.4–78.0)
Cardiovascular Disease/Risk Factor Prevalence			
CHD prevalence (N=5232)	249	4.8	4.2 (3.6–5.0)
Stroke prevalence (N=5230)	77	1.5	1.3 (1.0–1.7)
Diabetes (N=5313)	920	18.7	14.3 (13.1–15.7)
Hypertension (N=5313)	1295	26.3	21.6 (19.7–23.5)
Dyslipidemia (N=5308)	1931	39.2	37.6 (35.4–39.8)
Current smoker (N=5308)	891	18.1	20.6 (18.6–22.7)
Stress Indicators (Weighted Data)			
	Mean	SD	Possible Range
Chronic stress (N=5178)	1.81	1.66	0–8
Perceived stress (N=5176)	14.86	6.85	0–40
Traumatic events stress (lifetime) (N=5250)	2.11	1.72	0–10

^I Individual Ns vary due to missing data

Table 2 Associations between stress indicators and CHD and stroke prevalence: the HCHS/SOL Sociocultural Ancillary Study

Stress Indicator	CHD OR (95% CI)			Stroke OR (95% CI)		
	Model 1 ¹	Model 2 ²	Model 3 ³	Model 1 ¹	Model 2 ²	Model 3 ³
Perceived Stress	0.99 (0.96–1.01)	0.99 (0.96–1.02)	0.99 (0.97–1.02)	0.95 (0.90–1.01)	0.96 (0.91–1.01)	0.96 (0.91–1.02)
Chronic Stress	1.28* (1.16–1.42)	1.25* (1.13–1.37)	1.22* (1.10–1.36)	1.26* (1.02–1.56)	1.26* (1.03–1.55)	1.23 (0.99–1.52)
Traumatic Stress	0.98 (0.88–1.09)	1.00 (0.90–1.10)	1.03 (0.93–1.15)	1.03 (0.85–1.25)	1.05 (0.86–1.28)	1.09 (0.88–1.34)

OR = odds ratio; 95% CI = 95% confidence interval.

* p<.05.

¹ Controls for age, sex, Hispanic/Latino background, nativity/immigration, language of interview, income, education

² Controls for demographic covariates, as well as BMI, physical activity, smoking, alcohol consumption.

³ Controls for all variables in Models 1 and 2, as well as diabetes, hypertension, dyslipidemia.

Associations between stress indicators and cardiovascular risk factors: the HCHS/SOL Sociocultural Ancillary Study (N=4926)

Table 3

Stress Indicator	Diabetes OR (95% CI)		Hypertension OR (95% CI)		Dyslipidemia OR (95% CI)		Current Smoker OR (95% CI)	
	Model 1 ¹	Model 2 ²	Model 1 ¹	Model 2 ²	Model 1 ¹	Model 2 ²	Model 1 ¹	Model 2 ²
Perceived Stress	0.99 (0.97–1.01)	0.99 (0.97–1.01)	0.99 (0.97–1.01)	0.99 (0.98–1.01)	0.99 (0.98–1.00)	0.99 (0.98–1.00)	1.03* (1.01–1.05)	
Chronic Stress	1.25* (1.16–1.34)	1.20* (1.11–1.31)	1.14* (1.06–1.23)	1.10* (1.02–1.19)	1.03 (0.98–1.09)	1.00 (0.95–1.06)	0.95 (0.88–1.02)	
Traumatic Stress	0.87* (0.81–0.94)	0.89* (0.83–0.97)	0.86* (0.81–0.92)	0.88* (0.82–0.93)	0.96 (0.91–1.01)	0.97 (0.92–1.02)	1.15* (1.05–1.26)	

OR = odds ratio; 95% CI = 95% confidence interval.

* p<.05

¹ Controls for age, sex, Hispanic/Latino background, nativity/immigration, language of interview, income, education

² Controls for demographic covariates, as well as BMI, physical activity, smoking, alcohol consumption