UC San Diego

UC San Diego Electronic Theses and Dissertations

Title

The impact of neighborhood stress on cardiovascular health : : the association between perceptions of neighborhood physical and social disorder and cardiovascular disease risk in Latinas

Permalink

https://escholarship.org/uc/item/2dz3z57q

Author

Shivpuri, Smriti

Publication Date

2013

Peer reviewed|Thesis/dissertation

eScholarship.org Powered by the California Digital Library
University of California

UNIVERSITY OF CALIFORNIA, SAN DIEGO

SAN DIEGO STATE UNIVERSITY

The Impact of Neighborhood Stress on Cardiovascular Health: The Association between Perceptions of Neighborhood Physical and Social Disorder and Cardiovascular Disease Risk in Latinas

A dissertation submitted in partial satisfaction of the
requirements for the degree Doctor of Philosophy

in

Clinical Psychology

by

Smriti Shivpuri

Committee in charge:

University of California, San Diego

Professor Matthew A. Allison
Professor Paul J. Mills

San Diego State University

Professor Linda C. Gallo, Chair
Professor John P. Elder
Professor Scott C. Roesch

2013

Copyright

Smriti Shivpuri, 2013

All rights reserved.

The Dissertation of Smriti Shivpuri is approved, and it is acceptable in quality and form for publication on microfilm and electronically:

_____

_____

_____

_____

_____
Chair

University of California, San Diego

San Diego State University

2013

TABLE OF CONTENTS

Signature Page iii

Table of Contents iv

List of Tables vi

List of Figures vii

Acknowledgements viii

Vita ix

Abstract xi

Chapter 1: Background and Rationale 1

Neighborhood Disorder as a Stress Process 2

Subjective Perceptions of Neighborhood 2

Theoretical Underpinnings of Neighborhood Disorder and CVD 6

Pathophysiology of Chronic Stress and CVD Risk 7

Perceptions of Neighborhood Disorder and Cardiovascular Disease 9

Cardiovascular Disease Risk Factors 9

Cardiovascular disease endpoints. 13

Limitations in the Literature on Perceptions of Neighborhood Disorder and CVD Risk 14

Chapter 2: Current Study 20

Specific Aims 24

Chapter 3: Methods 28

Participants and Recruitment 28

Procedures 29

Measures 30

Cardiovascular Disease Risk Factors....30
Body composition. 30
Insulin resistance....30
Lipids. 30
Blood pressure. 31
Neighborhood Disorder 31
Behavioral Factors Related to CVD Risk 32
Psychological Distress 34
Covariates 36
Neighborhood SES....37
Statistical Analyses 38
Power Analysis 42
Chapter 4: Results 43
Descriptive Statistics and Bivariate Associations....43
Direct Effects of Neighborhood Physical and Social Disorder on CVD Risk Factors....43
Indirect Effects of Neighborhood Physical and Social Disorder on CVD Risk Factors through Behavioral Pathways 49
Indirect Effects of Neighborhood Physical and Social Disorder on CVD Risk Factors through Psychological Pathways....49
Neighborhood Disorder-CVD Risk Factor Associations by Levels of Neighborhood SES....51
Chapter 5: Discussion 53
Limitations and Future Research Directions....65
Conclusions....69
References....71

LIST OF TABLES

Table 1. Descriptive Statistics for Covariates, Neighborhood Physical and Social Disorder, and CVD Risk Factors 44

Table 2. Descriptive Statistics for Proposed Behavioral and Psychological Mediators, and Correlations of Mediators with CVD Risk Factors 46

LIST OF FIGURES

Figure 1. Model of Associations between Neighborhood Disorder and CVD Risk Factors. 25

Figure 2. Results of analyses examining direct pathways from neighborhood physical and social disorder to CVD risk factors. 48

Figure 3. Results of confirmatory factor analysis investigating a one-factor model of psychological distress. 50

ACKNOWLEDGEMENTS

I would like to dedicate this dissertation to people in my life who have been integral in the completion of my long journey to attaining a doctoral degree. These include my parents, Rajiv and Sushma Shivpuri, who have provided support (both monetary and emotional), encouragement, and love throughout my many years of schooling, and who are now extremely relieved that it is over! My sisters, Shruti Fernandes and Shubhra Shivpuri, and brother-in-law, Ashley Fernandes, who have all been very nurturing, caring, reassuring, and supportive and who have served as excellent role models for the rewards that come with hard work and perseverance. My two little nephews, Arav and Mithran, who have been a welcome source of amusement and distraction from the mundane work of dissertation writing.

Acknowledgement also goes to my advisor and chair, Linda Gallo, who has been a tireless advocate for me, as well as all her students, and whose brilliant insight, feedback, and attention to detail has helped me develop into a much improved writer and researcher, and whose support and commitment has been integral to my success. I would like to thank my colleagues, in particular, Addie Fortmann, Karla Espinosa de los Monteros, Wendy Davila and Patty Gonzalez, who have all in one capacity or another, helped me throughout this journey. Finally, I would like to acknowledge the monetary and training support I have received from the T32 predoctoral training fellowship in Cardiovascular Epidemiology (1T32HL079891-01A2 from the National Heart Lung and Blood Institute, National Institutes of Health [NHLBI/NIH]), as well as Dr. Matt Allison and Dr. Michael Criqui, the T32 training directors in the Department of Family and Preventative Medicine at UCSD.

VITA

2002	Bachelor of Science, Ohio State University
2005	Master of Arts, Michigan State University
2012	Master of Public Health, San Diego State University
2013	Doctor of Philosophy, San Diego State University/University of California, San Diego

PUBLICATIONS

Friede, A., Oswald, F. L., Schmitt, N., Drzakowski, S., Imus, A., Kim, B., & Shivpuri, S. (2009). Estimating trait and situational variance in a situational judgment test. *Human Performance, 22*, 44-63.

Gallo, L. C., Espinosa de los Monteros, K., & Shivpuri, S. (2009). Socioeconomic status and health: What is the role of reserve capacity? *Current Directions in Psychological Science, 18*(5), 269-274.

Gallo, L. C., Fortmann, A. L., Roesch, S. C., Barrett-Connor, E., Elder, J. P., Espinosa de los Monteros, K., … Matthews, K. A. (2012). Socioeconomic status, psychosocial resources and risk, and cardiometabolic risk in Mexican-American women. *Health Psychology, 31*(3), 334-342. doi: 10.1037/a0025689

Gallo, L. C., Jimenez, J. A., Shivpuri, S., Espinosa de los Monteros, K., & Mills, P. J. (2011). Domains of chronic stress, lifestyle factors, and allostatic load in middle-aged Mexican-American women. *Annals of Behavioral Medicine, 41*, 21-31.

Gallo, L. C., Shivpuri, S., Gonzalez, P., Fortmann, A. L., Espinosa de los Monteros, K., & Matthews, K. A. (in press). Socioeconomic status and stress in Mexican American women: A multi-method perspective. *Journal of Behavioral Medicine*.

Jiménez, J. A., Shivpuri, S., Espinosa de los Monteros, K., Matthews, K. A., Mills, P. J., & Gallo, L. C. (in press). Associations between socioeconomic status and catecholamine levels vary by acculturation status in Mexican-American women. *Annals of Behavioral Medicine*. doi: 10.1007/s12160-012-9365-6

Schmitt, N., Oswald, F. L., Kim, B. H., Drzakowski, S., Friede, A., Shivpuri, S. (2007). The use of background and ability profiles to predict college student outcomes. *Journal of Applied Psychology, 92*, 165-179.

Shivpuri, S., & Kim, B. (2004). College student development and assessment in today's world: Do employers and college administrators see eye to eye? *National Association of Colleges and Employers Journal, 65*, 37-44.

Shivpuri, S., Gallo, L. C., Crouse, J. C., & Allison, M. (2012). The association between chronic stress type and c-reactive protein in the Multi-Ethnic Study of Atherosclerosis (MESA): Does gender make a difference? *Journal of Behavioral Medicine, 35*, 74-85. doi: 10.1007/s10865-011-9345-5

Shivpuri, S., Gallo, L. C., Mills, P. J., Matthews, K. A., Elder, J. P., & Talavera, G. A. (2011). Trait anger, cynical hostility and inflammation in Latinas: Variations by anger type? *Brain, Behavior, and Immunity, 25*, 1256-1263. doi: 10.1016/j.bbi.2011.04.016

Shivpuri, S., Schmitt, N., Oswald, F. L., & Kim, B. (2006). Individual differences in academic growth: Do they exist and can we predict them? *Journal of College Student Development, 47*, 69-86.

ABSTRACT OF THE DISSERTATION

The Impact of Neighborhood Stress on Cardiovascular Health: The Association between Perceptions of Neighborhood Physical and Social Disorder and Cardiovascular Disease Risk in Latinas

by

Smriti Shivpuri

Doctor of Philosophy in Clinical Psychology

University of California, San Diego, 2013
San Diego State University, 2013

Professor Linda C. Gallo, Chair

Attention has been paid to "objective" characteristics but not "subjective" perceptions of neighborhood environments and associations with cardiovascular disease (CVD) risk; the latter may be of particular import since one of the ways in which impoverished neighborhood environments are theorized to impact health is through their roles as daily sources of stress. Few studies have examined how neighborhood

characteristics impact CVD risk within Latinos. The current study examined the relationship between perceptions of neighborhood physical and social disorder and CVD risk as assessed by body mass index (BMI), the Homeostasis Model of Assessment – Insulin Resistance (HOMA-IR), total to high-density lipoprotein cholesterol ratio (total/HDL-c), triglycerides, and mean arterial blood pressure (MAP). Indirect effects through behavioral (dietary patterns, sedentary behavior, exercise, smoking, sleep quality/quantity) and psychological (chronic stress, perceived stress, anxiety, depression) factors were also examined. A sample of 304 healthy, Mexican-American women, aged 40-65 years, were recruited from south San Diego communities of diverse SES. Participants completed questionnaires assessing demographics, health behaviors and neighborhood disorder. Trained assessors measured blood pressure and obtained anthropometric measurements. Levels of lipids, insulin, and glucose were collected through a fasting venous blood draw. Findings showed no significant relationship between physical disorder and CVD risk factors in either age-adjusted or fully-adjusted (controlling for SES, acculturation, menopausal status, and medications relevant to CVD risk factors) models. Social disorder was significantly associated with total/HDL-c ratio, triglycerides, and MAP and were marginally associated with HOMA-IR (p=.058). No associations remained significant in fully-adjusted models. In addition, no indirect effects through behavioral or psychological pathways were significant. Sensitivity analyses revealed an interaction between social disorder and neighborhood SES on MAP that approached significance (p= .053). Simple slope analyses showed a trend for a stronger association between social disorder and MAP with increasing neighborhood SES. In sum, results provide preliminary evidence of associations between greater

perceived social disorder in one's neighborhood and CVD risk through increased lipids and blood pressure in Latino women. However, these effects may be due to confounding with SES. Findings contribute to the understanding of how appraisals of macro-level influences relate to CVD risk within the Latino population.

Chapter 1: Background and Rationale

Cardiovascular disease (CVD) is the leading cause of mortality within the United States (Heron et al., 2009). Interventions to reduce CVD risk and promote health have historically been targeted at the individual level, and research has focused on risk factors that are modifiable through individual behavior change. However, with the introduction of the social-ecological model of health promotion (Stokols, 1996), attention has shifted to a greater appreciation of the multiple levels through which individual health is impacted, including the influence of environmental factors on CVD risk. The social-ecological model stresses the importance of an individual's surrounding social and physical environment when considering factors that influence health outcomes. Residential environments (e.g., "neighborhoods") have been the focus of increasing research, due in part to recognition of the frequency with which individuals are exposed to these environments, and an awareness of their physical and social characteristics that could impact health (Diez Roux, 2007).

The majority of work on the impact of neighborhoods on CVD risk has examined associations between neighborhood socioeconomic status (SES) or deprivation, and CVD risk factors and endpoints. Neighborhood SES has been associated with behavioral and biological risk factors for CVD risk including physical activity, smoking, obesity, hypertension, and cholesterol (Cubbin et al., 2006; Cubbin & Winkleby, 2005; Leal, Bean, Thomas, & Chaix, 2011). Greater neighborhood deprivation has also been linked with subclinical indicators of heart disease (Murray et al., 2010; Nordstrom, Diez Roux, Jackson, & Gardin, 2004). Lower neighborhood SES has been related to a higher incidence of myocardial infarction (MI), and heart disease mortality in several studies

(Chaix, Rosvall, & Merlo, 2007a; Deguen et al., 2010; Diez Roux et al., 2001; Foraker et al., 2011; Rose et al., 2009; Stjarne, Fritzell, De Leon, & Hallqvist, 2006; Winkleby, Sundquist, & Cubbin, 2007). Lower neighborhood SES has also been related to worse outcomes, including shorter survival and increased risk of recurrent events, in patients recovering from acute coronary events (Chaix, Rosvall, & Merlo, 2007b; Gerber, Benyamini, Goldbourt, & Drory, 2010; Gerber, Koton et al., 2010). Indeed, a recent systematic review on the association between geographic life environments (with residential environments being the focus of 90% of studies reviewed) and risk factors related to CVD reported that a majority of the studies assessed for an association between a sociodemographic factor (of which SES was the most widely researched) and CVD risk factors (Leal & Chaix, 2011). Thus, although a great deal of research attention has been directed toward evaluating relationships between neighborhood SES and cardiovascular outcomes, few studies have examined other characteristics of neighborhoods.

Neighborhood Disorder as a Stress Process

Subjective Perceptions of Neighborhood

Although there has been substantial work linking "objective" markers of neighborhood characteristics with poorer cardiovascular profiles and increased CVD rates, much less attention has been devoted to associations of "subjective" perceptions of neighborhood physical and social environments, such as ratings of neighborhood disorder, with health risk. In the systematic review on geographic life environments and CVD risk factors mentioned earlier, the authors noted that out of the studies reviewed, the vast majority (84%) used either administratively-defined measures of neighborhood characteristics (e.g., census-based sociodemographic information) or geographic

information systems-related data (e.g., straight-line distance to the nearest green space), and only 7% examined individuals' perceptions of the neighborhood environment (Leal & Chaix, 2011). This may be in part due to concern that perceptions are not an accurate reflection of "true" neighborhood characteristics and may be subject to individual biases. However variability in perceptions may reflect differences in experienced environments, which may be relevant to understanding effects of environments on health (Diez Roux, 2007).

Moreover, subjective perceptions of neighborhood characteristics may be of particular import, as one of the main ways in which impoverished neighborhood environments are theorized to impact health is through their role as chronic, daily sources of stress. It is posited that disadvantaged environments contain normative climates that promote unlawful behavior, lack of intact family structures, and little human capital or social cohesion, all of which lead to a breakdown in neighborhood orderliness, or engender increased "neighborhood disorder" (Ross & Mirowsky, 2001). Moreover, these types of neighborhoods display observable signs and cues that social control is weak, and therefore signal a greater likelihood of physical and social harm. These signs include aspects of *social* disorder such as loitering, crime, drug and alcohol use, trouble with neighbors, lack of trust in neighbors, lack of safety, and *physical* disorder such as graffiti, filth, noise, vandalism, and lack of upkeep (e.g., dilapidated and abandoned buildings). Consequently, aspects of crime, lack of safety, substance use and poor social relations with neighbors are all components of social disorder and can be sources of ongoing social stress. Similarly, aspects related to structural and environmental deterioration, ambient noise, destruction of property, and lack of cleanliness are subsumed within physical

disorder and can also be sources of daily stress. In other words, residents of disordered neighborhoods are exposed to noxious and threatening environments that can be daily sources of chronic physical and mental stress (Hill, Ross, & Angel, 2005).

Subjective appraisals of neighborhood disorder may be of notable significance when evaluating the effects of neighborhood disorder on health over and above objective characteristics such as neighborhood and individual SES. According to the transactional theory of stress (Lazarus & Folkman, 1984), external stressors interact with an individual's cognitive processes to influence health outcomes. In other words, an individual's recognition and appraisal of a stressor as threatening is critical in determining its emotional and physiological effects. If neighborhood disorder is conceptualized as a type of chronic stressor that impacts health, then considering perceptions of neighborhood disorder, rather than simply assessing objective characteristics of disorder, should be particularly relevant. For example, research suggests that perceptions of crime may be more predictive of health behaviors such as physical activity than actual crime levels (Foster & Giles-Corti, 2008). Moreover, others have argued that subjective assessments better capture the holistic and integrated effects of neighborhood environments on health (Stronegger, Titze, & Oja, 2010), and that they are more direct assessments of stress processes than objective indicators, which are proxy indicators of stress (Mulia, Schmidt, Bond, Jacobs, & Korcha, 2008).

Although it has generally been posited that lower neighborhood SES leads to poorer neighborhood perceptions (Ross & Mirowsky, 2001), there is some evidence that objective conditions and subjective perceptions may differ, and consequently, subjective perceptions may provide unique information about neighborhood disorder not contained

in objective assessments. For example, Keita, Cassazza, Thomas and Fernandez (2011) created an index of neighborhood deprivation using objective census-tract indicators of disadvantage including percentages of unemployment, poverty, female-headed households, and vacant housing, and also obtained subjective ratings of neighborhood disorder from parents of children in neighborhoods of diverse ethnic composition with European, African, and Hispanic-American residents. Results revealed that there was little variability across neighborhoods on the objective index of neighborhood deprivation (i.e., less than one-tenth of a point), indicating that the neighborhoods included in the study were essentially similar in terms of SES levels. However, respondents' ratings of neighborhood disorder differed markedly, with a little more than half of the respondents rating their neighborhoods as high on disorder and a little less than half rating their neighborhoods as low on disorder. Consequently, although objective markers indicated neighborhoods were similar in terms of disorder, subjective perceptions showed substantial variability. Similarly, Steptoe and Feldman (2001) reported that in their sample of 658 British residents there was variability in perceptions of neighborhood disorder, even amongst neighborhoods of similar SES (as defined by national geodemographic postcode classifications of the occupational status of postal sectors), and that only 11% of variability in perceptions of disorder was accounted for by neighborhood SES. Furthermore, research has shown that subjective perceptions of neighborhood disorder, although largely influenced by objective neighborhood characteristics, are also determined by individual sociodemographic factors such as age, ethnicity, nativity and length of residence (Elo, Mykyta, Margolis, & Culhane, 2009; Latkin, German, Hua, & Curry, 2009; Sampson & Raudenbush, 2005). This underscores

the notion that experiences of neighborhood stress or disorder may vary based on an individual's normative experiences and background, and consequently, similar objective markers of disorder may not translate into equivalent perceptions of subjective disorder.

Theoretical Underpinnings of Neighborhood Disorder and CVD

At least two theoretical frameworks exist that specifically model the relationship between aspects of physical and social residential environments and CVD risk, and both outline stress as a pathway by which neighborhood characteristics affect cardiovascular outcomes. Diez-Roux (2007) proposes a model in which aspects of the neighborhood physical and social environment impact "proximate biological factors" (i.e., major CVD risk factors), including blood pressure (BP), body mass index (BMI), diabetes, and lipids, which in turn, lead to clinical CVD. Stress is included in two phases of the model. Stress is shown to be a mediating mechanism between certain aspects of physical disorder in the environment, such as aesthetic quality and noise, and aspects of social disorder, such as safety/violence and social support/cohesion, and the "proximate biological factors". It is also included as a "proximate biological factor" itself in the form of a biological "stress response" that is impacted through the mediating mechanisms of health behaviors (e.g., smoking, diet, sleep) and psychological stress. Similarly, Chaix (2009) portrays the effect of neighborhood physical (e.g., building appearance, decay, aesthetic quality) and social (e.g., strong ties to neighbors, neighborhood identities) environments as impacting coronary heart disease in part through the intermediate influence of stress. These neighborhood environments are shown to operate through their effects on "experiential factors", in other words, by affecting the ways in which residents perceive and experience their neighborhood environments. Stress is included as an "experiential" factor, in the

realms of both relational experience (e.g., poor neighborhood environments increasing stressful interactions with neighbors) and psychological distress (e.g., poor neighborhood environments leading to greater affective experiences of stress). Stress, as well as other "experiential" factors, are then shown to affect health behaviors (e.g., physical activity, diet, smoking) and CVD risk factors (e.g., obesity, hypertension, cholesterol), and ultimately CVD outcomes such as atherosclerotic progression, incidence of coronary events, and survival post MI.

Pathophysiology of Chronic Stress and CVD Risk

Based on the conceptualization that neighborhood social and physical disorder are sources of chronic psychological stress, the processes by which they likely impact CVD risk factors and endpoints should be similar to those engendered by other enduring, repeated stressors. Research has generally supported a link between chronic psychosocial stress and increased risk for CVD (Bairey Merz et al., 2002; Chida & Steptoe, 2010; Dimsdale, 2008; Ramachandruni, Handberg, & Sheps, 2004). For example, the INTERHEART study, a multinational case-control study of greater than 25,000 individuals from 52 countries found that, after adjusting for relevant covariates such as age, gender, location, and smoking, individuals who reported chronic stress at work or home had more than double the risk for having an MI as compared to those who did not (Rosengren et al., 2004). Although the exact pathophysiological mechanisms through which chronic stress contributes to increased CVD risk are still poorly understood, it is hypothesized that multiple pathways contribute. Psychological stress is known to activate two systems related to cardiovascular functioning: the sympathetic-adrenal-medullary (SAM) system and hypothalamic-pituitary-adrenal (HPA) axis (Black &

Garbutt, 2002). The activation of these systems may induce functional adaptation of different organs including the heart in the short-term, but in the long-term, is thought to lead to deleterious consequences (Adameova, Abdellatif, & Dhalla, 2009).

