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Psychosocial Mediators of Ethnic Disparities in Allostatic Load

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

in

Clinical Psychology

by

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University of California, San Diego
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2013
DEDICATION

For my parents, Patricia Driediger and Leigh Tomfohr. Without your constant love, support and encouragement this dissertation would not be possible.

To my friends and colleagues, Kate Edwards, Erin Green, Rujvi Kamat, Mitch LeBlanc, Khaleel Meghji, Kimberly Payne, Meredith Pung, Amanda Schweizer, Robert Taylor, and Sarah Yager. You have each helped to make this process a pleasure. I am eternally grateful for your friendship and support.
Life is largely a process of adaptation to the circumstances in which we exist. (and) the secret of health and happiness lies in successful adjustment to the ever-changing conditions on this globe; the penalties of failure in this great process of adaptation are disease and unhappiness.

_Hans Selye_
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BOOK CHAPTER


ACADEMIC PRESENTATIONS


ABSTRACTS


Tomfohr, L.M., Ancoli-Israel, S., Dimsdale, J.E. (March 2009). Childhood socioeconomic status is associated with adult sleep structure. American Psychosomatic Society, Portland, OR.


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Wald, J., & Tomfohr, L.M. (June 2009) The efficacy of combining interoceptive exposure and trauma-related exposure therapy for sexual assault-related PTSD. Canadian Psychological Association. Montréal, QU.


ABSTRACT OF THE DISSERTATION

Psychosocial Mediators of Ethnic Disparities in Allostatic Load

by

Lianne M. Tomfohr

Doctor of Philosophy in Clinical Psychology

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

Professor Joel Dimsdale, Chair

Allostatic Load (AL) is a cumulative index of physiological dysregulation. African Americans (AA) have traditionally had higher AL than their Caucasian American (CA) counterparts. Differences in psychosocial factors and negative health practices that disproportionately occur in certain groups may account for ethnic differences in AL. The main aim of this dissertation was to investigate if differences in psychosocial stressors, non-stress psychological variables and health practices mediated ethnic differences in AL (Aim 1). A secondary aim was to investigate methodological issues in the concept of AL. An AL composite score was calculated using a z-score method as well as the traditional scoring algorithm. Analyses from Aim 1 were conducted using each of these composite...
scores as an outcome to investigate if findings changed depending on the scoring method used (Aim 2). Using the sub-factors of AL as outcome variables, the tertiary aim was to investigate if psychological and health behavior mediators of ethnic disparities in the AL composite score also emerged as mediators of the individual sub-factor variables (Aim 3).

Data were collected as part of a larger study investigating ethnic differences in cardiovascular risk factors. Participants included working AA (N = 75) and CA (N = 100), adult men and women. Participants completed questionnaires assessing demographics, psychosocial stressors, non-stress psychological variables, and health practices. Biological data were collected as part of an overnight hospital stay.

The covariates age, gender and socioeconomic status (SES) were held constant in each analysis. Findings showed significant ethnic differences in AL, such that AA had higher AL than their CA counterparts (p < 0.01). Non-stress psychological variables and health practices were partial mediators of the relationship between ethnicity and AL (p’s < 0.05). The experience of anger subscale from the Buss-Durkee Hostility Scale and the Pittsburgh Sleep Quality Index (PSQI) total score were significant individual mediators of the ethnicity-AL relationship (p < 0.05). Results were substantively the same regardless of the scoring algorithm used to create the AL composite score. Analyses using the sub-factors of AL were largely null. These results suggest that non-stress psychological factors and health practices play an important role in explaining ethnic differences in AL.
INTRODUCTION

African American Health Disparities

African Americans (AA) suffer disproportionate morbidity and mortality from a range of illnesses that affect multiple physiological systems. Compared to Caucasian Americans (CA), AA are at higher risk for developing hypertension and experiencing major cardiovascular events, such as myocardial infarction, stroke, heart failure and cardiac death (Lloyd-Jones et al., 2009; Lloyd-Jones et al., 2010; National Center for Health Statistics, 2011). AA also have higher prevalence and mortality rates associated with diabetes, asthma, cancer and numerous other illnesses (Bryant-Stephens, 2009; Herman et al., 2007; Jemal, Siegel, Xu, & Ward, 2010; National Center for Health Statistics, 2011; Smith, Hatcher-Ross, Wertheimer, & Kahn, 2005). These health disparities translate into significant differences in mortality, such that CA men live approximately 6 years longer than their AA peers and CA women live approximately 4 years longer than AA women. To date, AA men have the lowest life expectancy and highest mortality rate when compared to other ethnic and gender groups in the United States (Heron et al., 2009; National Center for Health Statistics, 2011).