SAM system activation results in the release of catecholamines into circulation, and elevated levels of these neuroendocrine markers are believed to directly induce myocardial cell damage and inflammatory activity, as well as stimulate increases in heart rate and BP, which over time can lead to elevation of tonic BP levels and the development of CVD (Adameova et al., 2009; Schwartz et al., 2003). Studies have demonstrated links between exposure to chronic psychosocial stress and elevated BP, including increased risk for hypertension (Dimsdale, 2008). Increases in glycemia, free fatty acids, lipoproteins, triglycerides and cholesterol are also believed to result from activation of the sympathetic nervous system, through the stimulation of adrenoreceptors in various organs of the body (Black & Garbutt, 2002; Vaz Serra, 2002). Activation of the other main system, the HPA axis, causes the release of glucocorticoids including cortisol (Adameova et al., 2009). Elevated levels of cortisol have been linked to increased deposition of fat in abdominal rather than subcutaneous stores, leading to greater central adiposity and increased CVD risk (Miller & O'Callaghan, 2002). Excessive cortisol, whether due to pharmacological treatment or stress, has been shown to produce many factors associated with CVD risk including visceral obesity, insulin resistance, dyslipidemia, dyscoagulation, and hypertension (Miller & O'Callaghan, 2002). The “wear and tear” on multiple physiological systems, including SAM and HPA axis systems, as a result of repeated exposure to chronic stress, and the associated physiological sequelae (i.e., elevations in catecholamines, cortisol, and indicators of CVD

functioning) has been termed "allostatic load" (McEwen, 1998). Allostatic load has been related to chronic stress across various domains (e.g., work, financial, caregiving; Gallo, Jimenez, Shivpuri, Espinosa de los Monteros, & Mills, 2011).

In addition to the direct effects of chronic stress on physiological systems, research has also shown an indirect association with CVD risk via poor health behaviors (Dimsdale, 2008; Schwartz et al., 2003). Greater chronic stress has been associated with the adoption of poorer dietary habits, increased smoking and alcohol use, decreased physical activity, and poorer sleep (Cohen, Schwartz, Bromet, & Parkinson, 1991; Ng & Jeffery, 2003). Notably, poorer "lifestyle" factors (i.e., health behaviors) have been proposed as major mediating mechanisms between chronic stress and indicators of allostatic load, which include many CVD risk factors (e.g., BP, lipids, abdominal obesity; McEwen, 1998). Connections between diet, physical activity, smoking and CVD are well-established, and are often targeted in interventional efforts (Mozaffarian, Wilson, & Kannel, 2008). Sleep quality has also been linked to risk for CVD events, and deprivation is associated with increases in risk parameters including cortisol, BP, glucose, proinflammatory cytokines and obesity (Malhotra & Loscalzo, 2009; Miller & Cappuccio, 2007; Wolk, Gami, Garcia-Touchard, & Somers, 2005).

Perceptions of Neighborhood Disorder and Cardiovascular Disease

Cardiovascular Disease Risk Factors

Whereas the association between objective neighborhood markers of deprivation, such as neighborhood SES, and CVD risk has been relatively extensively explored, fewer studies have examined how subjective perceptions of neighborhood disorder characteristics relate to CVD risk factors and endpoints, and results of the limited

available research have been equivocal. In terms of CVD risk factors, an association between perceptions of neighborhood disorder and BP has been reported in a few studies. For example, perceptions of neighborhood problems, including aspects of disorder such as crime, trash and litter, and poor lighting, were associated with BP control (i.e., having systolic BP < 130, and diastolic BP < 80) in a sample of 7,830 diabetics, such that those in neighborhoods with the highest tertile of problems had poorer control (25% controlled) versus those in the lowest tertile (31% controlled; Gary et al., 2008). Similarly, aspects of neighborhood social disorder, including greater perceived neighborhood safety and cohesion were associated with decreased odds of being hypertensive in a sample of 2612 individuals from the Multi-Ethnic Study of Atherosclerosis (MESA; Mujahid, Diez Roux, Morenoff, et al., 2008). In another study of the MESA cohort, elevated perceptions of neighborhood stress (i.e., physical and social disorder) were associated with greater hypertension prevalence (Mujahid, Diez Roux, Cooper, Shea, & Williams, 2011). However, in both studies, associations were attenuated to non-significance after accounting for race/ethnicity. Notably, this study measured perceptions of neighborhood disorder in a separate sample of individuals who resided in the same neighborhoods (with a median of eight respondents per neighborhood) as the MESA participants, rather than from the participants themselves.

Obesity has been examined in relation to aspects of neighborhood physical and social disorder in a handful of studies. Greater perceptions of neighborhood disorder (as measured by crime, noise, and filth) were related to obesity in a sample of 1338 Texas residents (Burdette & Hill, 2008), with each unit increase in perceived disorder corresponding to a 19% increased odds of obesity. Ratings of social disorder (including

loitering, gang activity, drug or alcohol-related activity, and disruptive/disorderly behavior) were also related to obesity in a sample of 2445 young mothers (Burdette, Wadden, & Whitaker, 2006). Women in neighborhoods with the highest tertile of social disorder had a 9% greater prevalence of obesity than women in neighborhoods with the lowest tertile. However, in another study conducted with the MESA cohort, greater perceived neighborhood social disorder (including aspects of crime, lack of safety and social cohesion obtained from independent raters) was unrelated to obesity in women, and was significantly associated with a decreased risk of obesity in men (Mujahid, Diez Roux, Shen, et al., 2008). Men who resided in environments with less social disorder had a higher mean BMI (0.52, 95% CI = 0.07, 0.97) than those residing in neighborhoods with more disorder. The authors noted that the finding was in contrast to previous studies of adverse social environments and obesity, and stated that residual confounding by individual SES (which was positively associated with BMI in men) could have contributed to results. Interestingly, in one study of 1032 urban residents, ratings of physical disorder including presence of graffiti, litter, broken glass and neighborhood upkeep were unrelated to odds of obesity; however, independent observers' ratings of physical disorder (a more comprehensive assessment, including presence of beer/liquor bottles or cans, cigarette/cigar butts or packages, condoms, drug-related paraphernalia, garbage/litter/broken glass, abandoned cars, graffiti and broken windows) were associated with obesity. Those in neighborhoods in the highest quartile of observer-rated disorder had 4 times the odds of being obese when compared to those in neighborhoods in the lowest quartile of disorder (Boehmer, Hoehner, Deshpande, Brennan Ramirez, & Brownson, 2007).

Two other CVD risk factors that have been explored in studies evaluating the relationship between characteristics of neighborhood disorder and health are inflammatory markers and glucose functioning. Associations of perceptions of neighborhood conditions (including aspects of social disorder such as safety and cohesiveness as well as general assessments of neighborhood pleasantness and satisfaction with neighborhood) with soluble receptors of the pro-inflammatory markers interleukin-6 (IL-6) and tumor necrosis factor alpha (TNF-α), as well as C-Reactive Protein (CRP), and adiponectin (an anti-inflammatory adipokine) were examined in a sample of 368 older African-American residents (Schootman et al., 2010). Neither inflammatory markers nor adiponectin were found to be associated with the perceived neighborhood conditions. However, a study in 5370 individuals from the MESA cohort found an association between perceptions of both neighborhood problems (i.e., aspects of physical disorder including broken sidewalks, trash and litter, excessive noise) and neighborhood safety (i.e., social disorder aspects including violence and crime) with levels of fibrinogen, IL-6, and CRP (Nazmi, Diez Roux, Ranjit, Seeman, & Jenny, 2010). Each standard deviation increase in neighborhood problems was associated with a 2% to 10% increase in the inflammatory markers, and each standard deviation increase in safety (or decrease in social disorder) was related to a 2% to 8% decrease in inflammatory markers; however, these relationships (with the exception of fibrinogen) were attenuated to nonsignificance with controls for ethnicity and behavioral factors. This study also found that higher perceptions of neighborhood problems and lower safety predicted increases in IL-6 across 3-4 years. Finally, neighborhood social disorder (including items related to crime and drug use) was positively associated with fasting plasma glucose

(FPG), and moderated the association between caregiving stress and glucose function (as measured by both FPG and hemoglobin A1c values) in a sample of caregivers and noncaregivers. In the latter regard, the positive association between caregiving stress and glucose functioning was only significant for those in neighborhoods with high levels of social disorder (Brummett et al., 2005).

Cardiovascular disease endpoints. Very few studies have evaluated the relationship between perceptions of neighborhood disorder and CVD indicators, and the few available studies have evaluated perceptions of social disorder only. A measure of social cohesion (including aspects of low social disorder such as trust in neighbors and friendliness of neighbors) was found to be associated with the extent of coronary artery calcification (CAC) measured five years later in a sample of 2974 young to middle-aged adults (Kim, Diez Roux, Kiefe, Kawachi, & Liu, 2010). Among women, being in neighborhoods in the lowest quartile of social cohesion (i.e., highest quartile of social disorder) was associated with almost double the risk (OR=1.87, 95% CI= 1.10, 3.16) of having CAC five years later as compared to those in the highest quartile, and in men, a similar and stronger relationship was apparent, but only for men in low SES neighborhoods (OR=3.77, 95% CI= 1.69, 8.43). Additionally, in a study of 7791 Swedish men and women (Chaix, Lindstrom, Rosvall, & Merlo, 2008), perceptions of neighborhood cohesion (assessed at the individual level and aggregated to the neighborhood level), but not safety, were associated with risk of mortality from acute MI across 3-years; the risk of mortality was three times greater (HR=3.21, 95% CI= 1.36, 9.28) for those individuals in neighborhoods in the lowest versus the highest quartile of cohesion, even after adjusting for individual and neighborhood sociodemographic factors.

Limitations in the Literature on Perceptions of Neighborhood Disorder and CVD Risk

One of the main limitations of the literature on perceptions of neighborhood disorder and CVD risk is the dearth of studies assessing this relationship. As mentioned previously, out of the 131 articles recently reviewed on geographic life environments and risk factors related to CVD, only 7% assessed for any type of perceived neighborhood characteristic, and likely far fewer than this measured perceptions of neighborhood disorder. Although this may be due in part to the fact that information on objective indicators is easier to obtain, or due to the assumption that objective indicators are less prone to biases and "more accurate", if neighborhood environments can be considered as a source of stress, obtaining individuals' perceptions of those environments is important. That objective indicators cannot be used as proxies for subjective experiences of neighborhood environments is underscored by the research demonstrating discrepancies between objective indicators of disorder (e.g., socioeconomic indicators) within a neighborhood and individuals' perceptions of disorder within the same neighborhood (e.g., Keita et al., 2011). By neglecting to investigate the relevance of subjective perceptions of neighborhood disorder in relation to CVD risk, the literature has not yet addressed the importance of appraisals of environmental stress to cardiovascular health.

Moreover, among the studies that do assess either social or physical disorder, many adopt a piecemeal approach, examining either one or two characteristics of disorder (e.g., crime, social cohesion for social disorder or litter and noise for physical disorder) rather than an integrated measure including multiple aspects of social (e.g., crime, lack of safety, substance use, poor social relations) and physical (structural and environmental deterioration, ambient noise, destruction of property, lack of cleanliness) disorder. It

could be that these aspects of disorder have an additive or synergistic effect such that if assessed concurrently, the resultant impact on the stress process or subsequent health outcomes would be clearer. In addition, relatively few studies examine both social and physical environments simultaneously. When examined individually, perceptions of aspects of social disorder were more commonly assessed than physical disorder, which is in contrast to the broader literature on (mainly objectively-assessed) characteristics of residential environments and CVD risk, which focuses more on physical disorder (Leal & Chaix, 2011). This may be because aspects of social disorder are more difficult to obtain objectively. Nonetheless, the examination of both types of disorder will lead to a better understanding of the pathways through which neighborhood environments affect cardiovascular functioning, especially if discrepant relationships are found with specific outcomes.

Additionally, for the majority of CVD risk factors or outcomes discussed (with the exception of obesity) few or no studies have been conducted assessing relationships with neighborhood stress. Furthermore, several of the studies reviewed were sampled from the same cohort of individuals participating in MESA (e.g., Mujahid et al., 2011; Mujahid, Diez Roux, Morenoff, et al., 2008; Mujahid, Diez Roux, Shen, et al., 2008; Nazmi et al., 2010). A few also did not report assessing for clustering by individuals within neighborhoods, and determining the use of single-level or multi-level analyses based on whether significant clustering existed (i.e., Boehmer et al., 2007; Brummett et al., 2005; Burdette & Hill, 2008; Burdette et al., 2006). It is also notable that findings from the extant research are equivocal, with some studies demonstrating effects for an outcome (e.g., Nazmi et al., 2010; for IL-6, CRP), and others no effect for the same

outcome (e.g., Schootman et al., 2010). In addition, in some studies, effects of perceived neighborhood disorder appeared to be explained by sociodemographic factors such as race/ethnicity (e.g., Mujahid et al., 2011; Mujahid, Diez Roux, Morenoff, et al., 2008; Nazmi et al., 2010), or were observed only when the highest and lowest levels of disorder were compared (e.g., Chaix et al., 2008; Kim et al., 2010; Mujahid, Diez Roux, Morenoff, et al., 2008).

Furthermore, although theoretical models incorporate behavioral factors as mediating mechanisms linking neighborhood disorder to CVD risk, and previous work has demonstrated the association between perceptions of neighborhood disorder and health behaviors including physical activity (Mendes de Leon et al., 2009; Poortinga, 2006; Stronegger et al., 2010), smoking (Burdette et al., 2006; Echeverria, Diez-Roux, Shea, Borrell, & Jackson, 2008; Ellaway & Macintyre, 2009), and sleep (Hale, Hill, & Burdette, 2010; Hill, Burdette, & Hale, 2009), few studies have tested whether behavioral factors serve as intermediate pathways between perceptions of neighborhood disorder and CVD risk factors. Exceptions are a study by Burdette and Hill (2008), which found that diet quality (rated from poor to excellent) and exercise (exercising moderately/vigorously or not) partially mediated the association between perceptions of neighborhood disorder and obesity, and a study by Chaix et al. (2008), which found that smoking (but not physical activity) accounted for 9% of the association between aspects of social disorder and acute MI mortality. Similarly, perceptions of disorder have been linked to psychological distress, including reported stress and depression (Gary, Stark, & LaVeist, 2007; Kim, 2008; Kruger, Reischl, & Gee, 2007; Latkin & Curry, 2003; Ross & Mirowsky, 2009). In the Burdette and Hill (2008) study, psychological distress (i.e.,

symptoms of stress and depression) was also tested as a mediator between disorder and obesity, and was found to account for a statistically significant amount (17%) of the shared variance. Another study found evidence for partial mediation by depression of the association between aspects of neighborhood social disorder and CAC in women; however, the statistical significance of indirect effects was not formally tested (Kim et al., 2010). Few other studies have tested whether chronic stress burden or depression serve as mediators between perceptions of disorder and CVD risk factors.

Finally, little attention has been paid to how neighborhood factors are associated with cardiovascular health within Latinos, the largest and most rapidly growing ethnic minority group in the U.S. This is a notable gap in the literature, since Latinos (in particular, Mexican-Americans) have higher rates of cardiometabolic disorders than non-Latino Whites (Ervin, 2009; Rojas et al., 2010). Some evidence suggests that neighborhood characteristics confer important contextual influences on health outcomes in Latinos, although the manner in which these characteristics relate to health remains unclear. For example, greater ethnic residential segregation (i.e., living in neighborhoods with high densities of co-ethnics) has been associated with less cancer incidence and prevalence as well less all-cause mortality in some studies (Bond Huie, Hummer, & Rogers, 2002; Eschbach, Mahnken, & Goodwin, 2005; Eschbach, Ostir, Patel, Markides, & Goodwin, 2004; Keegan, John, et al., 2010), but has also been linked to poorer outcomes (e.g., more advanced stage of cancer diagnosis, larger tumor size; Keegan, Quach, Shema, Glaser, & Gomez, 2010; Reyes-Ortiz, Eschbach, Zhang, & Goodwin, 2008) in others. Similarly, higher neighborhood SES has been linked to positive health outcomes, such as better cancer survival and less tumor thickness (Johnson, Hsiao, Jani,

& Master, 2011; Keegan, Quach, et al., 2010), but has also been predictive of greater cancer incidence (Eschbach et al., 2005; Keegan, John, et al., 2010).

Less work has focused on how neighborhood characteristics influence CVD risk factors and outcomes in Latinos. Greater neighborhood acculturation (as measured by a composite of overall percentage of foreign-born individuals and percentage arrived within 10 years as well as percentage of Spanish-speaking households that reported speaking English less than very well) was found to be unassociated with prevalence of metabolic syndrome in a sample of middle-aged Mexican-American women (Espinosa de los Monteros, Gallo, Elder, et al., 2008). Greater neighborhood residential segregation has been associated with increased obesity rates in a sample of over 80,000 young to middle-aged adult Latino residents of Utah, and this effect was amplified after accounting for immigrant concentration (Wen & Maloney, 2011). In contrast, greater residential segregation was unrelated to hypertension or heart attack prevalence, but was related to decreased stroke prevalence in a sample of 2668 older Mexican-Americans (Eschbach et al., 2004), and decreased 10 year coronary heart disease risk in a sample of middle-aged low-income Latino women (Mobley et al., 2006).

In sum, findings suggest that neighborhood factors such as neighborhood residential segregation, SES, and acculturation may influence health outcomes in Latinos, however results are equivocal regarding whether these neighborhood factors operate in a protective or detrimental capacity. In addition, there is some evidence that relationships between neighborhood factors and health outcomes may vary according to Latino subgroup (e.g., Lee & Ferraro, 2007), and consequently, examining relationships within particular subgroups of Latinos (e.g., Mexican-Americans, Puerto Ricans, Dominicans)

may be beneficial. In terms of CVD risk factors and outcomes in particular, the few findings that exist are equivocal, and apart from residential segregation, few other neighborhood factors have been examined in relation to CVD risk in Latinos. Additional work on the relationship between neighborhood characteristics, such as neighborhood physical and social disorder, and CVD risk in Latino subgroups, including Mexican-Americans, is needed.

Chapter 2: Current Study

The current study sought to address limitations in the extant literature by evaluating associations between perceptions of neighborhood physical and social disorder and CVD risk factors in a community sample of healthy, middle-aged Mexican American women. Specifically, the study examined markers that are theoretically purported to relate to neighborhood disorder and that are indicative of primary physiological pathways underlying CVD risk including (a) body composition [BMI], (b) insulin resistance [Homeostasis Model of Assessment - Insulin Resistance (HOMA-IR)], (c) lipids [total cholesterol to high density lipoprotein cholesterol ratio (total/HDL-c), triglycerides] and (d) blood pressure [mean arterial pressure (MAP)].

All indicators have demonstrated predictive utility in relation to CVD outcomes in previous research. BMI has been shown to be predictive of coronary heart disease risk (Owen et al., 2009; Whitlock, Lewington, & Mhurchu, 2002), as well as CVD mortality (Manson et al., 1995; Rosengren, Wedel, & Wilhelmsen, 1999), with BMIs in the overweight range associated with hazard ratios of 1.5 to 2.8 for CVD mortality as compared to normal weight BMIs (Lewis et al., 2009). HOMA-IR is a widely-used method for determining insulin resistance from basal glucose and insulin (Matthews et al., 1985; Wallace, Levy, & Matthews, 2004), with higher HOMA-IR levels indicating greater insulin resistance. Higher levels of HOMA-IR have been predictive of greater CVD incidence (including fatal and nonfatal MI and stroke, transient ischemic attack, peripheral vascular disease and revascularization procedures) in both initially healthy (Bonora et al., 2007) and diabetic (Bonora et al., 2002) samples. HOMA-IR has also been found to be a valid indicator of insulin resistance in Mexican-Americans (Haffner,

Miettinen, & Stern, 1997), and has been associated with other CVD risk factors (e.g., BMI, waist circumference, HDL-c; Andersen et al., 2006; Voruganti et al., 2008) as well as greater incident CVD (e.g., 2.5 times the odds of a CVD event in the highest compared to the lowest quintile of HOMA-IR; Hanley, Williams, Stern, & Haffner, 2002) in Mexican-Americans. Lipids, including both higher levels of HDL and lower levels of low density lipoprotein (LDL) cholesterol, have been associated with greater risk of non-fatal CVD (e.g., MI and stroke) and CVD mortality (Chirovsky, Fedirko, Cui, Sazonov, & Barter, 2009; Delahoy, Magliano, Webb, Grobler, & Liew, 2009; Mora, Rifai, Buring, & Ridker, 2008), with some estimates indicating that every 39 mg/dL decrease in LDL and every 10 mg/dL increase in HDL corresponds to a reduction of 20-30% in major CVD events (Genser & Marz, 2006; Hausenloy & Yellon, 2008). The ratio of total cholesterol to HDL cholesterol (total/HDL-c) has been found to be an efficient indicator for assessing the "two way" traffic of cholesterol and for predicting incident CVD events across ethnic populations (Hadaegh, Harati, Ghanbarian, & Azizi, 2006; Kannel, 2000; Kannel & Larson, 1993; Mediene-Benchekor, Brousseau, Richard, Benhamamouch, & Amouyel, 2001; Mora et al., 2008). Triglycerides have long been recognized as markers of CVD risk, and were highlighted recently in a scientific statement by the American Heart Association as important unique biomarkers of risk due to their pivotal role in lipid metabolism (Criqui, 1998; Gandotra & Miller, 2008; Miller et al., 2011). Triglyceride levels have been show to be predictive of stroke and MI incidence and CVD mortality in diverse populations (Arsenault et al., 2009; Carey et al., 2010; Lamarche et al., 1996; McBride, 2008; Okamura et al., 2010; Onat et al., 2006). Meta-analytic results indicate a 70% increased risk for coronary heart disease in participants with triglyceride levels in

the top versus bottom tertile (Sarwar et al., 2007). Moreover, across studies, relative risks associated with elevated triglycerides appear to be greater in women than in men (Criqui, 2007). Mean arterial pressure (MAP) is the average arterial pressure during a single cardiac cycle, and is calculated as a weighted average of systolic blood pressure (SBP) and diastolic blood pressure (DBP). Both SBP and DBP have been shown to be predictive of cardiovascular disease morbidity and mortality (Conen & Bamberg, 2008; Mancia, 2007; Wang, Staessen, Franklin, Fagard, & Gueyffier, 2005). MAP is an integrated measure of SBP and DBP, and greater MAP has been associated with greater incident CVD and CVD mortality (Gu, Dillon, Burt, & Gillum, 2010; Hadaegh, Shafiee, Hatami, & Azizi, 2011; Lawes et al., 2003; van Trijp, Grobbee, Peeters, van Der Schouw, & Bots, 2005) in initially healthy (Sesso et al., 2000) and diseased (Hadaegh, Hatami, Sheikholeslami, & Azizi, 2010) populations, with one study reporting more than double the risk of developing CVD over an 11 year period for individuals with MAP levels in the highest versus lowest quartile of the sample (Sesso et al., 2000).