The prominent health disparities observed in AAs emerge by middle age, exist across socioeconomic levels and are not fully explained by differences in obesity or health practices (Geronimus, Bound, Waidmann, Hillemeier, & Burns, 1996; Geronimus, Bound, Waidmann, Colen, & Steffick, 2001; Williams, 1999; Wong, Shapiro, Boscardin, & Ettner, 2002). What factors predispose AAs to disproportionately suffer from chronic
illness and die earlier in life? Increasingly, repeated exposure to psychosocial and/or economic stressors that affect certain ethnic groups (e.g., discrimination) are being recognized as contributing to the development of health disparities (Dressler, Oths, & Gravlee, 2005). An emerging theory hypothesizes that chronic psychosocial stressors impact health by disrupting homeostatic regulation across multiple physiological systems (Betancourt, 2006; Geronimus et al., 2001; Geronimus, Hicken, Keene, & Bound, 2006; Geronimus, 1992).

**Allostatic Load**

The concept of Allostatic Load (AL) was developed as a way to measure cumulative ‘wear and tear’ across multiple physiological systems resulting from repeated exposure and adaptation to stress (McEwen, 1998; McEwen & Seeman, 1999). Allostasis refers to physiological adaptations that occur across systems as the body works to return to a state of homeostasis after exposure to external stressors (e.g., the fight-or-flight response). Short-term, adaptation to stressors is not theorized to be physiologically harmful. However, repeated exposure to stress is posited to dysregulate multiple interdependent systems, in-turn decreasing their ability to return the body to a baseline state of functioning, chronic dysregulation is then theorized to result in a new and higher baseline (e.g., higher blood pressure (BP)). Assessing AL allows for the quantification of physiological dysregulation throughout the body and has been suggested as a way to measure the impact of chronic exposure to stress on early stages of disease processes (Geronimus et al., 2006; Mays, Cochran, & Barnes, 2007).
The AL score is composed of markers taken from a variety of regulatory systems, some of which are assumed to convey changes in other variables and others that are thought to convey independent risk. Theoretically, AL has been conceptualized as the physiological burden imposed by stress as indicated by categories representing primary mediators (e.g., substances the body releases in response to stress such as norepinephrine, epinephrine, and cortisol) and secondary mediators, effects that result from actions of primary mediators (e.g., elevated BP and changes in glucose metabolism) (McEwen, 1998; McEwen & Seeman, 1999). There is evidence suggesting that chronic inflammation is associated with exacerbation of the disease process and measures of inflammation have been increasingly incorporated into the conceptualization of AL (Gallo, Jiménez, Shivpuri, Espinosa de los Monteros, K., & Mills, 2011; Seeman et al., 2010). A recent factor analysis confirmed an AL factor, comprised of physiological dysregulation across six biological systems including heart rate variability, BP, metabolism, sympathetic nervous system (SNS) activity, hypothalamic pituitary adrenal (HPA) activity and inflammation (Seeman et al., 2010). The AL model posits that assessing AL can push forward biomedical advances aimed at early detection of those at a high risk of developing negative tertiary outcomes (Goldstein & McEwen, 2002; Juster, McEwen, & Lupien, 2010; McEwen & Seeman, 1999).

**Conceptual Issues in the Definition of Allostatic Load**

**Biomarker Inclusion.** Specific biomarkers included in the definition of AL have varied across studies and there has been debate concerning variable inclusion and combination approaches (Gersten, 2008; Loucks, Juster, & Pruessner, 2008; McDade,
As part of this dissertation, a review was conducted to investigate which variables have most often been included in the definition of AL, within the stress and AL literature. Fourteen investigations of the relationship between stress and AL were identified, revealing 41 variables that had been included within the definition. Figure 1 portrays the frequency with which markers were represented. (Bellingrath, Weigl, & Kudielka, 2009; Clark, Bond, & Hecker, 2007; Gallo et al., 2011; Glei, Goldman, Chuang, & Weinstein, 2007; Glover, 2006; Hawkley, Lavelle, Berntson, & Cacioppo, 2011; Juster et al., 2011; Juster, Marin et al., 2011; Kinnunen, Kaprio, & Pulkkinen, 2005; Kubzansky, Kawachi, & Sparrow, 1999; Li et al., 2007; Mair, Cutchin, & Kristen Peek, 2011; Roepke et al., 2011; Schnorpfeil et al., 2003). Many of these variables fell within the traditional domains of blood pressure, metabolism and sympathetic nervous system activity, HPA and inflammation, but other measures such as assessment of immune system activity, and coagulation were also included in the definition of AL.

**Scoring Method.** The optimal method for creating the AL cumulative score has been an area of debate. Traditionally, AL has been based on the algorithm used in the MacArthur Study of Healthy Aging, in which each biological parameter is classified into quartiles based on the distribution of scores in the cohort (Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). After quartiles have been created, an AL score is calculated by aggregating the number of parameters for which the subject fell into the highest-risk quartile (the top quartile for all parameters except for HDL cholesterol, for which membership in the lowest quartile corresponds to highest risk). This scoring method has been questioned, as the definition of “at risk” varies depending on the health of the population under study (Gersten, 2008; McDade, 2008).
Another popular scoring method creates AL scores by averaging z-scores across biological systems and summing them. This method allows for greater variation in the data and has been shown to yield comparable findings with respect to health risk (Seeman et al., 1997). Additionally, it has been suggested that averaging over continuous z-scores may more accurately reflect the continuous level of risk captured by AL scores (Hawkley et al., 2011; Mair et al., 2011). Despite the debate, investigation into different measurement and combination approaches has shown only moderate variance in predictive utility (Karlamangla, Singer, McEwen, Rowe, & Seeman, 2002; Seeman et al., 2010; Seplaki, Goldman, Glei, & Weinstein, 2005). For a full review of variables that have been included in the definition of AL and combination approaches, see Juster et al. (2010).

Sub-Factors of AL. AL is defined as a measure of physiologic dysregulation that occurs across multiple systems (Seeman et al., 1997). The physiological risks associated with AL are theorized to occur because of cumulative dysregulation of multiple systems, rather than extreme dysregulation in one system and some evidence suggests that the composite AL score is more predictive of physical and cognitive outcomes than subcomponents (Karlamangla et al., 2002).

According to the AL model, exposure to psychosocial stressors alters stress hormone functioning, leading to ‘wear and tear’ on larger physiological systems, eventually resulting in higher baseline-points (e.g., increases in resting BP) and negative end-points (e.g., morbidity and mortality from chronic illness) (Juster et al., 2011). An issue that has been raised in the AL literature is the lack of strong evidence identifying consistent associations between psychosocial stressors and the theorized primary
mediators in the AL model (e.g., catecholamines and cortisol). Some studies have found associations between stressors and SES and traditional risk factor pathways, (e.g., cardiovascular function), while failing to detect associations with stress hormones (Dowd & Goldman, 2006; Dowd, Simanek, & Aiello, 2009; Hawkley et al., 2011; Seeman et al., 2004). The issue then arises as to the extent that associations between psychosocial factors and AL are a manifestation of cardiovascular and metabolic risk pathways, associations that have been widely demonstrated in prior research (Dowd et al., 2009). If stressors and other psychosocial factors are not consistently related to theoretical primary pathways, then the theory underlying AL becomes called into question.

Multiple methodological and theoretical issues have been raised in the AL theory. Lack of clearly defined variable inclusion and scoring methods call into question the reliability of construct. Lack of clear evidence underlying theoretical pathways calls into question the validity of the model. Despite these limitations, the concept of AL is being used with increasing frequency, as a way to capture risk across multiple physiological systems. It is appealing to look at the concept of ‘health’ in a comprehensive fashion; however, it is clear that increasing investigation into the reliability and validity of the model are necessary.