The current study also explored possible indirect pathways between perceptions of disorder and CVD risk factors, including behavioral factors and psychological distress, which have received limited attention in previous research. Behavioral factors including dietary patterns, physical activity, tobacco and alcohol use, and sleep have all been linked to neighborhood disorder in previous research (as reviewed in preceding section), and have also been identified as hypothesized mediating mechanisms through which neighborhood disorder may affect CVD risks and outcomes (Chaix, Merlo, Evans, Leal, & Havard, 2009; Diez Roux, 2003, 2007). Inverse associations between consumption of dietary fiber (Bazzano, 2008; Fernandez, 2001; Pereira & Pins, 2000; Van Horn, 1997)

and CVD morbidity and mortality have been well-established, as have positive associations between dietary saturated fat intake and CVD endpoints (Hooper, 2010; Hooper et al., 2011; Hu, Manson, & Willett, 2001; Mozaffarian, Micha, & Wallace, 2010). Although a consistent link between physical activity and CVD outcomes has been demonstrated historically, more recently, the effect of sedentary behavior on CVD risk has also received increasing attention. Greater sedentary behavior, including total time spent sitting, has been associated with increased CVD mortality (Hamilton, Hamilton, & Zderic, 2007; Katzmarzyk, Church, Craig, & Bouchard, 2009; Warren et al., 2010) and has been shown in research to confer CVD risks separate from those associated with lack of exercise, including greater central adiposity, higher fasting triglycerides, and poorer values on markers of insulin resistance (Owen, Sparling, Healy, Dunstan, & Matthews, 2010). Smoking has been recognized as one of the strongest modifiable risk factors for CVD (Erhardt, 2009), and it has been estimated that the 10-year CVD mortality risk for smokers is double that of non-smokers for any given age, systolic blood pressure and cholesterol level (Conroy et al., 2003). Sleep duration has been found to have a non-linear association with CVD outcomes, with both short (i.e., $\leq$6 hours) and long (i.e., $\geq$9 hours) sleep duration being associated with greater risk for coronary heart disease and stroke morbidity and mortality than sleep not in either category (Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011; Heslop, Smith, Metcalfe, Macleod, & Hart, 2002; Kripke, Garfinkel, Wingard, Klauber, & Marler, 2002; Patel et al., 2004; Sabanayagam & Shankar, 2010). Poorer sleep quality has also been associated with increased risk of MI in healthy populations as well as recurrent CVD events in those with diagnosed CVD (Leineweber, Kecklund, Janszky, Akerstedt, & Orth-Gomer, 2003; Miller & Cappuccio,

2007; Newman et al., 2000; Schwartz et al., 1998). In terms of psychological mediators, both increased depression (Meijer et al., 2011; Van der Kooy et al., 2007; van Melle et al., 2004) and psychological stress (Dimsdale, 2008; Ramachandruni et al., 2004; Rozanski, Blumenthal, & Kaplan, 1999; Torpy, Lynm, & Glass, 2007; Vitaliano et al., 2002) have demonstrated consistent positive relationships with initial and recurrent CVD events and CVD mortality.

Specific Aims

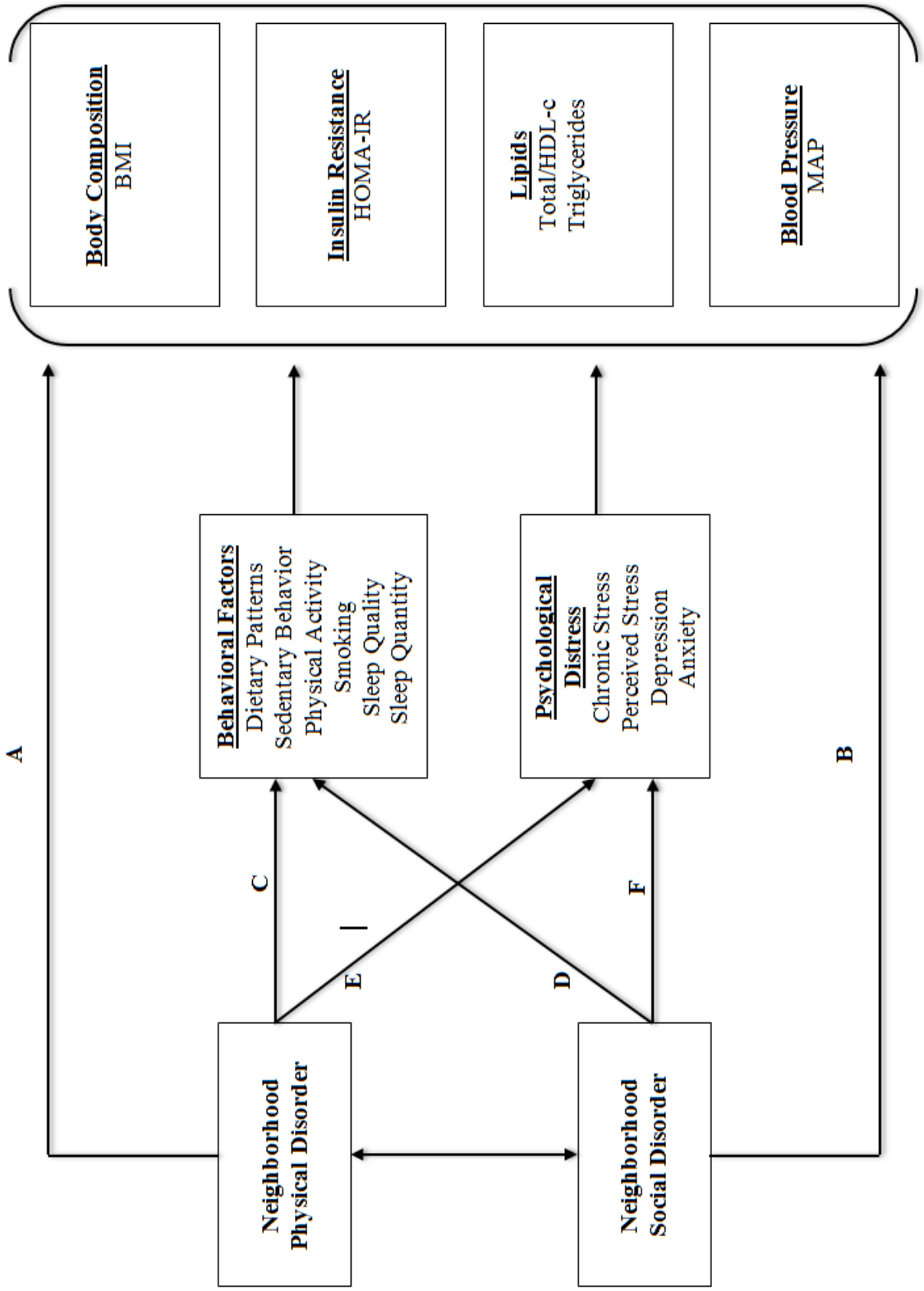
The theoretical model and hypothesized relationships associated with all specific aims are depicted in Figure 1.

Aim One: To determine the relationship between perceptions of neighborhood physical (e.g., graffiti, vandalism, noise, filth) and social (e.g., crime, drug/alcohol use, distrust/conflict with neighbors, loitering) disorder and CVD risk as assessed by indicators of body composition, insulin resistance, lipids and blood pressure (i.e., pathways A & B, Figure 1).

Aim Two: To determine whether the relationship between perceptions of neighborhood physical and social disorder and individual CVD risk factors operate indirectly through behavioral factors related to CVD risk including dietary patterns, sedentary behavior, physical activity, smoking, and sleep quality and quantity (i.e., pathways C & D, Figure 1).

Aim Three: To determine whether the relationship between perceptions of neighborhood physical and social disorder and individual CVD risk factors operate indirectly through psychological distress (i.e., chronic stress, perceived stress, depression, anxiety; pathways E & F, Figure 1).

Figure 1. Model of Associations between Neighborhood Disorder and CVD Risk Factors. This figure illustrates hypothesized relationships between neighborhood physical and social disorder, behavioral and psychological mediators and CVD risk parameters. Arrows A and B represent direct effects of physical and social disorder, respectively, on CVD risk factors. Arrows C and D show indirect effects of physical and social disorder, respectively, on CVD risk factors through behavioral factors. Arrows E and F indicate indirect effects of physical and social disorder, respectively, on CVD risk factors through psychological distress.


Body Composition
BMI
Insulin Resistance
HOMA-IR
Lipids
Total/HDL-c
Triglycerides
Blood Pressure
MAP
Behavioral Factors
Dietary Patterns
Sedentary Behavior
Physical Activity
Smoking
Sleep Quality
Sleep Quantity
Psychological Distress
Chronic Stress
Perceived Stress
Depression
Anxiety
Neighborhood Physical Disorder
Neighborhood Social Disorder
A
B
C
D
E
F

Due to the limited and inconsistent literature on the effects of neighborhood stress on CVD risk markers, explicit a priori directional hypotheses for all pathways described were not advanced. However, in general, both neighborhood physical and social disorder were expected to relate to each CVD risk factor, and effects of neighborhood disorder were expected to operate in part indirectly through behavioral factors and psychological distress. In addition to primary aims, since prior research suggests that there may be a complex interplay between perceptions of neighborhood disorder and objective neighborhood SES on health outcomes (Diez Roux, 2001; Ross & Mirowsky, 2001), secondary sensitivity analyses also examined the stability of the perceived neighborhood disorder-CVD risk factor associations across levels of neighborhood SES.

Chapter 3: 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 through a commercial database that used varied records (i.e., credit cards, real estate, phone number listings) to gather information such as names, addresses, and general demographics for a particular region. Potential participants were randomly selected from a list of households with Latino surnames that had a female occupant in the targeted age range. They were subsequently contacted via phone or mail, and, once reached, screened for eligibility and invited for participation. Sample SES (as indicated by household income) was monitored throughout the enrollment period to ensure that the SES distribution was similar to that of the sampling frame. Low SES households were oversampled in the last year of enrollment to ensure adequate representation.

Eligible participants were female, between the ages of 40-65, Mexican-American (self-identified), residing in selected recruitment areas, and free of cardiovascular diseases, diabetes, kidney disease, chronic inflammatory conditions, or other major illnesses, pregnancy, or use of medications with sympathetic 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 all portions of the physical exam or blood draw, resulting in a final sample of N=304 for the current study. Additional exclusions were made on an individual analysis basis for specific CVD risk factors, including $N=1$ in BMI analyses due to no information

on height, *N=13* in HOMA-IR analyses for undiagnosed diabetes indicated by hemoglobin A1c levels greater than or equal to 6.5, *N*=1 in triglyceride analyses due to the use of fibrates, and *N*=3 in MAP analyses due the use of antihypertensives.

Community Characteristics. Communities with high densities of Mexican American residents and a relatively wide range of SES in San Diego County were targeted for recruitment. These included the South Bay communities of Chula Vista, Bonita, and National City. Study participants residing in these areas were represented by 68 census tracts as defined by the Year 2000 Census. According to summary data from the American Community Survey for the years 2005-2009 (the period within which data was collected for the larger study), the average percent of individuals whose income was below the poverty level across census tracts was 14.7%, with a range of 0.8% to 42.1%. Average percent of individuals with a high school education or less was 54%, with a range of 20.9% to 80.5%. The mean number of individuals within a census tract was 4.70, however, a considerable number of census tracts (22 out of 68) had 2 or fewer individuals.

Procedures

After enrollment, participants were scheduled for two consecutive weekday home visits by study staff. Participants were rescheduled if they reported any symptoms of acute illness. During the first visit, a bilingual research assistant obtained written informed consent and administered a battery of measures (in Spanish or English based on participant preference) assessing sociodemographic characteristics, behavioral risk factors, health history, and psychosocial variables. Participants were also given instructions for a 12-hour, overnight fast. During the second visit, a licensed

phlebotomist obtained a fasting blood draw, and a bilingual research assistant performed physical measurements (BP, height, weight). Participants were instructed 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. Protocols were standardized, all research staff were bilingual and bicultural and thoroughly trained in study procedures, and regular quality control checks were conducted. The San Diego State University and University of California, San Diego institutional review boards approved all study procedures.

Measures

Cardiovascular Disease Risk Factors

Body composition. Height was measured to the nearest centimeter, using a Seca Portable Stadiometer, placed on a flat surface. Weight was measured to the nearest tenth of a kilogram using a Seca Medical 882 Digital Scale, while participants were barefoot and in light clothing. BMI was calculated using the standard algorithm [i.e., weight (kilograms)/height (meters)^2].

Insulin resistance. Serum glucose was measured by Hexokinase/G6PDH and the reduction of NADP+ NADPH+H+ was followed kinetically at 340nm in an AU5400 analyzer. The method has within and between assay coefficients of variation (CVs) of <3%. Serum insulin was determined by a solid-phase, enzyme-labeled chemiluminescent immunometric assay (intra and inter assay CVs <3% - 6%). HOMA-IR was calculated using the formula [glucose (mg/dl) * insulin (μU/ml)]/405.

Lipids. Serum total and HDL cholesterol and triglycerides were measured by modified enzymatic methods (intra and inter assay CVs <3%) on an AU5400 random

access analyzer, following standards set by the Lipid Standardization Program of the Centers for Disease Control and Prevention (Warnick, 2000). Serum total cholesterol was divided by HDL-c to calculate the total/HDL-c ratio.

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). MAP was calculated using the equation [(2*DBP + SBP)/3]. Blood specimens used to derive lipids, glucose, and insulin were processed by Quest Diagnostics, West Hills, CA laboratories, which adheres to standards set forth by the College of American Pathologists (Neeley, 1972; Warnick, 2000).

Neighborhood Disorder

Individuals' experience with daily stressors related to their neighborhood environment was measured using the 13-item Perceived Neighborhood Disorder Scale (Ross & Mirowsky, 1999), which was comprised of two subscales: neighborhood physical and social disorder. Neighborhood physical disorder assessed for the degree to which physical conditions such as graffiti, noise, vandalism, abandoned buildings, filth, and lack of upkeep are present in one's neighborhood (e.g., "Vandalism is common in my neighborhood"). Neighborhood social disorder assessed for the degree to which social conditions such as loitering, crime, drug and alcohol use, conflicts with neighbors, and safety issues are present (e.g., "I am always having problems with my neighbors"). Participants responded on a scale from 0 "never" to 3 "very often". The scale was originally developed based on theoretical work by Skogan (1990) on visible cues of neighborhood disorder and decay and effects on neighborhood residents (Ross &

Mirowsky, 1999). Items were derived based on previous measures of neighborhood civilities, problems, and disorder. Scale items were refined through structural equation modeling techniques, and the final scale was validated and found to relate in expected ways with sociodemographic neighborhood characteristics (e.g., neighborhood income and education negatively correlated with disorder). The overall scale has been shown to be reliable (α= .91; Ross & Mirowsky, 1999) and has demonstrated relationships with self-rated health (Ross & Mirowsky, 2001), and psychological, behavioral and biological CVD risk factors (Burdette & Hill, 2008; Franzini, Caughy, Spears, & Esquer, 2005; Hill & Angel, 2005). Both the physical (α= .71) and social (α= .71) disorder subscales demonstrated adequate reliability in the current sample. Items were translated by bilingual/bicultural staff members for the current study, via a multistep process that included forward and back translation procedures and a review by committee to ensure semantic equivalence.

Behavioral Factors Related to CVD Risk

Dietary patterns were assessed using two brief screening instruments that have been shown to be highly correlated with the full-length, Block-Food Frequency Questionnaire in a validation study with a large, multi-ethnic sample (Block, Gillespie, Rosenbaum, & Jenson, 2000). Total daily consumption of fiber (grams) and saturated fat (grams) were utilized for the current study. Spanish translated versions of these scales were developed for a previous study of Mexican-Americans with minor modifications to include examples of ethnically-appropriate foods (e.g., tortillas, lard; Elder et al., 2005). Sedentary behavior was measured using a subscale from the International Physical Activity Questionnaire (IPAQ; Craig et al., 2003). The IPAQ has demonstrated adequate

reliability and validity cross-culturally, and Spanish versions were translated via forward and back translation procedures (Craig et al., 2003). Participants were asked to specify total time in minutes spent sitting during a typical week (with higher scores reflecting more sedentary lifestyles). Leisure time physical activity was assessed via the Leisure Time Exercise Questionnaire (Godin, Jobin, & Bouillon, 1986). The scale is a simple measure of the frequency and intensity of leisure time physical activity and has demonstrated adequate reliability and concurrent validity (Godin et al., 1986). Respondents were instructed to report how many times per week they engaged in strenuous, moderate, and mild exercise for more than 15 minutes during their free time. A total score was calculated by multiplying the frequency of weekly exercise by its metabolic equivalent based on intensity (i.e., 9, strenuous, 5, moderate, and 3, mild). Items were translated by bilingual/bicultural staff members as previously described (i.e., forward and back translation with reconciliation by committee). Smoking was assessed by an item asking participants if they currently smoke (responses coded yes/no). Sleep quality and quantity were assessed using items from the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Overall self-rated sleep quality was determined using the composite score from the PSQI. Sleep quantity was assessed based on an item on the PSQI asking respondents to indicate how many hours of actual sleep per night they have obtained in the past month. Based on self-reported sleep duration, participants were categorized as "short" (i.e., $\leq$6 hours), "long" sleepers ($\geq$9 hours), or "normal" sleepers (7-8 hours).

Psychological Distress

Chronic stress was measured using a 9-item scale developed for use in the Study of Women's Health Across the Nation, and designed to tap into life problems in diverse domains widely applicable to women of varying socioeconomic and ethnic backgrounds (Bromberger & Matthews, 1996). The scale asked participants to indicate if they experienced ongoing stress for at least 12 months duration in eight major life domains (e.g., personal health stress, drug or alcohol problems in a family member, financial strain, work stress, relationship stress) or any "other" domain. The response format was on a four-point scale with 0 indicating that the stressor did not occur, 1 indicating occurrence of the stressor but with little or no subjective stress ("not upsetting"), and 2 and 3 indicating occurrence of the stressor with moderate or severe subjective stress ("somewhat upsetting" and "very upsetting"), respectively. Responses on each item were summed to tabulate total "chronic stress" burden. Items were translated into Spanish by bilingual/bicultural staff members as described previously (i.e., forward and back translation with reconciliation by committee). Reliability for this measure in the current sample was adequate (α= .68). The 4-item Perceived Stress Scale (PSS; Cohen & Williamson, 1988) was used to examine generalized stress appraisals in the past month, including the degree to which individuals felt life situations were unpredictable, uncontrollable, and overloading (e.g., "how often have you felt difficulties were piling up so high that you could not overcome them?"). This scale has demonstrated a high level of internal consistency (α=.72) in prior research (Cohen, Kamarck, & Mermelstein, 1983), similar to the original 10-item measure (α=.76), and has also been found to relate similarly as the 10-item measure to sociodemographic characteristics, other stress

measures (e.g., measures of life event, work stress), self-rated health, healthcare utilization, and health behaviors (Cohen & Williamson, 1988). Responses were on a three point scale, with 1 indicating "never", 2 "sometimes", and 3 "often". Items were summed to obtain a total perceived stress score. Translation of the original 10-item Perceived Stress Scale (from which the 4-item measure was drawn) into Spanish for a Mexican population was conducted by the Mapi Research Institute using a multi-step (forward and back-translation) approach to ensure linguistic and conceptual validity (Acquadro, Conway, Giroudet, & Mear, 2006; Acquadro, Jambon, Ellis, & Marquis, 1996). Reliability for the 4-item version of the PSS in the current sample was relatively low (α= .61). However, internal consistency is necessarily truncated when few items comprise a scale (Henson, 2001), and even low alpha values do not seriously attenuate regression coefficients (Schmitt, 1996). Depression was measured with the widely-used Center for Epidemiological Studies Depression Scale - Revised (CESD-R; Eaton, Muntaner, Smith, Tien, & Ybarra, 1999). The scale has strong psychometric properties and has been translated into Spanish, with preliminary validation work performed in a Mexican sample (Reyes-Ortega et al., 2003). The CESD-R demonstrated good reliability in the current sample (α= .91). Anxiety was assessed using the 10-item Spielberger Trait Anxiety Inventory, Short Form (Spielberger, Sydeman, Owen, & Marsh, 1999). The short form version correlates highly with the full version and has demonstrated good psychometric properties with a reported test-retest reliability of α=.73 (Bromberger & Matthews, 1996). Internal consistency for the scale in the current sample was good (α= .79).

Covariates

Demographic and cultural factors associated with neighborhood disorder in prior research that could represent potential confounding influences in the relationship between neighborhood disorder and CVD risk factors were selected as covariates. Self-reported date of birth was utilized to calculate age at assessment. A dichotomous variable (coded 0, 1) was created to indicate menopausal status (participants who reported no menstruation for the past 12 months were considered post-menopausal). Use of antidepressants, statins, and thyroid medications, which have all been associated with one or more of the markers of CVD risk assessed in the present study in previous work (Biasucci, Biasillo, & Stefanelli, 2010; Danzi & Klein, 2003; Maron, Fazio, & Linton, 2000; Warrington, Padgham, & Lader, 1989), were also represented dichotomously. Educational attainment was categorized as follows: (1) less than 9^{th} grade; (2) 9^{th} -11^{th} grade; (3) high school diploma or equivalent; (4) some college; (5) bachelors degree; and 6) graduate or professional degree. Monthly gross household income was assessed on an ordinal scale in $500 increments, ranging from less than $500/month to more than $8,000 per month. An education/income composite was created by standardizing and summing the variables. For those participants with missing income data, education only was used to represent SES. Acculturation status was measured using the Adult English Proficiency, Adult Pattern of English versus Spanish Language Usage, and Child Language Experiences subscales from the Hazuda Acculturation and Assimilation Scales (Hazuda, Stern, & Haffner, 1988). The scales were developed and validated in a population-based sample of Mexican-American adults aged 25-64 years (Hazuda et al., 1988). These scales are available in English and Spanish and have been shown to be

reliable with alpha coefficients ranging from 0.85-0.95 (Haffner et al., 1994). In previous analyses, results from a principal components analysis (PCA) with varimax rotation showed that these scales represented a single latent construct with high factor loadings for each indicator (.75 - .93). Thus, regression-based factor scores were used to represent acculturation. Acculturation was included as a covariate as previous research has shown that perceptions of disorder are influenced by many factors including sociodemographic characteristics (Sampson & Raudenbush, 2005), and previous work with the current sample has demonstrated associations between acculturation and CVD risk factors (Espinosa de los Monteros, Gallo, Elder, & Talavera, 2008). Additionally, composites for both acculturation and SES were utilized because of the high degree of correlation between individual indicators, and in order to increase model parsimony.

Neighborhood SES

In order to obtain neighborhood SES for secondary analyses, census-tract level data for educational attainment and household income were extracted from the American Community Survey for the years 2005-2009 (U.S. Census Bureau, n.d.). Median household income for each census tract was used to represent income. A single score for census tract education was calculated based on a previously defined algorithm (Gump, Matthews, & Raikkonen, 1999), wherein the seven categories of education (ranging from less than 9^{th} grade to graduate or professional degree) were assigned a numerical value from 1 to 7 (with "1" representing the lowest level of education and "7" the highest). Each numerical value was multiplied by the number of people in a census tract reporting the corresponding level of education, and these values were summed and divided by the total number of individuals in that census tract, resulting in a single education score.

Education scores and median household income for each census tract were then standardized and summed to create a census tract/neighborhood-level SES composite. Interaction terms were created by multiplying this composite by neighborhood physical and social disorder, respectively.

Statistical Analyses

In order to determine whether associations between neighborhood disorder variables, behavioral and psychological variables, and CVD risk factors (i.e., Aims 1-3) should be examined in multi-level analyses to account for clustering within neighborhoods (i.e., census tracts), preliminary analyses were conducted in Hierarchical Linear Modeling (HLM) Version 6.08 for Windows (Scientific Software International, Inc., Lincolnwood, IL). However, all intra-class correlation coefficients (ICCs) for unconditional models for each outcome specified were nonsignificant, indicating very little interdependence (i.e., ≤2% of variance accounted for) at the neighborhood level. Consequently, single-level analyses (i.e., ordinary least squares regression) were pursued using Mplus, Version 6.11 (Muthén & Muthén, Los Angeles, CA) and SPSS, Version 18.0 for Macintosh (SPSS, Chicago, Illinois, USA).

To address Aim 1, which involved examining the direct association between perceptions of neighborhood physical and social disorder and the five CVD risk factors, distributions of the CVD risk factors were first evaluated. With the exception of MAP, distributions for all CVD risk factors were significantly skewed, and were therefore natural log- transformed. Transformed distributions approached normality. Distributions for neighborhood social and physical disorder, and physical activity were also highly

skewed; however, transformation did not achieve distributions approaching normality, and therefore, these variables were categorized into quintiles.

Path analytic models were tested to explore the relationships between perceptions of neighborhood physical and social disorder and CVD risk factors. The maximum likelihood robust (MLR) estimation procedure employed by MPlus (Muthén & Muthén, 2006) was used to estimate model parameters. This procedure provides a chi-square test statistic (Yuan-Bentler T_2 [Y-Bχ^2]; Yuan & Bentler, 2000) and standard errors that are adjusted for multivariate non-normality and missing data. For each CVD risk factor, a model specifying direct paths from both neighborhood physical and social disorder to the CVD risk factor was examined, first adjusting for age only, and then fully adjusting for all covariates.

To address Aim 2, which involved exploring whether relationships between perceptions of neighborhood physical and social disorder and individual CVD risk factors operated indirectly through behavioral factors related to CVD risk including dietary patterns, sedentary behavior, physical activity, smoking, and sleep quality and quantity, a path analytic model including direct paths from neighborhood predictors to the CVD risk factor *and* indirect paths through behavioral factors was specified for each CVD risk factor, adjusting for all covariates. However, prior to including behavioral factors in path analytic models, in order to reduce the number of analyses and minimize Type I error risk, a measurement model for a latent "behavioral factors" construct was first examined. Three descriptive fit indexes were used to assess model fit: the comparative fit index (CFI; Bentler, 1990), the root mean square error of approximation (RMSEA; Steiger, 1990), and the standardized root mean square residual (SRMR; Hu & Bentler, 1999). All

three parameters represent descriptive fit indices of overall model fit, with CFI values of 0.90 or more and RMSEA and SRMR values of 0.05 or less indicating good fit. RMSEA and SRMR values between 0.05 and 0.08 indicate acceptable fit (Chen, 2007).

If fit indices indicated good or adequate model fit, the latent behavioral factors construct was retained; if poor model fit was indicated, individual components were examined in simple mediation models. These mediation models tested if any of the individual behavioral factors partially accounted for the relationship between neighborhood physical or social disorder and a given CVD risk factor. Bootstrap estimates based upon 5,000 resamples were obtained for each indirect effect using the SPSS Macro for Simple Mediation (Preacher & Hayes, 2004). The use of bootstrapping is recommended over the Sobel test (Sobel, 1982, 1986) and the casual steps approach (Baron & Kenny, 1986; Kenny, Kashy, & Bolger, 1998) because it does not impose the assumption of normality of the sampling distribution, and has greater power while maintaining reasonable control of Type I error (MacKinnon, Lockwood, & Williams, 2004; Preacher & Hayes, 2008). Behavioral factors that accounted for significant indirect effects in simple mediation analyses were then included in the indirect effects path analytic model described above, in order to examine all indirect effects for both physical and social disorder simultaneously.

In the indirect effects path analytic model, in order to test the significance of specific estimates of behavioral indirect effects, methods outlined by MacKinnon, Fritz, Williams, and Lockwood (2007) were utilized. Using this method, the significance of individual indirect effects can be ascertained in models where multiple indirect effects are being estimated simultaneously. Values for the two paths involved in the indirect effects

and their standard errors are entered in the PRODCLIN (distribution of the PRODuct Confidence Limits for INdirect effects) program, and the distribution of the product confidence limits are computed. This method tests the significance of and confidence limits for indirect effects based on the distribution of the product, which results in more accurate Type I error rates and greater power than more commonly used tests (MacKinnon et al., 2007).

To address Aim 3, which involved exploring whether relationships between perceptions of neighborhood physical and social disorder and individual CVD risk factors operated indirectly through psychological factors including chronic stress, perceived stress, depression, and anxiety, similar steps were followed as for Aim 2. First, a measurement model of a latent "psychological distress" construct was examined. If model indices indicated good or adequate fit, this latent construct was retained, if not, individual components were tested in simple mediation models. Psychological factors that represented significant indirect effects in simple mediation analyses (or the latent psychological distress construct) were then included in path analytic models for each CVD risk factor, specifying direct paths from neighborhood disorder predictors to the CVD risk factor *and* indirect paths through psychological factors, adjusting for all covariates. The significance of specific estimates of psychological indirect effects in path analytic models were subsequently evaluated as defined above (MacKinnon et al., 2007).

Secondary analyses examined whether the direct associations between perceptions of neighborhood physical and social disorder and CVD risk factors differed by levels of objective neighborhood SES. Interaction terms were created by multiplying

neighborhood physical and social disorder, respectively, by the neighborhood SES composite. Both interaction terms, along with the neighborhood SES composite, were added to path analytic models examining the direct association of neighborhood physical and social disorder with each CVD risk factor, adjusting for all covariates. Significant interaction effects were viewed as evidence for effect modification by neighborhood SES.

Power Analysis

Guidelines by Fritz and MacKinnon (2007) for sample sizes necessary to detect mediation effects when relationships between predictor-mediator and mediator-outcome are expected to be of small-to-medium size indicate that a sample size of 161 is necessary for detecting effects with 80% power using the method outlined by MacKinnon et al. (2007). Consequently, the current study was adequately powered to detect the hypothesized effects.

Chapter 4: Results

Descriptive Statistics and Bivariate Associations

Table 1 displays descriptive statistics for all covariates, neighborhood physical and social disorder, and CVD risk factors. On average, participants were 49.76 years old (SD=6.58). The majority had a monthly income of $3000 or greater and had completed high school or more education. Approximately half of participants were post-menopausal and a small percentage were using antidepressants (3%), statins (2%), or thyroid-regulating medications (6%). Average levels of neighborhood physical and social disorder were on the low end of the scale, indicating that participants perceived little disorder overall. BMI was on average in the overweight range, and average levels of other CVD risk factors were within normal limits.

Table 2 displays descriptive statistics for behavioral and psychological factors, as well as correlations between these variables and CVD risk factors. Greater fat intake (r= 0.16, p< .01) and poorer sleep quality (r= 0.13, p< .05) were associated with greater BMI. Poorer sleep quality was also related to greater HOMA-IR levels (r= 0.13, p< .05). Women who reported increased physical activity demonstrated lower HOMA-IR (r= - 0.25), total/HDL-c ratio (r= - 0.24), and triglycerides (r= -0.19; all ps< .01). No other behavioral or psychological mediators were associated with any CVD risk factors.

Direct Effects of Neighborhood Physical and Social Disorder on CVD Risk Factors

Results of path analytic models examining direct effects of neighborhood physical and social disorder in relation to the five CVD risk factors (i.e., Aim 1) are presented in Figure 2. In models adjusted for age-only, greater perceptions of social disorder were associated with a higher total/HDL-c ratio, and higher levels of triglycerides and MAP

Table 1. Descriptive Statistics for Covariates, Neighborhood Physical and Social Disorder, and CVD Risk Factors

Variable	
Covariates	
Age (years) [*M* (SD)]	49.76 (6.58)
Household Monthly Income [*N* (%)]	
<$3000	124 (41.47)
≥$3000	175 (58.53)
Educational Attainment [*N* (%)]	
< High school/GED	105 (34.54)
≥ High school diploma/GED	199 (65.46)
Acculturation [*M* (SD)]	
Hazuda Adult English Proficiency	3.02 (0.89)
Hazuda Adult Language Pattern	2.26 (0.99)
Hazuda Child Language Experiences	1.37 (0.87)
Menopausal Status [*N* (%)]	145 (47.70)
Medication Use [*N* (%)]	
Antidepressants	10 (3.29)
Statins	5 (1.64)
Thyroid-regulating	19 (6.25)

(table continues)

Table 1. Descriptive Statistics for Covariates, Neighborhood Physical and Social Disorder, and CVD Risk Factors, Continued

	N	*Min*	*Max*	*M*	*SD*
Neighborhood Disorder					
Physical Disorder	304	0	13.50	2.72	2.76
Social Disorder	304	0	16.00	3.13	3.00
CVD Risk Factors					
BMI (kg/m^2)	303	19.15	48.18	28.61	5.40
HOMA-IR	291	0.19	9.04	1.73	1.43
Total/HDL-c	304	1.60	8.10	3.80	1.09
Triglycerides (mg/dL)	304	38.00	436.00	133.17	67.69
MAP (mm Hg)	301	66.83	112.67	87.91	9.84

Note. CVD=Cardiovascular Disease. BMI=Body Mass Index. HOMA-IR=Homeostasis Model of Assessment – Insulin Resistance. Total/HDL-c=Total Cholesterol to High Density Lipoprotein Cholesterol Ratio. MAP=Mean Arterial Pressure. Range for Hazuda scales is 1-4. Menopausal status refers to individuals who are post-menopausal. Distributions of income and education and Hazuda scales presented for descriptive purposes; composites of income and education (i.e., socioeconomic status), and Hazuda scales (i.e., acculturation) used in analyses.

Table 2. Descriptive Statistics for Proposed Behavioral and Psychological Mediators, and Correlations of Mediators with CVD Risk Factors

Variable	*M* (SD)	BMI *r*	HOMA-IR *r*	Total/HDL-c *r*	Triglycerides *r*	MAP *r*
Behavioral Factors						
Dietary Patterns						
Daily intake of fat (gms)	89.21 (21.94)	0.16**	0.07	0.05	0.03	0.06
Daily intake of fiber (gms)	18.00 (5.46)	-0.01	-0.01	-0.06	0.02	0.04
Sedentary Behavior	28.15 (16.24)	0.09	0.07	-0.01	-0.03	0.03
(avg hours sitting/week)						
Physical Activity	19.00 (19.11)	-0.11	-0.25**	-0.24**	-0.19**	-0.09
(avg MET-mins/week)						
Current Smoker	27 (8.88)	-0.03	-0.00	0.00	0.00	-0.02
PSQI Sleep Quality	4.81 (3.12)	0.13*	0.13*	-0.00	0.01	-0.02
PSQI Sleep Quantity (hrs/night)	6.91 (1.22)	-0.11	-0.02	0.06	0.06	0.03
Psychological Distress						
Chronic Stress Scale	4.86 (4.51)	0.08	0.06	-0.02	0.02	0.01
Perceived Stress Scale	5.54 (1.52)	0.04	0.03	0.03	-0.01	-0.04
CESD-R Depression Scale	8.10 (10.65)	0.05	0.10	0.00	0.01	-0.03

(table continues)

Table 2. Descriptive Statistics for Proposed Behavioral and Psychological Mediators, and Correlations of Mediators with CVD Risk Factors, Continued

Variable	*M* (SD)	BMI *r*	HOMA-IR *r*	Total/HDL-c *r*	Triglycerides *r*	MAP *r*
Spielberger Trait Anxiety Inventory	16.33 (4.76)	0.06	0.04	0.05	-0.00	-0.04

Note. CVD=Cardiovascular Disease. BMI=Body Mass Index. HOMA-IR=Homeostasis Model of Assessment – Insulin Resistance. Total/HDL-c=Total Cholesterol to High Density Lipoprotein Cholesterol Ratio. MAP=Mean Arterial Pressure. PSQI=Pittsburgh Sleep Quality Inventory. CESD-R=Center for Epidemiological Studies Depression Scale-Revised. Higher values equate to poorer sleep quality. *p< .05 **p< .01.

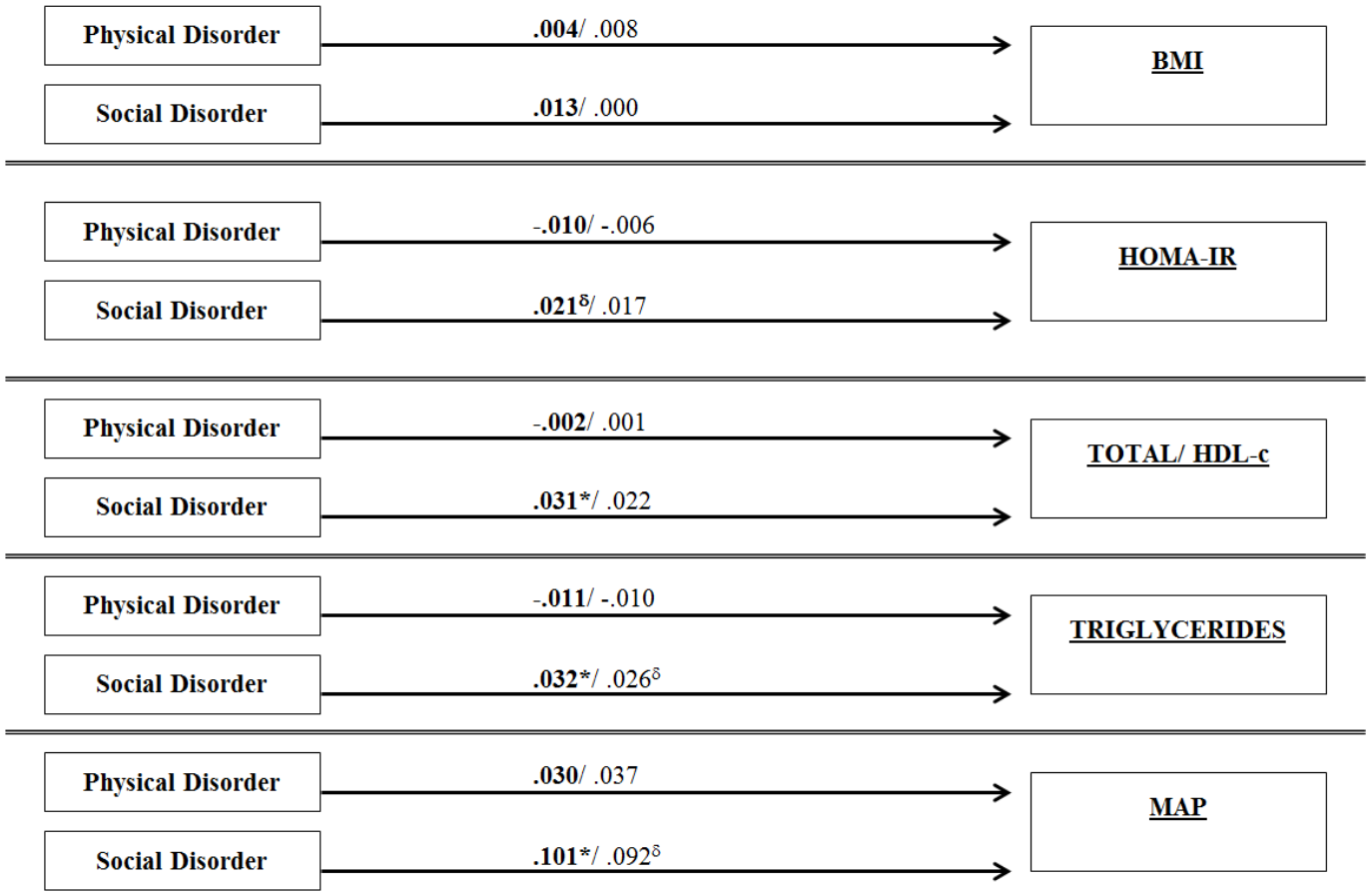


Figure 2. Results of analyses examining direct pathways from neighborhood physical and social disorder to CVD risk factors. Standardized structural/path coefficients represent age- adjusted analyses (in bold), and fully adjusted analyses (adjusted for age, socioeconomic status, acculturation, menopausal status, and medications relevant to CVD risk factors). *$p < .05$. $^{\delta}p < .10$.

(*p*s< .05). The association between social disorder and HOMA-IR also approached significance (*p*= .058). Physical disorder was not significantly associated with any CVD risk factor. After adjustment for additional covariates including SES, acculturation, menopausal status, and medications, relationships of social disorder with triglycerides and MAP were attenuated (*p*s< .10), and all other relationships were not statistically significant. Likewise, physical disorder was unassociated with CVD risk factors in fully-adjusted models.

Indirect Effects of Neighborhood Physical and Social Disorder on CVD Risk Factors through Behavioral Pathways

Fit for the latent one-factor behavioral measurement model (comprised of dietary patterns, sedentary behavior, smoking, and sleep quality and quantity) was poor according to descriptive fit indices (Y-Bχ^2(df=9) = 30.68, *p* = .00, CFI = .61, RMSEA = .09, SRMR = .05), indicating that the behavioral variables did not load onto a single, coherent latent construct. Consequently, this latent variable was not retained in further analyses.

Indirect effects for individual behavioral factors (i.e., Aim 2) were thus examined in simple mediation models. Bootstrap estimates in simple mediation models revealed no significant indirect effects for relationships between either physical or social disorder and any CVD risk factor via behavioral pathways.

Indirect Effects of Neighborhood Physical and Social Disorder on CVD Risk Factors through Psychological Pathways

Fit for the latent one-factor "psychological distress" measurement model was good (Y-Bχ^2(df=2) = 3.55, *p* = .17, CFI = .99, RMSEA = .05, SRMR = .02; see Figure 3),

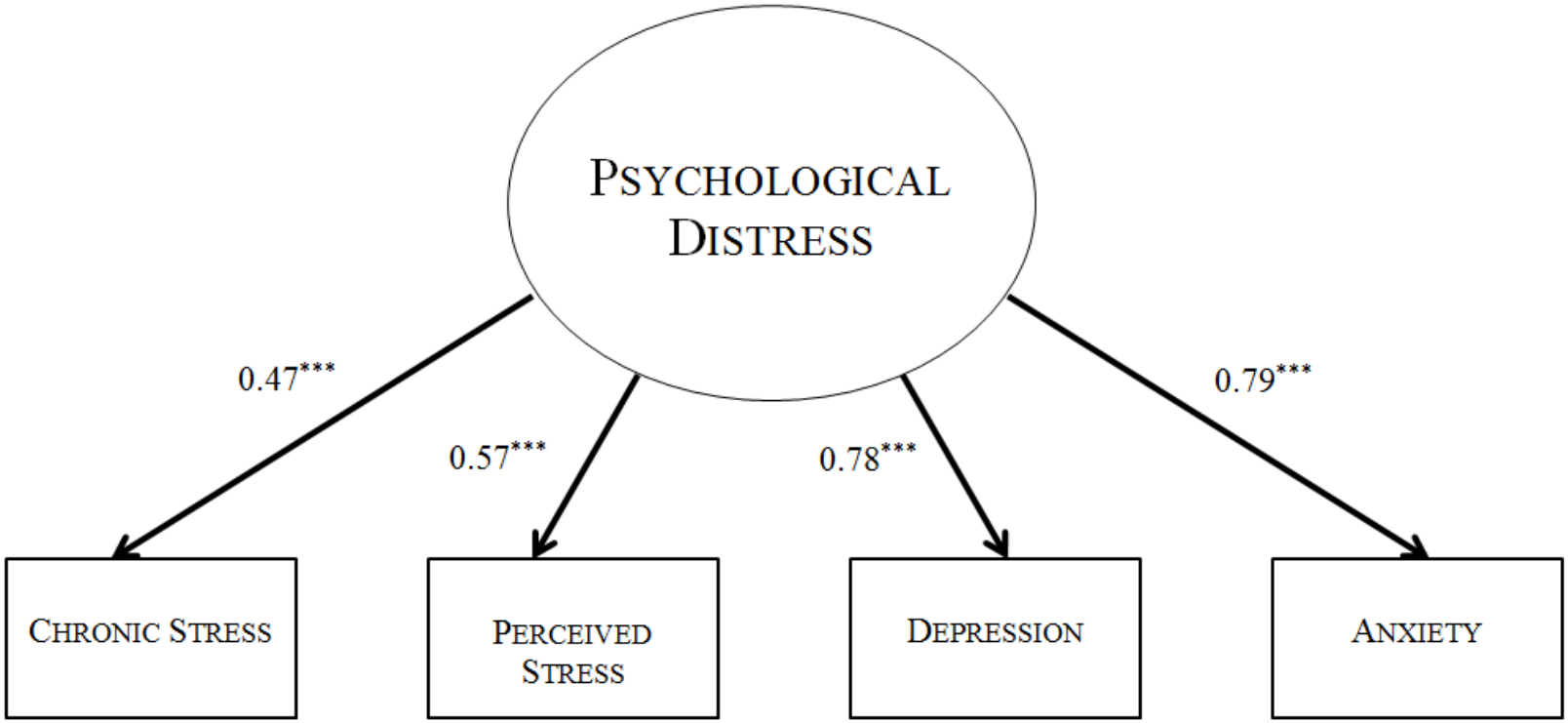


Figure 3. Results of confirmatory factor analysis investigating a one-factor model of psychological distress. Factor loadings are standardized. *** p< .001.

indicating that as predicted, psychological variables of interest loaded onto a single latent construct. Thus, this latent "psychological distress" variable was retained in path analyses.

Tests in path analysis of the model of indirect effects of neighborhood physical and social disorder through the psychological stress latent factor (i.e., Aim 3), adjusting for covariates, indicated poor model fit (Y-Bχ^2(df=33) = 132.18, p = .00, CFI = .77, RMSEA = .10, SRMR = .05). In addition, standardized path coefficients of indirect effects via the psychological distress factor for both neighborhood physical and social disorder were nonsignificant for all CVD risk factors.

Neighborhood Disorder-CVD Risk Factor Associations by Levels of Neighborhood SES

Secondary analyses tested whether the associations between perceptions of neighborhood physical and social disorder and CVD risk factors differed by levels of objective neighborhood SES. The neighborhood social disorder by neighborhood SES interaction effect on MAP approached significance (β= .10, p= .053). To examine this interaction pattern further, simple slopes analyses were conducted examining the association between social disorder and MAP at the mean of neighborhood SES, and at one standard deviation below and above the mean (Preacher, Curran, & Bauer, 2006). Neighborhood social disorder was unrelated to MAP at low neighborhood SES (i.e., one standard deviation below the mean; β= -.10, p= .88). However, greater neighborhood social disorder was marginally significantly associated with greater MAP at average levels of neighborhood SES (β= .85, p= .07), and significantly associated with greater

MAP at high neighborhood SES (i.e., 1 standard deviation above the mean; β= 1.79, p= .01).

Chapter 5: Discussion

The current study examined the relationship between perceptions of neighborhood physical and social disorder and CVD risk in relatively healthy, middle-aged Mexican-American women from a border community. The study sought to address limitations in the extant literature by utilizing integrated measures of neighborhood disorder that tap into diverse aspects of social (e.g., crime, poor social relations, substance use) and physical (structural and environmental deterioration, ambient noise, lack of cleanliness, destruction of property) disorder. The study also examined multiple indicators of CVD risk that (with the exception of BMI) have seldom been evaluated in relation to neighborhood disorder, including indicators of obesity, insulin resistance, dyslipidemia and blood pressure. Moreover, psychological and behavioral factors that could partially account for the relationship between neighborhood disorder and CVD risk factors were explored. Finally, the current study evaluated the neighborhood disorder-CVD risk factor association in a sample of middle-aged Latino women, a large and rapidly growing U.S. ethnic minority group that has been understudied in previous work in the area.

The primary aim of the study was to determine the nature of the associations between perceptions of neighborhood physical and social disorder with CVD risk factors. No significant associations were found between perceptions of physical disorder and any of the CVD risk factors in either age-adjusted or fully-adjusted models. Perceptions of social disorder were significantly associated with lipids (i.e., total/HDL-c ratio, triglycerides) and blood pressure (i.e., MAP) in age-adjusted models. However, after adjustment for relevant demographic, cultural, and biological covariates, all relationships were attenuated to non-significance.

Few previous studies have assessed the association between neighborhood physical disorder with CVD risk factors, and the available evidence is mixed. A prior study of 1032 urban residents reported that residents' ratings of physical disorder including presence of graffiti, litter, broken glass and neighborhood upkeep were unrelated to odds of obesity (Boehmer et al., 2007). In contrast, another study found that physical disorder (as measured by crime, noise, and filth) related to greater obesity risk in a sample of 1338 Texas residents (Burdette & Hill, 2008). Moreover, greater perceptions of physical disorder (as measured by the state of sidewalks, playgrounds and parks, trash and litter in the neighborhood, adequate shopping, heaving traffic/speeding, excessive noise, and violence) related to higher levels of fibrinogen and increases in IL-6 over 4 years in the MESA cohort (Nazmi et al., 2010). Notably, in both of the latter studies, while characteristics of physical disorder (as defined in the current study) were predominant in the measure utilized, characteristics of social disorder (i.e., crime, violence) were also present. Consequently, it is possible that the significant relationships found could have been due to the influence of social disorder. Indeed, a study comparing the strength of the associations of physical versus social disorder with self-reported health, physical functioning, and the absence of chronic conditions, found that social disorder had a significantly greater negative relationship with health than did physical disorder (Ross & Mirowsky, 2001).

Several factors may help explain the relatively weaker influence of physical disorder in the current and prior studies. It may be that perceptions of physical disorder, although presumably based on "objective" neighborhood characteristics, do not accurately capture the true health-affecting physical conditions of a neighborhood. There

are several studies that have found associations between objectively-assessed physical neighborhood characteristics and CVD outcomes, with greater objectively-assessed physical disorder being associated with poorer outcomes (Augustin, Glass, James, & Schwartz, 2008; Ellaway, Macintyre, & Bonnefoy, 2005; Glass, Rasmussen, & Schwartz, 2006; Sundquist et al., 2006). However, evidence suggests that individuals' perceptions of disorder vary, even in neighborhoods with comparable levels of objectively-defined disorder (Keita et al., 2011). In fact, one study that utilized data from two large population-based cohort studies of urban-dwelling older adults reported that the intra-neighborhood correlation coefficient (which represents the degree of inter-resident agreement in a neighborhood) for neighborhood physical disorder (as represented by characteristics including trash and litter, vandalism, loud noises, poor sidewalks/curbs, low lighting, unsafe traffic and walking areas, and presence of strangers) was .11 for the cohort in which neighborhood was defined as census block groups, and .33 for the cohort in which neighborhood was defined by the City Department of Planning (which uses community definitions of existing neighborhoods; Cagney et al., 2009). Consequently, agreement between residents on perceptions of physical disorder in any given neighborhood was low. Although variability in perceptions of physical disorder for residents of a given neighborhood may be indicative of differing "micro-environment" experiences of individuals within that neighborhood, it could also simply reflect differences in awareness of the physical characteristics of neighborhoods. In the latter case reality may be more impactful than perception, in that aspects of physically-disordered neighborhoods (e.g., noise, lack of cleanliness, dilapidation) could exert negative health effects on residents regardless of whether or not residents perceive these

disordered aspects to be present. For example, in the study by Boehmer et al. (2007) residents' perceptions of neighborhood physical disorder were unrelated to odds of obesity; however, independent observers' ratings of the physical disorder in the neighborhood were significantly associated with obesity. Thus, perceptions of disorder, if inaccurate and/or considerably variant, may be expected to have weak associations with physical health indicators. This may not be as applicable to social disorder perceptions, as they may not have as much of a direct environmental effect on health. For example, many physical disorder elements (e.g., noise, dirt, pollution, air quality) are ambient and may affect an individual's health without that individual's conscious awareness of their presence, whereas social disorder elements (e.g., distrust in neighbors, lack of safety, conflicts with neighbors) are more likely to require conscious cognizance by the individual to have an impact.

It is also possible that the measure of physical disorder employed in the current study was insufficiently comprehensive, in that it may not have tapped into certain aspects of the physical neighborhood environment that were most relevant to cardiovascular health. Theoretical models of the influence of neighborhood physical environment on CVD include factors captured in the measure of disorder utilized in the current study such as building appearance and condition, physical decay and noise (Chaix, 2009; Diez Roux, 2003). However, these models also include physical disorder characteristics that were not represented in the measure, such as neighborhood aesthetic quality (e.g., the presence of green spaces, interesting features, and pleasant surroundings), air pollution, condition of sidewalks, and food and tobacco advertising. Other physical disorder characteristics that have been found to relate to CVD risk factors

in previous studies include crowding, traffic speeding, and traffic noise (Leal & Chaix, 2011; Nazmi et al., 2010). Perhaps the inclusion of some of these additional physical disorder characteristics would have resulted in stronger associations between perceptions of physical disorder and CVD risk factors.

Another potential explanation could be that perceptions of physical disorder may not have as much of an impact on CVD risk in this population of Latino women living close to the U.S.-Mexico border as in other populations. It has been posited that the effects of neighborhood disorder on CVD may require chronic exposure to adverse conditions over extended periods of time, perhaps even the lifecourse (Diez Roux, 2007). Although the average length of time participants in the current study reported living in their most recent residence was 10 years (with a range from 1 to 40 years), this may not represent the true degree of exposure participants have had to their current physical neighborhood environments. It is not uncommon for U.S. members of U.S.-Mexico border communities to spend significant amounts of time on the Mexican side of the border, commuting to work, purchasing goods, engaging in dining and entertainment, and visiting friends and family (Anderson & Gerber, 2008). Moreover, the San Diego, CA-Tijuana, MX border crossing is one of the world's busiest land ports of entry, with over 50,000 vehicles and 20,000 pedestrians crossing into the U.S. daily (U.S. General Services Administration, n.d.), and the vast majority of participants in the study (75%) are immigrants, and consequently likely to have familial ties to Mexico. Thus, although in the current study data were not collected on participants' frequency of travel across the border, it is very possible that women in the study spend a considerable amount of time exposed to neighborhood environments other than their current residential environments.

Furthermore, frequent cross-border travel and Mexican nativity could have led study participants to have familiarity and experience with neighborhood environments that on average tend to be more impoverished than those in the U.S. Having exposure to markedly more disadvantaged neighborhood environments may have caused some participants to be less likely to endorse "a lot" of physical disorder in their own neighborhood, even when they may have perceived aspects of disorder to be present. The resultant measurement error may have led to a decreased likelihood of finding a significant effect. Indeed, it has been shown that foreign-born women tend to report lower levels of physical disorder than U.S.-born women, and that Latino women report lower levels of physical disorder than non-Latino Black women (Elo et al., 2009). Therefore, participants' lack of chronic exposure to their current neighborhood environments and/or downgraded endorsement of physical disorder due to familiarity with noticeably more impoverished neighborhood environments could have contributed to the null association found between physical disorder and CVD risk factors in the current study.

The significant associations found between perceptions of social disorder and MAP in age-adjusted models in the current study are generally consistent with findings in the few other studies on the relationship between social disorder and blood pressure. For example, characteristics of social disorder (i.e., crime, drug use) were positively associated with SBP and/or DBP in Turkish and Moroccan ethnic subgroups in a sample of 1286 residents of Amsterdam, Netherlands, and similar (nonsignificant) trends were evident for the native-Dutch subgroup (Agyemang et al., 2007). Aspects of neighborhood social disorder, including greater perceived neighborhood safety and

cohesion were associated with decreased odds of being hypertensive in a sample of 2612 individuals (Mujahid, Diez Roux, Morenoff, et al., 2008), and no differences in the nature of this relationship were found by race/ethnicity (i.e., among non-Latino White, non-Latino Black, and Latino subgroups). The significant association between perceptions of social disorder and total/HDL-c ratio and triglycerides in age-adjusted models in the current study appear to be novel, as no previous studies have evaluated associations between perceptions of social disorder and lipids in Latino or non-Latino samples(Leal & Chaix, 2011). Notably, very few studies have even examined the relationship between *objective* neighborhood factors and lipids. Those that have (Cubbin, Hadden, & Winkleby, 2001; Diez Roux, Jacobs, & Kiefe, 2002; Murakami, Sasaki, Takahashi, & Uenishi, 2010), have reported complex relationships between neighborhood SES and lipids that seem to vary by race/ethnicity and sex. For example, Murukami et al. (2010) reported no association between neighborhood SES and HDL or triglycerides in a sample of young (aged 19 – 22 years) Japanese women; however, Diez Roux et al. (2002) found higher neighborhood SES was associated with greater levels of HDL in White men and women but not in Black men and women, and was associated with lower triglycerides in all groups except Black men. Interestingly, Cubbin et al. (2001) reported a positive association between neighborhood SES and non-HDL cholesterol (i.e., total cholesterol minus HDL cholesterol) for Black men, but no association for Black women, or Mexican-American or White men or women. Consequently, the association between neighborhood factors and lipids appears be complex, and is an area requiring further research attention.

Although in the current study social disorder was not significantly associated with HOMA-IR in age-adjusted models, there appeared to be a trend (p=.058) for an association. A few other studies have found relationships between indicators of social disorder and insulin resistance, including one which found that perceptions of social disorder (including items related to crime and drug use) were positively associated with fasting plasma glucose in a sample of caregivers and noncaregivers (Brummett et al., 2005), and another which reported that Black veterans with diabetes who lived in neighborhoods that were rated higher by residents on an aspect of social capital (i.e., how often residents work together to improve the neighborhood) had better glucose control (as measured by hemoglobin A1c levels) than those that lived in neighborhoods rated poorer on social capital (Long, Field, Armstrong, Chang, & Metlay, 2010). It is possible that neighborhood social disorder may not be as strongly associated with insulin resistance in Latinos as in other racial/ethnic groups, however the lack of studies assessing the social disorder-insulin resistance relationship in both Latino and non-Latino samples precludes any conclusions in this regard. Interestingly, an ethnographic study of Mexican-American diabetics and their caregivers found that factors related to neighborhood social stress (e.g., lack of neighborhood safety) were reported by these individuals as contributing to higher levels of blood glucose (Clark, Vincent, Zimmer, & Sanchez, 2009). Thus, in Latinos, there may be a perception that neighborhood social factors are important determinants of levels of insulin resistance; whether this is borne out empirically is yet to be determined.

The lack of a significant association between perceptions of social disorder and BMI in age-adjusted models in the current study is not surprising given that the literature

to date is equivocal regarding this relationship. The systematic review on neighborhood environments and cardiometabolic risk factors referenced earlier reported that of the 14 studies that evaluated associations between social aspects of neighborhood environments (e.g., social capital, social cohesion, crime, safety) and body weight, 8 found significant associations and 6 did not(Leal & Chaix, 2011) . While no studies to my knowledge have assessed the perceived social disorder-BMI relationship in Latinos specifically, there have been a few that have been conducted in samples including Latinos (comprising 16-25% of the total sample). Of these studies, greater perceived neighborhood social disorder was associated with greater obesity in some (Burdette & Hill, 2008; Burdette et al., 2006), but not in others (Mujahid, Diez Roux, Shen, et al., 2008). Notably, in the Mujahid et al. (2008) study, greater perceived social disorder was unassociated with obesity in women, but was associated with *decreased* risk of obesity in men. Moreover, in this study, unlike in the Burdette and Hill (2008) and Burdette et al. (2006) studies, differences in the relationship between social-disorder and obesity by race/ethnicity (i.e., non-Latino White, non-Latino Black, and Latino) were formally tested, and no differences were found. Therefore, results from the current study and previous studies suggest that the nature of the relationship between perceptions social disorder and obesity in Latinos and non-Latinos is not as yet clear, and that relationships may vary by sex.

Although in age-adjusted models relationships between perceptions of social disorder and several CVD risk factors were apparent, all relationships were attenuated and no longer significant after control for relevant sociodemographic, biological, and medication-related covariates. Of these covariates, individual SES accounted for the majority of the confounding with social disorder. Although control for individual-level

sociodemographic factors has been recommended in order to tease apart the "independent" effects of neighborhood factors from their association with the individual characteristics of people residing in these neighborhoods, there is some debate regarding whether this approach is appropriate (Diez Roux, 2001). For example, Diez Roux (2001) argues that "to the extent that neighborhoods influence the life chances of individuals, neighborhood social and economic characteristics may be related to health through their effects on achieved income, education, and occupation, making these individual-level characteristics mediators (at least in part) rather than confounders" (p. 1786). Conversely, differences in individual-level characteristics could result in differences in individuals' actual personal neighborhood experiences, and accordingly, influence their perceptions of neighborhood characteristics, which in turn may impact their health through stress pathways (Latkin et al., 2009). Indeed, individual factors such as age, marital status, SES (e.g., education and employment status), nativity, and race/ethnicity have all been shown to influence perceptions of social disorder (Elo et al., 2009; Kruger et al., 2007; Sampson & Raudenbush, 2004). In the current sample, perceptions of neighborhood social disorder were correlated $r = -.18$ with SES ($p < .01$). Although some studies have shown significant relationships between neighborhood disorder perceptions and CVD risk factors even after controlling for individual-level variables (Burdette & Hill, 2008; Burdette et al., 2006), other studies, similar to the current study, have found that significant relationships were attenuated to nonsignificance after control for sociodemographic variables (e.g., race/ethnicity; Mujahid et al., 2011; Mujahid, Diez Roux, Morenoff, et al., 2008; Nazmi, et al., 2010). Thus, whether controlling for individual-level characteristics such as SES and race/ethnicity that may shape perceptions

of neighborhood disorder is "over-controlling" and removing a meaningful source of variance in perceptions of disorder that may influence health, or is appropriately controlling for confounding to glean the "independent" effect of perceptions of disorder, remains a matter of debate.

Secondary and tertiary aims of the current study were to examine whether the effects of physical and social disorder on CVD risk factors operated indirectly through behavioral and psychological pathways. No indirect effects were found to be significant. This is not surprising given the lack of a direct association between perceptions of physical or social disorder and CVD risk factors in fully-adjusted models. Additionally, correlations between the majority of the behavioral and psychological factors and the CVD risk factors (with the exception of physical activity) were relatively small in magnitude (absolute value of *r*s .00-.16). Consequently, it is possible that the behavioral and psychological factors examined do not play a large part in determining CVD risk in this sample of middle-aged healthy Latino women, or alternatively, that the self-report measures of behavioral and/or psychological factors employed do not adequately capture the influence of these factors. The use of more objective or comprehensive measures of behavioral factors (e.g., 24-hour dietary recalls, pedometers for physical activity, accelerometers or actigraphs for sleep) or psychological factors may have been preferable. Few previous studies have assessed for indirect effects of neighborhood disorder on CVD risk factors via behavioral and/or psychological pathways, however, one exception is a study by Burdette and Hill (2008), which found that diet quality (rated from poor to excellent), exercise (exercising moderately/vigorously or not), and psychological distress (i.e., symptoms of stress and depression) partially mediated the

association between perceptions of neighborhood disorder and obesity. Other studies have reported indirect effects of neighborhood social disorder on acute MI mortality via smoking (Chaix et al., 2008) and on CAC via depression (Kim et al., 2010). Hence, there is some preliminary evidence that behavioral and psychological factors may serve as indirect pathways through which perceptions of disorder relate to CVD risk. Although no indirect relationships were found in the current study, the employment of more objective or comprehensive measures of these factors in future studies may better test whether indirect effects exist in Latino populations.

Finally, in sensitivity analyses, whether the association between perceptions of neighborhood physical and social disorder and CVD risk factors differed by levels of objective neighborhood SES was examined. A neighborhood social disorder by neighborhood SES interaction effect on MAP approached significance (p=.053). Results indicated a trend for a stronger association between perceptions of social disorder and MAP with increasing neighborhood SES. This finding is somewhat surprising considering that levels of social disorder, as to be expected, were lower in neighborhoods with greater SES (r= -.35, p<.001). Women in lower SES neighborhoods may be more likely to come from environments where they have had frequent exposure to high levels of social disorder during their lifecourse, and thus, these women may have a higher threshold for perceiving social disorder around them, or may be less psychologically and physiologically impacted by social disorder when it does occur than women who come from environments where the experience of social disorder is more rare (Mormede, Dantzer, Michaud, Kelley, & Le Moal, 1988; Sapolsky, 1994; Schneiderman, McCabe, & Baum, 1992). In addition, Latino women in neighborhoods with greater SES may have

more difficulty coping with chronic neighborhood social stressors, and therefore display greater physiological reactivity when they do occur, due to greater overall social stressor burden from social stressors related to "non-traditional" gender role life choices (e.g., decisions not to marry or have children; Galanti, 2003), placement in demanding high-level positions, and greater social pressures to balance multiple societal roles (e.g., employment, familial and domestic responsibilities). The interaction found in the current study suggests a potential complex interplay between objective neighborhood SES and subjective perceptions of neighborhood social disorder on blood pressure. However, the replicability of and causes for this finding need to be explored in future research, particularly given that the finding only approached statistical significance, and that no other neighborhood SES by social disorder interaction effects were identified.

Limitations and Future Research Directions

The current study possesses several strengths, including utilizing integrated measures of neighborhood disorder that tap into diverse aspects of social (e.g., crime, poor social relations, substance use) and physical (structural and environmental deterioration, ambient noise, lack of cleanliness, destruction of property) disorder, examining multiple indicators of CVD risk that have previously been largely unevaluated in relation to neighborhood disorder, and assessing the neighborhood disorder-CVD risk factor association in an understudied, but important ethnic minority group of middle-aged Latino women. However, methodological limitations must also be considered in interpreting the current findings.

Due to the cross-sectional nature of the study, conclusions cannot be drawn regarding the temporal nature or causal associations of effects reported. It is possible,

although less intuitive, that women's cardiovascular health status influenced their perceptions of neighborhood social disorder rather than that perceived disorder predicted cardiovascular health. In order to better elucidate directionality of the effects, prospective study designs should be employed that examine whether changes in perceptions of neighborhood disorder over time predict changes in CVD risk factors. The perceived social disorder-CVD risk findings reported were also only significant in age-adjusted models and not fully-adjusted models, and consequently, confounding by individual SES cannot be ruled out. Though beyond the scope of the current study, future research could examine perceived disorder-CVD risk relationships by differing levels of individual SES in order to more clearly disentangle the effects of each predictor on CVD risk. The sample size for the current study, although adequately powered to detect small-to-medium size effects, was relatively small compared to those in typical epidemiological studies. It is possible that with a larger sample size, there could be increased power to detect effects of perceived neighborhood disorder on CVD risk factors independent of individual SES. Moreover, the sample included in the current study was selected to be "healthy" (i.e., free from any cardiovascular diseases, diabetes, hypertension, kidney disease, chronic inflammatory conditions, or other major illnesses), and thus, effects seen could be conservative estimates of effects in less healthy populations.

The measures of behavioral factors employed were all self-report, and thus prone to measurement error including biases associated with inaccurate recall. The use of more objective measures, such as 24-hour dietary recalls, pedometers for physical activity, and accelerometers or actigraphs for sleep may reduce error variance associated with these behavioral factors, and perhaps result in stronger and more detectable indirect effects.

Although the ethnic and gender composition of the current sample was a strength in that it allowed for the assessment of neighborhood disorder-CVD risk relationships in an important and understudied group, it also precludes the ability to draw conclusions about the nature of this relationship in other ethnic and gender groups. Thus, whether results of the current study generalize to men and other ethnic groups (including other Latino subgroups) should be examined further. Moreover, the sample was distinctive in that it was comprised of women who reside in communities near the U.S.-Mexico border. Consequently, the degree of exposure to varying levels of social and physical disorder that these women may have experienced given their proximity to Mexico, and the fact that a large majority were immigrants, could be different than Latino women living in non-border communities.

The current study examined markers of CVD risk that represented relevant pathways through which perceived neighborhood disorder could impact cardiovascular health based on previous research, however, there are other markers that could also potentially be relevant. For example, inflammatory markers such as IL-6 and CRP have been associated with CVD (Bisoendial, Boekholdt, Vergeer, Stroes, & Kastelein, 2010), and may represent a pathway by which neighborhood disorder relates to CVD. The few studies (Nazmi et al., 2010; Schootman et al., 2010) that have examined associations of perceived neighborhood disorder with inflammatory markers have yielded inconsistent results, and further research is needed. Other, more precise indicators of body composition, such as percentage fat and lean mass (De Lorenzo, Mukherjee, Kadziola, Suleiman, & Kakkar, 1998; Sharp, Andrew, Burchfiel, Violanti, & Wactawski-Wende, 2012) could also be evaluated in relation to neighborhood disorder. Additionally, as

stress has been conceptualized as a pathway through which perceptions of neighborhood disorder contribute to CVD risk, physiological markers of stress such as catecholamines and cortisol are relevant outcomes for future research (Do et al., 2011; Dulin-Keita, Casazza, Fernandez, Goran, & Gower, 2012).

A strength of the current study was the ability to separately, but simultaneously examine the effects of perceived physical and social disorder on CVD risk factors, and results were found to be markedly different across neighborhood disorder types. This is not surprising given that they represent two substantively different types of neighborhood stress that could have differential impacts on cardiovascular health. Although a few studies do make distinctions between these two types of neighborhood disorder and examine their unique effects on health outcomes (e.g., Nazmi et al., 2010), the majority combines various aspects of each into a measure of overall neighborhood disorder (e.g., Burdette & Hill, 2008). Combining these measures precludes the ability to determine which aspects of neighborhood disorder are associated with the CVD outcome being examined. Future studies should evaluate separate effects of social and physical disorder on CVD risk, and might also consider subdimensions within each type of disorder (e.g., aspects of social disorder associated with neighbor relations versus aspects related to crime/violence). In addition, examining each disorder type simultaneously can help to elucidate which has a greater impact on CVD risk in specific populations (e.g., ethnic, gender subgroups; Ross & Mirowsky, 2001).

Results from the current study suggest that there may be a complex interplay between objective neighborhood SES and subjective markers of neighborhood disorder on CVD risk that warrants further examination in future research. Whether other

"objective" neighborhood-level factors that may be relevant to health, such as neighborhood residential segregation and neighborhood density change the nature of the relationship between perceptions of disorder and CVD risk could also be informative to explore (Diez Roux, 2001; Inagami et al., 2006). Moreover, a comparison of how "objective" assessments of neighborhood disorder (e.g., based on violent crime rate, calls to city agencies about street problems, number of off-site liquor licenses; Augustin et al., 2008) relate to CVD risk versus "subjective" perceptions of disorder would help to determine the degree to which perceptions are important in conferring CVD risk. For example, in one study, ratings of physical disorder (e.g., graffiti, litter, broken glass) by residents was unrelated to odds of obesity; however, independent observers' ratings of physical disorder (e.g., presence of beer/liquor bottles or cans, garbage/litter/broken glass, abandoned cars, graffiti and broken windows) were associated with obesity (Boehmer et al., 2007). Consequently, the incorporation of both objective and subjective indicators of neighborhood characteristics in future studies will help to clarify how relevant perceptions of neighborhood disorder are for impacting CVD risk over and above objective markers of disorder, and will aid in understanding the objective neighborhood-level factors than may enhance or attenuate relationships between perceived disorder and CVD risk factors.

Conclusions

The current study provides preliminary evidence of associations between greater perceptions of neighborhood social disorder and greater levels of CVD risk, as indicated by markers of lipids (total/HDL-c; triglycerides) and blood pressure (MAP) in Latino women. However, whether these associations are independent of the effects of individual

SES on CVD risk is not clear. Perceptions of physical disorder do not appear to relate to increased CVD risk in this sample of Latino women. Additionally, neither social nor physical disorder effects on CVD markers appear to operate through indirect behavioral and psychological pathways. Overall, these findings highlight the complexity of the associations between perceptions of disorder and CVD risk in Latino women and underscore the need for additional work in this and other vulnerable populations to better understand the relevance of perceptions of neighborhood characteristics for CVD risk over and above objective markers of these same characteristics. Such efforts may be instrumental in elucidating how neighborhood environments impact cardiovascular health, and may eventually inform the most efficient way in which to target interventional efforts (e.g., at social versus physical aspects of disorder; focus on changing perceptions versus actual conditions).

References

Acquadro, C., Conway, K., Giroudet, C., & Mear, I. (2006). Linguistic validation manual for patient-reported outcomes (PRO) instruments. *Hispanic Journal of Behavioral Science, 28*(4), 498-515.

Acquadro, C., Jambon, B., Ellis, D., & Marquis, P. (1996). Language and translation issues. In B. Spilker (Ed.), *Quality of life and pharmacoeconomics in clinical trials* (pp. 575-585). Philadelphia, PA: Lippincott-Raven Publishers.

Adameova, A., Abdellatif, Y., & Dhalla, N. S. (2009). Role of the excessive amounts of circulating catecholamines and glucocorticoids in stress-induced heart disease. *Canadian Journal of Physiology and Pharmacology, 87*(7), 493-514. doi:10.1139/y09-042

Agyemang, C., van Hooijdonk, C., Wendel-Vos, W., Ujcic-Voortman, J. K., Lindeman, E., Stronks, K., & Droomers, M. (2007). Ethnic differences in the effect of environmental stressors on blood pressure and hypertension in the Netherlands. *BMC Public Health, 7*, 118-128.

Andersen, L. B., Boreham, C. A., Young, I. S., Davey Smith, G., Gallagher, A. M., Murray, L., & McCarron, P. (2006). Insulin sensitivity and clustering of coronary heart disease risk factors in young adults. The Northern Ireland young hearts study. *Preventive Medicine, 42*(1), 73-77. doi:10.1016/j.ypmed.2005.10.009

Anderson, J. B., & Gerber, J. (2008). *Fifty years of change on the U.S.-Mexico border: Growth, development, and quality of life*. Austin, TX: University of Texas Press.

Arsenault, B. J., Rana, J. S., Stroes, E. S., Despres, J. P., Shah, P. K., Kastelein, J. J., … Khaw, K. T. (2009). Beyond low-density lipoprotein cholesterol: Respectivecontributions of non-high-density lipoprotein cholesterol levels, triglycerides, and the total cholesterol/high-density lipoprotein cholesterol ratio to coronary heart disease risk in apparently healthy men and women. *Journal of the American College of Cardiology, 55*(1), 35-41. doi:10.1016/j.jacc.2009.07.057

Augustin, T., Glass, T. A., James, B. D., & Schwartz, B. S. (2008). Neighborhood psychosocial hazards and cardiovascular disease: The Baltimore memory study. *American Journal of Public Health, 98*(9), 1664-1670. doi:10.2105/ajph.2007.125138

Bairey Merz, C. N., Dwyer, J., Nordstrom, C. K., Walton, K. G., Salerno, J. W., & Schneider, R. H. (2002). Psychosocial stress and cardiovascular disease: Pathophysiological links. *Behavioral Medicine, 27*(4), 141-147.

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality & Social Psychology, 51*(6), 1173-1182.

Bazzano, L. A. (2008). Effects of soluble dietary fiber on low-density lipoprotein cholesterol and coronary heart disease risk. *Current Atherosclerosis Reports, 10*(6), 473-477.

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107*(2), 238-246. doi: 10.1037/0033-2909.107.2.238

Biasucci, L. M., Biasillo, G., & Stefanelli, A. (2010). Inflammatory markers, cholesterol and statins: Pathophysiological role and clinical importance. *Clinical Chemistry and Laboratory Medicine, 48*(12), 1685-1691. doi:10.1515/CCLM.2010.277

Bisoendial, R. J., Boekholdt, S. M., Vergeer, M., Stroes, E. S., & Kastelein, J. J. (2010). C-reactive protein is a mediator of cardiovascular disease. *European Heart Journal, 31*(17), 2087-2091. doi: 10.1093/eurheartj/ehq238

Black, P. H., & Garbutt, L. D. (2002). Stress, inflammation and cardiovascular disease. *Journal of Psychosomatic Research, 52*(1), 1-23.

Block, G., Gillespie, C., Rosenbaum, E. H., & Jenson, C. (2000). A rapid food screener to assess fat and fruit and vegetable intake. *American Journal of Preventive Medicine, 18*(4), 284-288.

Boehmer, T. K., Hoehner, C. M., Deshpande, A. D., Brennan Ramirez, L. K., & Brownson, R. C. (2007). Perceived and observed neighborhood indicators of obesity among urban adults. *International Journal of Obesity (London), 31*(6), 968-977. doi:10.1038/sj.ijo.0803531

Bond Huie, S. A., Hummer, R. A., & Rogers, R. G. (2002). Individual and contextual risks of death among race and ethnic groups in the United States. *Journal of Health and Social Behavior, 43*(3), 359-381.

Bonora, E., Formentini, G., Calcaterra, F., Lombardi, S., Marini, F., Zenari, L., … Muggeo, M. (2002). HOMA-estimated insulin resistance is an independent predictor of cardiovascular disease in type 2 diabetic subjects: prospective data from the Verona Diabetes Complications Study. *Diabetes Care, 25*(7), 1135-1141.

Bonora, E., Kiechl, S., Willeit, J., Oberhollenzer, F., Egger, G., Meigs, J. B., … Muggeo, M. (2007). Insulin resistance as estimated by homeostasis model assessment predicts incident symptomatic cardiovascular disease in Caucasian subjects from the general population: The Bruneck study. *Diabetes Care, 30*(2), 318-324.

doi:10.2337/dc06-0919

Bromberger, J. T., & Matthews, K. A. (1996). A longitudinal study of the effects of pessimism, trait anxiety, and life stress on depressive symptoms in middle-aged women. *Psychology & Aging, 11*(2), 207-213.

Brummett, B. H., Siegler, I. C., Rohe, W. M., Barefoot, J. C., Vitaliano, P. P., Surwit, R. S., … Williams, R. B. (2005). Neighborhood characteristics moderate effects of caregiving on glucose functioning. *Psychosomatic Medicine, 67*(5), 752-758. doi:10.1097/01.psy.0000174171.24930.11

Burdette, A. M., & Hill, T. D. (2008). An examination of processes linking perceived neighborhood disorder and obesity. *Social Science and Medicine, 67*(1), 38-46. doi:10.1016/j.socscimed.2008.03.029

Burdette, H. L., Wadden, T. A., & Whitaker, R. C. (2006). Neighborhood safety, collective efficacy, and obesity in women with young children. *Obesity (Silver Spring), 14*(3), 518-525. doi:10.1038/oby.2006.67

Buysse, D. J., Reynolds, C. F., III, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh sleep quality index: A new instrument for psychiatric practice and research. *Psychiatry Research, 28*(2), 193-213. doi:0165-1781(89)90047-4

Cagney, K. A., Glass, T. A., Skarupski, K. A., Barnes, L. L., Schwartz, B. S., & Mendes de Leon, C. F. (2009). Neighborhood-level cohesion and disorder: measurement and validation in two older adult urban populations. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences, 64*(3), 415-424. doi:10.1093/geronb/gbn041

Cappuccio, F. P., Cooper, D., D'Elia, L., Strazzullo, P., & Miller, M. A. (2011). Sleep duration predicts cardiovascular outcomes: A systematic review and meta-analysis of prospective studies. *European Heart Journal, 32*(12), 1484-1492. doi:10.1093/eurheartj/ehr007

Carey, V. J., Bishop, L., Laranjo, N., Harshfield, B. J., Kwiat, C., & Sacks, F. M. (2010). Contribution of high plasma triglycerides and low high-density lipoprotein cholesterol to residual risk of coronary heart disease after establishment of low-density lipoprotein cholesterol control. *American Journal of Cardiology, 106*(6), 757-763. doi:10.1016/j.amjcard.2010.05.002

Chaix, B. (2009). Geographic life environments and coronary heart disease: A literature review, theoretical contributions, methodological updates, and a research agenda. *Annual Review of Public Health, 30*, 81-105.

Chaix, B., Lindstrom, M., Rosvall, M., & Merlo, J. (2008). Neighbourhood social interactions and risk of acute myocardial infarction. *Journal of Epidemiology and*

Community Health, 62(1), 62-68. doi: 10.1136/jech.2006.056960

Chaix, B., Merlo, J., Evans, D., Leal, C., & Havard, S. (2009). Neighbourhoods in eco-epidemiologic research: Delimiting personal exposure areas. A response to Riva, Gauvin, Apparicio and Brodeur. *Social Science and Medicine, 69*(9), 1306-1310. doi:10.1016/j.socscimed.2009.07.018

Chaix, B., Rosvall, M., & Merlo, J. (2007a). Neighborhood socioeconomic deprivation and residential instability: Effects on incidence of ischemic heart disease and survival after myocardial infarction. *Epidemiology, 18*(1), 104-111

Chaix, B., Rosvall, M., & Merlo, J. (2007b). Recent increase of neighborhood socioeconomic effects on ischemic heart disease mortality: A multilevel survival analysis of two large swedish cohorts. *American Journal of Epidemiology, 165*(1), 22-26.

Chen, F. F. (2007). Sensitivity of goodness of fit indexes to lack of measurement invariance. *Structural Equation Modeling: A Multidisciplinary Journal, 14*(3), 464-504.

Chida, Y., & Steptoe, A. (2010). Greater cardiovascular responses to laboratory mental stress are associated with poor subsequent cardiovascular risk status: A meta-analysis of prospective evidence. *Hypertension, 55*(4), 1026-1032. doi:10.1161/HYPERTENSIONAHA.109.146621

Chirovsky, D. R., Fedirko, V., Cui, Y., Sazonov, V., & Barter, P. (2009). Prospective studies on the relationship between high-density lipoprotein cholesterol and cardiovascular risk: A systematic review. *European Journal of Cardiovascular Prevention and Rehabilitation, 16*(4), 404-423. doi:10.1097/HJR.0b013e32832c8891

Clark, L., Vincent, D., Zimmer, L., & Sanchez, J. (2009). Cultural values and political economic contexts of diabetes among low-income Mexican Americans. *Journal of Transcultural Nursing, 20*(4), 382-394. doi:10.1177/1043659609334851

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*(4), 385-396.

Cohen, S., Schwartz, J. E., Bromet, E. J., & Parkinson, D. K. (1991). Mental health, stress, and poor health behaviors in two community samples. *Preventive Medicine, 20*(2), 306-315.

Cohen, S., & Williamson, G. M. (1988). Perceived stress in a probability sample of the United States. In S. Spacapan & S. Oskamp (Eds.), *The social psychology of health* (pp. 31-67). Newbury Park, CA: Sage.

Conen, D., & Bamberg, F. (2008). Noninvasive 24-h ambulatory blood pressure and cardiovascular disease: A systematic review and meta-analysis. *Journal of Hypertension, 26*(7), 1290-1299.

Conroy, R. M., Pyorala, K., Fitzgerald, A. P., Sans, S., Menotti, A., De Backer, G., … Graham, I. M. (2003). Estimation of ten-year risk of fatal cardiovascular disease in Europe: The SCORE project. *European Heart Journal, 24*(11), 987-1003.

Craig, C. L., Marshall, A. L., Sjostrom, M., Bauman, A. E., Booth, M. L., Ainsworth, B. E., … Validity Study, G. (2003). International Physical Activity Questionnaire (IPAQ): 12-country reliability and validity. *Medical Science and Sports Exercise, 35*, 1381-1395.

Criqui, M. H. (1998). Triglycerides and cardiovascular disease. A focus on clinical trials. *European Heart Journal, 19*(Suppl A), A36-39.

Criqui, M. H. (2007). Triglycerides and coronary heart disease revisited (again). *Annals of Internal Medicine, 147*(6), 425-427.

Cubbin, C., Hadden, W. C., & Winkleby, M. A. (2001). Neighborhood context and cardiovascular disease risk factors: The contribution of material deprivation. *Ethnicity and Disease, 11*(4), 687-700.

Cubbin, C., Sundquist, K., Ahlen, H., Johansson, S. E., Winkleby, M. A., & Sundquist, J. (2006). Neighborhood deprivation and cardiovascular disease risk factors: protective and harmful effects. *Scandinavian Journal of Public Health, 34*(3), 228-237. doi:10.1080/14034940500327935

Cubbin, C., & Winkleby, M. A. (2005). Protective and harmful effects of neighborhood-level deprivation on individual-level health knowledge, behavior changes, and risk of coronary heart disease. *American Journal of Epidemiology, 162*(6), 559-568.

Danzi, S., & Klein, I. (2003). Thyroid hormone and blood pressure regulation. *Current Hypertension Reports, 5*(6), 513-520.

De Lorenzo, F., Mukherjee, M., Kadziola, Z., Suleiman, S., & Kakkar, V. V. (1998). Association of overall adiposity rather than body mass index with lipids and procoagulant factors. *Thrombosis and Haemostasis, 80*(4), 603-606.

Deguen, S., Lalloue, B., Bard, D., Havard, S., Arveiler, D., & Zmirou-Navier, D. (2010). A small-area ecologic study of myocardial infarction, neighborhood deprivation, and sex: A Bayesian modeling approach. *Epidemiology, 21*(4), 459-466. doi:10.1097/EDE.0b013e3181e09925

Delahoy, P. J., Magliano, D. J., Webb, K., Grobler, M., & Liew, D. (2009). The relationship between reduction in low-density lipoprotein cholesterol by statins and reduction in risk of cardiovascular outcomes: An updated meta-analysis. *Clinical Therapeutics, 31*(2), 236-244. doi:10.1016/j.clinthera.2009.02.017

Diez Roux, A. V. (2001). Investigating neighborhood and area effects on health. *American Journal of Public Health, 91*(11), 1783-1789.

Diez Roux, A. V. (2003). Residential environments and cardiovascular risk. *Journal of Urban Health, 80*(4), 569-589. doi:10.1093/jurban/jtg065

Diez Roux, A. V. (2007). Neighborhoods and health: Where are we and were do we go from here? *Revue d' Epidemiologie et de Sante Publique, 55*(1), 13-21. doi:10.1016/j.respe.2006.12.003

Diez Roux, A. V., Jacobs, D. R., & Kiefe, C. I. (2002). Neighborhood characteristics and components of the insulin resistance syndrome in young adults: The coronary artery risk development in young adults (CARDIA) study. *Diabetes Care, 25*(11), 1976-1982.

Diez Roux, A. V., Merkin, S. S., Arnett, D., Chambless, L., Massing, M., Nieto, F. J., … Watson, R. L. (2001). Neighborhood of residence and incidence of coronary heart disease. *New England Journal of Medicine, 345*(2), 99-106.

Dimsdale, J. E. (2008). Psychological stress and cardiovascular disease. *Journal of the American College of Cardiology, 51*(13), 1237-1246. doi:10.1016/j.jacc.2007.12.024

Do, D. P., Diez Roux, A. V., Hajat, A., Auchincloss, A. H., Merkin, S. S., Ranjit, N., … Seeman, T. (2011). Circadian rhythm of cortisol and neighborhood characteristics in a population-based sample: the Multi-Ethnic Study of Atherosclerosis. *Health Place, 17*(2), 625-632. doi:10.1016/j.healthplace.2010.12.019

Dulin-Keita, A., Casazza, K., Fernandez, J. R., Goran, M. I., & Gower, B. (2012). Do neighbourhoods matter? Neighbourhood disorder and long-term trends in serum cortisol levels. *Journal of Epidemiology and Community Health, 66*(1), 24-29. doi:10.1136/jech.2009.092676

Eaton, W. W., Muntaner, C., Smith, C., Tien, A., & Ybarra, M. (1999). Center for Epidemiologic Studies Depression Scale: Review and revision (CESD and CESDR). In J. Masten (Ed.), *The use of psychological testing for treatment planning and outcomes assessment* (pp. 363-377). Mahwah, NJ: Lawrence Erlbaum.

Echeverria, S., Diez-Roux, A. V., Shea, S., Borrell, L. N., & Jackson, S. (2008).

Associations of neighborhood problems and neighborhood social cohesion with mental health and health behaviors: The Multi-Ethnic Study of Atherosclerosis. *Health Place, 14*(4), 853-865. doi:10.1016/j.healthplace.2008.01.004

Elder, J. P., Ayala, G. X., Campbell, N. R., Slymen, D., Lopez-Madurga, E. T., Engelberg, M., & Baquero, B. (2005). Interpersonal and print nutrition communication for a Spanish-dominant Latino population: Secretos de la buena vida. *Health Psychology, 24*(1), 49-57.

Ellaway, A., & Macintyre, S. (2009). Are perceived neighbourhood problems associated with the likelihood of smoking? *Journal of Epidemiology and Community Health, 63*(1), 78-80. doi:10.1136/jech.2007.068767

Ellaway, A., Macintyre, S., & Bonnefoy, X. (2005). Graffiti, greenery, and obesity in adults: Secondary analysis of European cross sectional survey. *BMJ, 331*(7517), 611-612. doi:10.1136/bmj.38575.664549.F7

Elo, I. T., Mykyta, L., Margolis, R., & Culhane, J. F. (2009). Perceptions of neighborhood disorder: The role of individual and neighborhood characteristics. *Social Science Quarterly, 90*(5), 1298. doi:10.1111/j.1540-6237.2009.00657.x

Erhardt, L. (2009). Cigarette smoking: an undertreated risk factor for cardiovascular disease. *Atherosclerosis, 205*(1), 23-32. doi:10.1016/j.atherosclerosis.2009.01.007

Ervin, R. B. (2009). Prevalence of metabolic syndrome among adults 20 years of age and over, by sex, age, race and ethnicity, and body mass index: United States, 2003-2006. *National Health Stat Report, 13*, 1-7.

Eschbach, K., Mahnken, J. D., & Goodwin, J. S. (2005). Neighborhood composition and incidence of cancer among Hispanics in the United States. *Cancer, 103*(5), 1036-1044. doi:10.1002/cncr.20885

Eschbach, K., Ostir, G. V., Patel, K. V., Markides, K. S., & Goodwin, J. S. (2004). Neighborhood context and mortality among older Mexican Americans: Is there a barrio advantage? *American Journal of Public Health, 94*(10), 1807-1812. doi:10.2105/ajph.94.10.1807

Espinosa de los Monteros, K., Gallo, L. C., Elder, J. P., & Talavera, G. A. (2008). Individual and area-based indicators of acculturation and the Metabolic Syndrome in low income, border region Mexican-American women. *American Journal of Public Health, 98*(11), 1979-1986.

Fernandez, M. L. (2001). Soluble fiber and nondigestible carbohydrate effects on plasma lipids and cardiovascular risk. *Current Opinion in Lipidology, 12*(1), 35-40.

Foraker, R. E., Rose, K. M., Kucharska-Newton, A. M., Ni, H., Suchindran, C. M., & Whitsel, E. A. (2011). Variation in rates of fatal coronary heart disease by neighborhood socioeconomic status: The atherosclerosis risk in communities surveillance (1992-2002). *Annals of Epidemiology, 21*(8), 580-588. doi: 10.1016/j.annepidem.2011.03.004

Foster, S., & Giles-Corti, B. (2008). The built environment, neighborhood crime and constrained physical activity: An exploration of inconsistent findings. *Preventive Medicine, 47*(3), 241-251. doi:10.1016/j.ypmed.2008.03.017

Franzini, L., Caughy, M., Spears, W., & Esquer, M. E. F. (2005). Neighborhood economic conditions, social processes, and self-rated health in low-income neighborhoods in Texas: A multilevel latent variables model. *Social Science & Medicine, 61*, 1135-1150.

Fritz, M. S., & Mackinnon, D. P. (2007). Required sample size to detect the mediated effect. *Psychology Science, 18*(3), 233-239. doi:10.1111/j.1467-9280.2007.01882.x

Galanti, G. A. (2003). The Hispanic family and male-female relationships: An overview. *Journal of Transcultural Nursing, 14*(3), 180-185.

Gallo, L. C., Jimenez, J. A., Shivpuri, S., Espinosa de Los Monteros, K., & Mills, P. J. (2011). Domains of chronic stress, lifestyle factors, and allostatic load in middle-aged Mexican-American women. *Annals of Behavioral Medicine, 41*(1), 21-31. doi:10.1007/s12160-010-9233-1

Gandotra, P., & Miller, M. (2008). The role of triglycerides in cardiovascular risk. *Current Cardiology Reports, 10*(6), 505-511.

Gary, T. L., Safford, M. M., Gerzoff, R. B., Ettner, S. L., Karter, A. J., Beckles, G. L., & Brown, A. F. (2008). Perception of neighborhood problems, health behaviors, and diabetes outcomes among adults with diabetes in managed care: The Translating Research Into Action for Diabetes (TRIAD) study. *Diabetes Care, 31*(2), 273-278. doi:10.2337/dc07-1111

Gary, T. L., Stark, S. A., & LaVeist, T. A. (2007). Neighborhood characteristics and mental health among African Americans and Whites living in a racially integrated urban community. *Health Place, 13*(2), 569-575. doi:10.1016/j.healthplace.2006.06.001

Genser, B., & Marz, W. (2006). Low density lipoprotein cholesterol, statins and cardiovascular events: A meta-analysis. *Clinical Research in Cardiology, 95*(8), 393-404. doi:10.1007/s00392-006-0403-x

Gerber, Y., Benyamini, Y., Goldbourt, U., & Drory, Y. (2010). Neighborhood socioeconomic context and long-term survival after myocardial infarction. *Circulation, 121*(3), 375-383. doi: 10.1161/CIRCULATIONAHA.109.882555

Gerber, Y., Koton, S., Goldbourt, U., Myers, V., Benyamini, Y., Tanne, D., & Drory, Y. (2010). Poor neighborhood socioeconomic status and risk of ischemic stroke after myocardial infarction. *Epidemiology, 22*(2), 162-169. doi: 10.1097/EDE.0b013e31820463a3

Glass, T. A., Rasmussen, M. D., & Schwartz, B. S. (2006). Neighborhoods and obesity in older adults: The Baltimore memory study. *American Journal of Preventive Medicine, 31*(6), 455-463. doi:10.1016/j.amepre.2006.07.028

Godin, G., Jobin, J., & Bouillon, J. (1986). Assessment of leisure time exercise behavior by self-report: A concurrent validity study. *Canadian Journal of Public Health. Revue Canadienne de Sante Publique, 77*(5), 359-362.

Gu, Q., Dillon, C. F., Burt, V. L., & Gillum, R. F. (2010). Association of hypertension treatment and control with all-cause and cardiovascular disease mortality among US adults with hypertension. *American Journal of Hypertension, 23*(1), 38-45. doi:10.1038/ajh.2009.191

Gump, B. B., Matthews, K. A., & Raikkonen, K. (1999). Modeling relationships among socioeconomic status, hostility, cardiovascular reactivity, and left ventricular mass in African American and White children. *Health Psychology, 18*(2), 140-150.

Hadaegh, F., Harati, H., Ghanbarian, A., & Azizi, F. (2006). Association of total cholesterol versus other serum lipid parameters with the short-term prediction of cardiovascular outcomes: Tehran lipid and glucose study. *European Journal of Cardiovascular Prevention and Rehabilitation, 13*(4), 571-577. doi:10.1097/01.hjr.0000216552.81882.ca

Hadaegh, F., Hatami, M., Sheikholeslami, F., & Azizi, F. (2010). Pulse pressure and systolic blood pressure are powerful independent predictors of cardiovascular disease in diabetic adults: Results of an 8.4 years follow-up of Tehran lipid and glucose study. *Experimental and Clinical Endocrinology and Diabetes, 118*(9), 638-643. doi:10.1055/s-0030-1254085

Hadaegh, F., Shafiee, G., Hatami, M., & Azizi, F. (2011). Systolic and diastolic blood pressure, mean arterial pressure and pulse pressure for prediction of cardiovascular events and mortality in a Middle Eastern population. *Blood Pressure*. Advance online publication. doi:10.3109/08037051.2011.585808

Haffner, S., Gonzalez Villalpando, C., Hazuda, H., Valdez, R., Mykkanen, L., & Stern,

M. (1994). Prevalence of hypertension in Mexico City and San Antonio, Texas. *Circulation, 90*(3), 1542-1549.

Haffner, S. M., Miettinen, H., & Stern, M. P. (1997). The homeostasis model in the San Antonio Heart Study. *Diabetes Care, 20*(7), 1087-1092.

Hale, L., Hill, T. D., & Burdette, A. M. (2010). Does sleep quality mediate the association between neighborhood disorder and self-rated physical health? *Preventive Medicine, 51*(3-4), 275-278. doi:10.1016/j.ypmed.2010.06.017

Hamilton, M. T., Hamilton, D. G., & Zderic, T. W. (2007). Role of low energy expenditure and sitting in obesity, metabolic syndrome, type 2 diabetes, and cardiovascular disease. *Diabetes, 56*(11), 2655-2667. doi:10.2337/db07-0882

Hanley, A. J., Williams, K., Stern, M. P., & Haffner, S. M. (2002). Homeostasis model assessment of insulin resistance in relation to the incidence of cardiovascular disease: The San Antonio Heart Study. *Diabetes Care, 25*(7), 1177-1184.

Hausenloy, D. J., & Yellon, D. M. (2008). Targeting residual cardiovascular risk: Raising high-density lipoprotein cholesterol levels. *Heart, 94*(6), 706-714. doi:10.1136/hrt.2007.125401

Hazuda, H. P., Stern, M. P., & Haffner, S. M. (1988). Acculturation and assimilation among Mexican Americans: Scales and population-based data. *Social Science Quarterly, 69*(3), 687-706.

Henson, R. K. (2001). Understanding internal consistency reliability estimates: A conceptual primer on coefficient alpha. *Measurement and Evaluation in Counseling and Development, 34*(3), 177-189.

Heron, M. P., Hoyert, D. L., Murphy, S. L., Xu, J. Q., Kochanek, K. D., & Tejada-Vera, B. (2009). Deaths: Final data for 2006. *National Vital Statistics Reports, 57*(14), 1-135.

Heslop, P., Smith, G. D., Metcalfe, C., Macleod, J., & Hart, C. (2002). Sleep duration and mortality: The effect of short or long sleep duration on cardiovascular and all-cause mortality in working men and women. *Sleep Medicine, 3*(4), 305-314.

Hill, T. D., & Angel, R. J. (2005). Neighborhood disorder, psychological distress, and heavy drinking. *Social Science & Medicine, 61*(5), 975.

Hill, T. D., Burdette, A. M., & Hale, L. (2009). Neighborhood disorder, sleep quality, and psychological distress: Testing a model of structural amplification. *Health Place, 15*(4), 1006-1013. doi:10.1016/j.healthplace.2009.04.001

Hill, T. D., Ross, C. E., & Angel, R. J. (2005). Neighborhood disorder, psychophysiological distress, and health. *Journal of Health & Social Behavior, 46*(2), 170-186.

Hooper, L. (2010). Meta-analysis of RCTs finds that increasing consumption of polyunsaturated fat as a replacement for saturated fat reduces the risk of coronary heart disease. *Evidence-Based Medicine, 15*(4), 108-109. doi:10.1136/ebm1093

Hooper, L., Summerbell, C. D., Thompson, R., Sills, D., Roberts, F. G., Moore, H., & Davey Smith, G. (2011). Reduced or modified dietary fat for preventing cardiovascular disease. *Cochrane Database of Systematic Reviews, 7*, CD002137. doi:10.1002/14651858.CD002137.pub2

Hu, F. B., Manson, J. E., & Willett, W. C. (2001). Types of dietary fat and risk of coronary heart disease: a critical review. *Journal of the American College of Nutrition, 20*(1), 5-19.

Hu, L. T., & Bentler, P. M. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modeling, 6*(1), 1-55. doi:10.1080/10705519909540118

Inagami, S., Borrell, L. N., Wong, M. D., Fang, J., Shapiro, M. F., & Asch, S. M. (2006). Residential segregation and Latino, black and white mortality in New York City. *Journal of Urban Health, 83*(3), 406-420. doi:10.1007/s11524-006-9035-8

Johnson, T. V., Hsiao, W., Jani, A., & Master, V. A. (2011). Increased mortality among Hispanic testis cancer patients independent of neighborhood socioeconomic status: A SEER study. *Journal of Immigrant and Minority Health, 13*(5), 818-824. doi:10.1007/s10903-010-9419-4

Kannel, W. B. (2000). The Framingham study: ITS 50-year legacy and future promise. *Journal of Atherosclerosis Thrombosis, 6*(2), 60-66.

Kannel, W. B., & Larson, M. (1993). Long-term epidemiologic prediction of coronary disease. The Framingham experience. *Cardiology, 82*(2-3), 137-152.

Katzmarzyk, P. T., Church, T. S., Craig, C. L., & Bouchard, C. (2009). Sitting time and mortality from all causes, cardiovascular disease, and cancer. *Medicine and Science in Sports and Exercise, 41*(5), 998-1005. doi:10.1249/MSS.0b013e3181930355

Keegan, T. H., John, E. M., Fish, K. M., Alfaro-Velcamp, T., Clarke, C. A., & Gomez, S. L. (2010). Breast cancer incidence patterns among California Hispanic women: differences by nativity and residence in an enclave. *Cancer Epidemiology, Biomarkers and Prevention, 19*(5), 1208-1218. doi:10.1158/1055-9965.EPI-10-

0021

Keegan, T. H., Quach, T., Shema, S., Glaser, S. L., & Gomez, S. L. (2010). The influence of nativity and neighborhoods on breast cancer stage at diagnosis and survival among California Hispanic women. *BMC Cancer, 10*, 603-614. doi:10.1186/1471-2407-10-603

Keita, A. D., Casazza, K., Thomas, O., & Fernandez, J. R. (2011). Neighborhood perceptions affect dietary behaviors and diet quality. *Journal of Nutrition Education and Behavior, 43*(4), 244-250. doi:10.1016/j.jneb.2009.07.004

Kenny, D. A., Kashy, D. A., & Bolger, N. (1998). Data analysis in social psychology. In D. T. Gilbert & S. T. Fiske (Eds.), *The handbook of social psychology, vol. 2* (pp. 233-265). New York, NY: Oxford University Press.

Kim, D. (2008). Blues from the neighborhood? Neighborhood characteristics and depression. *Epidemiologic Reviews, 30*, 101-117. doi:10.1093/epirev/mxn009

Kim, D., Diez Roux, A. V., Kiefe, C. I., Kawachi, I., & Liu, K. (2010). Do neighborhood socioeconomic deprivation and low social cohesion predict coronary calcification?: The CARDIA study. *American Journal of Epidemiology, 172*(3), 288-298. doi:10.1093/aje/kwq098

Kripke, D. F., Garfinkel, L., Wingard, D. L., Klauber, M. R., & Marler, M. R. (2002). Mortality associated with sleep duration and insomnia. *Archives of General Psychiatry, 59*(2), 131-136.

Kruger, D. J., Reischl, T. M., & Gee, G. C. (2007). Neighborhood social conditions mediate the association between physical deterioration and mental health. *American Journal of Community Psychology, 40*(3-4), 261-271. doi:10.1007/s10464-007-9139-7

Lamarche, B., Despres, J. P., Moorjani, S., Cantin, B., Dagenais, G. R., & Lupien, P. J. (1996). Triglycerides and HDL-cholesterol as risk factors for ischemic heart disease. Results from the Quebec cardiovascular study. *Atherosclerosis, 119*(2), 235-245.

Latkin, C. A., & Curry, A. D. (2003). Stressful neighborhoods and depression: A prospective study of the impact of neighborhood disorder. *Journal of Health and Social Behavior, 44*(1), 44.

Latkin, C. A., German, D., Hua, W., & Curry, A. D. (2009). Individual-level influences on perceptions of neighborhood disorder: A multilevel analysis. *Journal of Community Psychology, 37*(1), 122-133. doi: 10.1002/jcop.20284

Lawes, C. M., Bennett, D. A., Parag, V., Woodward, M., Whitlock, G., Lam, T. H., …

Rodgers, A. (2003). Blood pressure indices and cardiovascular disease in the Asia Pacific region: A pooled analysis. *Hypertension, 42*(1), 69-75. doi:10.1161/01.HYP.0000075083.04415.4B

Lazarus, R. S., & Folkman, S. (1984). *Stress appraisal and coping*. New York, NY: Springer.

Leal, C., Bean, K., Thomas, F., & Chaix, B. (2011). Are associations between neighborhood socioeconomic characteristics and body mass index or waist circumference based on model extrapolations? *Epidemiology, 22*(5), 694-703. doi:10.1097/EDE.0b013e3182257784

Leal, C., & Chaix, B. (2011). The influence of geographic life environments on cardiometabolic risk factors: A systematic review, a methodological assessment and a research agenda. *Obesity Reviews, 12*(3), 217-230. doi:10.1111/j.1467-789X.2010.00726.x

Lee, M. A., & Ferraro, K. F. (2007). Neighborhood residential segregation and physical health among Hispanic Americans: Good, bad, or benign? *Journal of Health and Social Behavior, 48*(2), 131-148. doi:10.1177/002214650704800203 48

Leineweber, C., Kecklund, G., Janszky, I., Akerstedt, T., & Orth-Gomer, K. (2003). Poor sleep increases the prospective risk for recurrent events in middle-aged women with coronary disease. *The Stockholm Female Coronary Risk Study. Journal of Psychosomatic Research, 54*(2), 121-127.

Lewis, C. E., McTigue, K. M., Burke, L. E., Poirier, P., Eckel, R. H., Howard, B. V., … Pi-Sunyer, F. X. (2009). Mortality, health outcomes, and body mass index in the overweight range: A science advisory from the American Heart Association. *Circulation, 119*(25), 3263-3271. doi:10.1161/CIRCULATIONAHA.109.192574

Long, J. A., Field, S., Armstrong, K., Chang, V. W., & Metlay, J. P. (2010). Social capital and glucose control. *Journal of Community Health, 35*(5), 519-526. doi:10.1007/s10900-010-9222-0

MacKinnon, D. P., Fritz, M. S., Williams, J., & Lockwood, C. M. (2007). Distribution of the product confidence limits for the indirect effect: program PRODCLIN. *Behavior Research Methods, 39*(3), 384-389.

MacKinnon, D. P., Lockwood, C. M., & Williams, J. (2004). confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate Behavioral Research, 39*(1), 99-128.

Malhotra, A., & Loscalzo, J. (2009). Sleep and cardiovascular disease: An overview. *Progress in Cardiovascular Diseases, 51*(4), 279-284.

doi:10.1016/j.pcad.2008.10.004
Mancia, G. (2007). Blood pressure reduction and cardiovascular outcomes: past, present, and future. *American Journal of Cardiology, 100*(3A), 3J-9J. doi:10.1016/j.amjcard.2007.05.008 [doi]

Manson, J. E., Willett, W. C., Stampfer, M. J., Colditz, G. A., Hunter, D. J., Hankinson, S. E., … Speizer, F. E. (1995). Body weight and mortality among women. *New England Journal of Medicine, 333*(11), 677-685. doi:10.1056/NEJM199509143331101

Maron, D. J., Fazio, S., & Linton, M. F. (2000). Current perspectives on statins. *Circulation, 101*(2), 207-213.

Matthews, D. R., Hosker, J. P., Rudenski, A. S., Naylor, B. A., Treacher, D. F., & Turner, R. C. (1985). Homeostasis model assessment: Insulin resistance and β-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia, 28*(7), 412-419. doi:10.1007/bf00280883

McBride, P. (2008). Triglycerides and risk for coronary artery disease. *Current Atherosclerosis Reports, 10*(5), 386-390.

McEwen, B. S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine, 338*(3), 171-179.

Mediene-Benchekor, S., Brousseau, T., Richard, F., Benhamamouch, S., & Amouyel, P. (2001). Blood lipid concentrations and risk of myocardial infarction. *Lancet, 358*(9287), 1064-1065. doi:10.1016/S0140-6736(01)06200-6

Meijer, A., Conradi, H. J., Bos, E. H., Thombs, B. D., van Melle, J. P., & de Jonge, P. (2011). Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: A meta-analysis of 25 years of research. *General Hospital Psychiatry,* 33(3), 203-216. doi:10.1016/j.genhosppsych.2011.02.007

Mendes de Leon, C. F., Cagney, K. A., Bienias, J. L., Barnes, L. L., Skarupski, K. A., Scherr, P. A., & Evans, D. A. (2009). Neighborhood social cohesion and disorder in relation to walking in community-dwelling older adults: A multilevel analysis. *Journal of Aging and Health, 21*(1), 155-171. doi:10.1177/0898264308328650

Miller, M. A., & Cappuccio, F. P. (2007). Inflammation, sleep, obesity and cardiovascular disease. *Current Vascular Pharmacology, 5*(2), 93-102.

Miller, D. B., & O'Callaghan, J. P. (2002). Neuroendocrine aspects of the response to stress. *Metabolism: Clinical and Experimental, 51*(6 Suppl 1), 5-10.

Miller, M. A., Stone, N. J., Ballantyne, C., Bittner, V., Criqui, M. H., Ginsberg, H. N., … Pennathur, S. (2011). Triglycerides and cardiovascular disease: A scientific statement from the American Heart Association. *Circulation, 123*(20), 2292-2333. doi:10.1161/CIR.0b013e3182160726

Mobley, L. R., Root, E. D., Finkelstein, E. A., Khavjou, O., Farris, R. P., & Will, J. C. (2006). Environment, obesity, and cardiovascular disease risk in low-income women. *American Journal of Preventive Medicine, 30*(4), 327-332. doi:10.1016/j.amepre.2005.12.001

Mora, S., Rifai, N., Buring, J. E., & Ridker, P. M. (2008). Fasting compared with nonfasting lipids and apolipoproteins for predicting incident cardiovascular events. *Circulation, 118*(10), 993-1001. doi:10.1161/CIRCULATIONAHA.108.777334

Mormede, P., Dantzer, R., Michaud, B., Kelley, K. W., & Le Moal, M. (1988). Influence of stressor predictability and behavioral control on lymphocyte reactivity, antibody responses and neuroendocrine activation in rats. *Physiology and Behavior, 43*(5), 577-583. doi:0031-9384(88)90211-9

Mozaffarian, D., Micha, R., & Wallace, S. (2010). Effects on coronary heart disease of increasing polyunsaturated fat in place of saturated fat: A systematic review and meta-analysis of randomized controlled trials. *PLoS Med, 7*(3), e1000252. doi:10.1371/journal.pmed.1000252

Mozaffarian, D., Wilson, P. W., & Kannel, W. B. (2008). Beyond established and novel risk factors: Lifestyle risk factors for cardiovascular disease. *Circulation, 117*(23), 3031-3038. doi:10.1161/CIRCULATIONAHA.107.738732

Mujahid, M. S., Diez Roux, A. V., Cooper, R. C., Shea, S., & Williams, D. R. (2011). Neighborhood stressors and race/ethnic differences in hypertension prevalence (the Multi-Ethnic Study of Atherosclerosis). *American Journal of Hypertension, 24*(2), 187-193. doi: 10.1038/ajh.2010.200

Mujahid, M. S., Diez Roux, A. V., Morenoff, J. D., Raghunathan, T. E., Cooper, R. S., Ni, H., & Shea, S. (2008). Neighborhood characteristics and hypertension. *Epidemiology, 19*(4), 590-598. doi: 10.1097/EDE.0b013e3181772cb2

Mujahid, M. S., Diez Roux, A. V., Shen, M., Gowda, D., Sanchez, B., Shea, S., … Jackson, S. A. (2008). Relation between neighborhood environments and obesity in the Multi-Ethnic Study of Atherosclerosis. *American Journal of Epidemiology, 167*(11), 1349-1357. doi:10.1093/aje/kwn047

Mulia, N., Schmidt, L., Bond, J., Jacobs, L., & Korcha, R. (2008). Stress, social support and problem drinking among women in poverty. *Addiction, 103*(8), 1283-1293.

doi:10.1111/j.1360-0443.2008.02234.x

Murakami, K., Sasaki, S., Takahashi, Y., & Uenishi, K. (2010). Neighborhood socioeconomic status in relation to dietary intake and insulin resistance syndrome in female Japanese dietetic students. *Nutrition, 26*(5), 508-514. doi:10.1016/j.nut.2009.08.025

Murray, E. T., Diez Roux, A. V., Carnethon, M., Lutsey, P. L., Ni, H., & O'Meara, E. S. (2010). Trajectories of neighborhood poverty and associations with subclinical atherosclerosis and associated risk factors: The multi-ethnic study of atherosclerosis. *American Journal of Epidemiology, 171*(10), 1099-1108. doi: 10.1093/aje/kwq044

Muthén, L. K., & Muthén, B. O. (2006). *Mplus*. Los Angeles, CA: Muthén & Muthén.

Nazmi, A., Diez Roux, A., Ranjit, N., Seeman, T. E., & Jenny, N. S. (2010). Cross-sectional and longitudinal associations of neighborhood characteristics with inflammatory markers: Findings from the multi-ethnic study of atherosclerosis. *Health Place, 16*(6), 1104-1112. doi:10.1016/j.healthplace.2010.07.001

Neeley, W. E. (1972). Simple automated determination of serum or plasma glucose by a hexokinase/glucose-6-phosphate dehydrogenase method. *Clinical Chemistry, 18*(6), 509-515.

Newman, A. B., Spiekerman, C. F., Enright, P., Lefkowitz, D., Manolio, T., Reynolds, C. F., & Robbins, J. (2000). Daytime sleepiness predicts mortality and cardiovascular disease in older adults. The cardiovascular health study research group. *Journal of the American Geriatrics Society, 48*(2), 115-123.

Ng, D. M., & Jeffery, R. W. (2003). Relationships between perceived stress and health behaviors in a sample of working adults. *Health Psychology, 22*(6), 638-642. doi:10.1037/0278-6133.22.6.638

Nordstrom, C. K., Diez Roux, A. V., Jackson, S. A., & Gardin, J. M. (2004). The association of personal and neighborhood socioeconomic indicators with subclinical cardiovascular disease in an elderly cohort. The cardiovascular health study. *Social Science and Medicine, 59*(10), 2139-2147.

Okamura, T., Kokubo, Y., Watanabe, M., Higashiyama, A., Ono, Y., Miyamoto, Y., … Okayama, A. (2010). Triglycerides and non-high-density lipoprotein cholesterol and the incidence of cardiovascular disease in an urban Japanese cohort: The Suita study. *Atherosclerosis, 209*(1), 290-294. doi:10.1016/j.atherosclerosis.2009.09.012

Onat, A., Sari, I., Yazici, M., Can, G., Hergenc, G., & Avci, G. S. (2006). Plasma triglycerides, an independent predictor of cardiovascular disease in men: A prospective study based on a population with prevalent metabolic syndrome.

International Journal of Cardiology, 108(1), 89-95. doi:10.1016/j.ijcard.2005.06.056

Owen, C. G., Whincup, P. H., Orfei, L., Chou, Q. A., Rudnicka, A. R., Wathern, A. K., … Cook, D. G. (2009). Is body mass index before middle age related to coronary heart disease risk in later life? Evidence from observational studies. *International Journal of Obesity (London), 33*(8), 866-877. doi:10.1038/ijo.2009.102

Owen, N., Sparling, P. B., Healy, G. N., Dunstan, D. W., & Matthews, C. E. (2010). Sedentary behavior: Emerging evidence for a new health risk. *Mayo Clinic Proceedings, 85*(12), 1138-1141. doi:10.4065/mcp.2010.0444

Patel, S. R., Ayas, N. T., Malhotra, M. R., White, D. P., Schernhammer, E. S., Speizer, F. E., … Hu, F. B. (2004). A prospective study of sleep duration and mortality risk in women. *Sleep, 27*(3), 440-444.

Pereira, M. A., & Pins, J. J. (2000). Dietary fiber and cardiovascular disease: Experimental and epidemiologic advances. *Current Atherosclerosis Reports, 2*(6), 494-502.

Poortinga, W. (2006). Perceptions of the environment, physical activity, and obesity. *Social Science and Medicine, 63*(11), 2835-2846. doi:10.1016/j.socscimed.2006.07.018

Preacher, K. J., & Hayes, A. F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments & Computers, 36*(4), 731.

Preacher, K. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods, 40*(3), 879-891. doi:10.3758/brm.40.3.879

Preacher, K. J., Curran, P. J., & Bauer, D. J. (2006). Computational tools for probing interactions in multiple linear regression, multilevel modeling, and latent curve analysis. *Journal of Educational and Behavioral Statistics, 31*(4), 437-448.

Ramachandruni, S., Handberg, E., & Sheps, D. S. (2004). Acute and chronic psychological stress in coronary disease. *Current Opinion in Cardiology, 19*(5), 494-499.

Reyes-Ortega, M., Soto-Hernandez, A. L., Milla-Kegel, J. G., Garcia-Ramirez, A., Hubard-Vignau, L., Mendoza-Sanchez, H., … Wagner-Echeagaray, F. A. (2003). Revision of the Center for Epidemiologic Studies Depression Scale (CES-D). Pilot study with a Mexican geriatric sample. *Salud Mental, 26*(1), 59-68.

Reyes-Ortiz, C. A., Eschbach, K., Zhang, D. D., & Goodwin, J. S. (2008). Neighborhood composition and cancer among Hispanics: Tumor stage and size at time of diagnosis. *Cancer Epidemiology, Biomarkers and Prevention, 17*(11), 2931-2936. doi:10.1158/1055-9965.EPI-07-0430

Rojas, R., Aguilar-Salinas, C. A., Jimenez-Corona, A., Shamah-Levy, T., Rauda, J., Avila-Burgos, L., … Ponce, E. L. (2010). Metabolic syndrome in Mexican adults: Results from the National Health and Nutrition Survey 2006. *Salud Publica Mexico, 52*(Suppl 1), S11-18. doi:S0036-36342010000700004

Rose, K. M., Suchindran, C. M., Foraker, R. E., Whitsel, E. A., Rosamond, W. D., Heiss, G., & Wood, J. L. (2009). Neighborhood disparities in incident hospitalized myocardial infarction in four U.S. communities: The ARIC surveillance study. *Annals of Epidemiology, 19*(12), 867-874. doi: 10.1016/j.annepidem.2009.07.092

Rosengren, A., Hawken, S., Ounpuu, S., Sliwa, K., Zubaid, M., Almahmeed, W. A., … Yusuf, S. (2004). 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, 364*(9438), 953-962.

Rosengren, A., Wedel, H., & Wilhelmsen, L. (1999). Body weight and weight gain during adult life in men in relation to coronary heart disease and mortality. A prospective population study. *European Heart Journal, 20*(4), 269-277.

Ross, C. E., & Mirowsky, J. (1999). Disorder and decay. *Urban Affairs Review, 34*(3), 412-432. doi: 10.1177/107808749903400304

Ross, C. E., & Mirowsky, J. (2001). Neighborhood disadvantage, disorder, and health. *Journal of Health & Social Behavior, 42*(3), 258-276.

Ross, C. E., & Mirowsky, J. (2009). Neighborhood disorder, subjective alienation, and distress. *Journal of Health and Social Behavior, 50*(1), 49-64.

Rozanski, A., Blumenthal, J. A., & Kaplan, J. (1999). Impact of psychological factors on the pathogenesis of cardiovascular disease and implications for therapy. *Circulation, 99*(16), 2192-2217.

Sabanayagam, C., & Shankar, A. (2010). Sleep duration and cardiovascular disease: Results from the National Health Interview Survey. *Sleep, 33*(8), 1037-1042.

Sampson, R. J., & Raudenbush, S. W. (2004). Seeing disorder: Neighborhood stigma and the social construction of 'broken windows'. *Social Psychology Quarterly, 67*(4), 319-342. doi: 10.1177/019027250406700401

Sampson, R. J., & Raudenbush, S. W. (2005). Neighborhood stigma and the perception

of disorder. *Focus, 24*(1), 7-11.

Sapolsky, R. M. (1994). Individual differences and the stress response. *Seminars in Neuroscience, 6*(4), 261-269. doi:10.1006/smns.1994.1033

Sarwar, N., Danesh, J., Eiriksdottir, G., Sigurdsson, G., Wareham, N., Bingham, S., … Gudnason, V. (2007). Triglycerides and the risk of coronary heart disease: 10,158 incident cases among 262,525 participants in 29 Western prospective studies. *Circulation, 115*(4), 450-458. doi:10.1161/CIRCULATIONAHA.106.637793

Schmitt, N. (1996). Uses and abuses of coefficient alpha. *Psychological Assessment, 8*(4), 350-353. doi:10.1037/1040-3590.8.4.350

Schneiderman, N., McCabe, P., & Baum, A. (1992). *Stress and disease processes*. Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Schootman, M., Andresen, E., Wolinsky, F., Malmstrom, T., Morley, J., & Miller, D. (2010). Adverse housing and neighborhood conditions and inflammatory markers among middle-aged African Americans. *Journal of Urban Health, 87*(2), 199-210. doi:10.1007/s11524-009-9426-8

Schwartz, A. R., Gerin, W., Davidson, K. W., Pickering, T. G., Brosschot, J. F., Thayer, J. F., … Linden, W. (2003). Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosomatic Medicine, 65*(1), 22-35.

Schwartz, S. W., Cornoni-Huntley, J., Cole, S. R., Hays, J. C., Blazer, D. G., & Schocken, D. D. (1998). Are sleep complaints an independent risk factor for myocardial infarction? *Annals of Epidemiology, 8*(6), 384-392.

Sesso, H. D., Stampfer, M. J., Rosner, B., Hennekens, C. H., Gaziano, J. M., Manson, J. E., & Glynn, R. J. (2000). Systolic and diastolic blood pressure, pulse pressure, and mean arterial pressure as predictors of cardiovascular disease risk in Men. *Hypertension, 36*(5), 801-807.

Sharp, D. S., Andrew, M. E., Burchfiel, C. M., Violanti, J. M., & Wactawski-Wende, J. (2012). Body mass index versus dual energy x-ray absorptiometry-derived indexes: Predictors of cardiovascular and diabetic disease risk factors. *American Journal of Human Biology*. Advance online publication. doi: 10.1002/ajhb.22221

Skogan, W. G. (1990). *Disorder and decline*. Berkeley, CA: University of California Press.

Sobel, M. E. (1982). Asymptotic confidence intervals for indirect effects in structural equation models. *Sociological Methodology, 13*, 290-312.

Sobel, M. E. (1986). Some new results in indirect effects and their standard errors in covariance structure models. In N. Tuma (Ed.), *Sociological methodology* (pp. 159-186). Washington, DC: American Sociological Association.

Spielberger, C. D., Sydeman, S. J., Owen, A. E., & Marsh, B. J. (1999). Measuring anxiety and anger with the State-Trait Anxiety Inventory (STAI) and the State-Trait Anger Expression Inventory (STAXI). In M. E. Maruish (Ed.), *The use of psychological testing for treatment planning and outcomes assessment* (pp. 993-1021). Mahwah, NJ: Erlbaum.

Steiger, J. H. (1990). Structural model evaluation and modification: an interval estimation approach. *Multivariate Behavioral Research, 25*(2), 173 - 180.

Steptoe, A., & Feldman, P. J. (2001). Neighborhood problems as sources of chronic stress: Development of a measure of neighborhood problems, and associations with socioeconomic status and health. *Annals of Behavioral Medicine, 23*(3), 177-185.

Stjarne, M. K., Fritzell, J., De Leon, A. P., & Hallqvist, J. (2006). Neighborhood socioeconomic context, individual income and myocardial infarction. *Epidemiology, 17*(1), 14-23.

Stokols, D. (1996). Translating social ecological theory into guidelines for community health promotion. *American Journal of Health Promotion: AJHP, 10*(4), 282-298.

Stronegger, W. J., Titze, S., & Oja, P. (2010). Perceived characteristics of the neighborhood and its association with physical activity behavior and self-rated health. *Health Place, 16*(4), 736-743. doi: 10.1016/j.healthplace.2010.03.005

Sundquist, K., Theobald, H., Yang, M., Li, X., Johansson, S. E., & Sundquist, J. (2006). Neighborhood violent crime and unemployment increase the risk of coronary heart disease: A multilevel study in an urban setting. *Social Science and Medicine, 62*(8), 2061-2071. doi: 10.1016/j.socscimed.2005.08.051

Torpy, J. M., Lynm, C., & Glass, R. M. (2007). JAMA patient page. Chronic stress and the heart. *JAMA, 298*(14), 1722. doi:10.1001/jama.298.14.1722

U.S. Census Bureau. (n.d.). *American fact finder*. Retrieved from http://factfinder.census.gov

U.S. General Services Administration. (n.d.). *Project overview*. Retrieved from http://www.gsa.gov/sanysidrolpoe

Van der Kooy, K., van Hout, H., Marwijk, H., Marten, H., Stehouwer, C., & Beekman, A. (2007). Depression and the risk for cardiovascular diseases: Systematic review

and meta analysis. *International Journal of Geriatric Psychiatry, 22*(7), 613-626.

Van Horn, L. (1997). Fiber, lipids, and coronary heart disease. A statement for healthcare professionals from the Nutrition Committee, American Heart Association. *Circulation, 95*(12), 2701-2704.

Van Melle, J. P., de Jonge, P., Spijkerman, T. A., Tijssen, J. G., Ormel, J., van Veldhuisen, D. J., … van den Berg, M. P. (2004). Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: a meta-analysis. *Psychosomatic Medicine, 66*(6), 814-822. doi:10.1097/01.psy.0000146294.82810.9c

Van Trijp, M. J., Grobbee, D. E., Peeters, P. H., van Der Schouw, Y. T., & Bots, M. L. (2005). Average blood pressure and cardiovascular disease-related mortality in middle-aged women. *American Journal of Hypertension, 18*(2 Pt 1), 197-201. doi:10.1016/j.amjhyper.2004.09.005

Vaz Serra, A. (2002). Stress and cardiovascular diseases. *Revista Pportuguesa de Cardiologia, 21*(10), 1173-1187.

Vitaliano, P. P., Scanlan, J. M., Zhang, J., Savage, M. V., Hirsch, I. B., & Siegler, I. C. (2002). A path model of chronic stress, the metabolic syndrome, and coronary heart disease. *Psychosomatic Medicine, 64*(3), 418-435.

Voruganti, V. S., Lopez-Alvarenga, J. C., Nath, S. D., Rainwater, D. L., Bauer, R., Cole, S. A., … Comuzzie, A. G. (2008). Genetics of variation in HOMA-IR and cardiovascular risk factors in Mexican-Americans. *Journal of Molecular Medicine (Berlin), 86*(3), 303-311. doi:10.1007/s00109-007-0273-3

Wallace, T. M., Levy, J. C., & Matthews, D. R. (2004). Use and abuse of HOMA modeling. *Diabetes Care, 27*(6), 1487-1495.

Wang, J. G., Staessen, J. A., Franklin, S. S., Fagard, R., & Gueyffier, F. (2005). Systolic and diastolic blood pressure lowering as determinants of cardiovascular outcome. *Hypertension, 45*(5), 907-913. doi:10.1161/01.HYP.0000165020.14745.79

Warnick, G. R. (2000). Measurement of cholesterol and other lipoprotein constituents in the clinical laboratory. *Clinical Chemistry and Laboratory Medicine, 38*(4), 287-300.

Warren, T. Y., Barry, V., Hooker, S. P., Sui, X., Church, T. S., & Blair, S. N. (2010). Sedentary behaviors increase risk of cardiovascular disease mortality in men. *Medicine and Science in Sports and Exercise, 42*(5), 879-885. doi:10.1249/MSS.0b013e3181c3aa7e

Warrington, S. J., Padgham, C., & Lader, M. (1989). The cardiovascular effects of

antidepressants. *Psychological Medicine. Monograph Supplement, 16*, i-iii, 1-40.

Wen, M., & Maloney, T. N. (2011). Latino residential isolation and the risk of obesity in Utah: The role of neighborhood socioeconomic, built-environmental, and subcultural context. *Journal of Immigrant and Minority Health, 13*(6), 1134-1141. doi:10.1007/s10903-011-9439-8

Whitlock, G., Lewington, S., & Mhurchu, C. N. (2002). Coronary heart disease and body mass index: A systematic review of the evidence from larger prospective cohort studies. *Seminars in Vascular Medicine, 2*(4), 369-381. doi:10.1055/s-2002-36766

Winkleby, M., Sundquist, K., & Cubbin, C. (2007). Inequities in CHD incidence and case fatality by neighborhood deprivation. *American Journal of Preventive Medicine, 32*(2), 97-106. doi:10.1016/j.amepre.2006.10.002

Wolk, R., Gami, A. S., Garcia-Touchard, A., & Somers, V. K. (2005). Sleep and cardiovascular disease. *Current Problems in Cardiology, 30*(12), 625-662. doi:10.1016/j.cpcardiol.2005.07.002 [doi]

Yuan, K. H., & Bentler, P. M. (2000). Three likelihood-based methods for mean and covariance structure analysis with nonnormal missing data. *Sociological Methodology, 30*(1), 165-200. doi: 10.1111/0081-1750.00078