**Allostatic Load, Chronic Disease and All-Cause Mortality**

Results from the MacArthur Successful Aging cohort have shown that higher baseline AL scores predicted an increased risk of cardiovascular disease events at a 2.5-year follow-up and all-cause mortality and incident cardiovascular disease events at a 7-year follow-up (Seeman et al., 1997; Seeman, McEwen, Rowe, & Singer, 2001).
Similarly, Goldman et al. (2006) found that in data collected as part of the Taiwanese, Social Environment and Biomarkers of Aging Study (SEBAS), increased AL predicted increased all-cause mortality at a 3-year follow-up (Goldman et al., 2006; Goldman, Turra, Glei, Lin, & Weinstein, 2006). Finally, using data from the National Health and Nutrition Examination Survey (NHANES; III) linked to the National Death Index, Borrell and colleagues (2010) found that increasing AL was associated with increasing all-cause mortality after controlling for age, gender, race/ethnicity, education and income. Additionally, higher AL scores were associated with increased all-cause mortality regardless of race/ethnicity (Borrell, Dallo, & Nguyen, 2010). In each of these studies, increasing AL was associated with worse outcomes over time. This trend persisted across socioeconomic and racial/ethnic groups. Interestingly, data from the MacArthur Successful Aging cohort also showed that changes in AL over a 2.5-year period were associated with all-cause mortality 4.5 years later, such that decreasing scores over time were associated with better outcomes (Karlamangla, Singer, & Seeman, 2006).

**Ethnic Differences in Allostatic Load**

Using data from the NHANES (IV), Geronimus and colleagues (2006) reported that on average, AAs had higher AL scores than their CA counterparts and that differences persisted after controlling for socioeconomic status (SES) (Geronimus et al., 2006). Similarly, in the Coronary Artery Risk Development in Young Adults (CARDIA) study, Seeman and colleagues (2010) found that AAs had significantly higher AL than CAs and that differences were not fully explained after covarying for indices of poverty (Seeman et al., 2010).
If differences in SES do not account for ethnic disparities in AL, the next natural question is what is driving the association? As exposure to chronic stress is theorized to increase AL and AA’s experience more chronic stressors than their CA counterparts, it has been theorized that differential exposure to stressors may mediate ethnic differences in AL (Geronimus et al., 2006; Mays et al., 2007). Other candidate mediators include differences in personality variables and health behaviours. What follows is a review and summary of the evidence that has shown associations between chronic stress, psychological and personality variables, health practices and AL.

**Chronic Stress and Allostatic Load**

According to the model of AL, chronic stressors should demonstrate a clear and marked relationship with AL (Pearlin, Schieman, Fazio, & Meersman, 2005). However, many studies have inferred stress based on socioeconomic status or social support (Gallo et al., 2011). Lower SES has been repeatedly associated with higher AL (see Dowd et al., 2009 for full review) and studies have shown that lower levels of social support are also associated with higher AL (Dowd et al., 2009; Seeman, Singer, Ryff, Dienberg Love, & Levy-Storms, 2002; Seeman, Glei et al., 2004). More recently, studies have emerged showing associations between other indices of stress and AL; however the literature has been unclear as to which types of stress (e.g., chronic or acute) influence AL and whether the association varies according to perceptions of stress (e.g., objectively or subjectively assessed).

**Perceived Stress and Major Life Events:** Perceptions of stress may have stronger relationships with AL than traditional, objective markers of major stressful
events (Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). Data from the SEBAS cohort found that perceived stress was concurrently associated with AL and was predictive of increases in AL over a 4-year period (Goldman, Glei, Seplaki, Liu, & Weinstein, 2005). Exposure to serious life stressors in adults also predicted increased AL over time; however there was some evidence that the effect was mediated by perceived stress (Glei et al., 2007). In an additional analysis of stressful life events in the SEBAS study, no relationship was detected between major life stressors and a neuroendocrine measure of AL; however, perceived stress was associated with increased neuroendocrine AL in women (Gersten, 2008). In a sample of mothers of pediatric cancer survivors and controls, a measure of stressful life events was not associated with AL, whereas symptoms of PTSD, a chronic stress disorder, were (Glover, 2006). Similarly, in a longitudinal study of caregivers, perceived stress was predictive of increases in the primary mediators of AL; in contrast, more objective environmental stressors were only weakly associated (Clark et al., 2007). These findings suggest that perceptions of stress may be more directly associated with AL than discrete stressful events.

In a group of healthy workers, chronic stress across the domains of work and social functioning were found to be associated with AL (Juster et al., 2011). Similarly, in a study of Mexican American women, self-reported chronic stress in some domains (work, caregiving, financial and relationship) was associated with higher AL load scores even after controlling for health behaviours, whereas chronic stress in other domains such as health of self or family, or housing stressors were unrelated (Gallo et al., 2011). Again, negative life events and daily hassles were associated with AL in a sample of participants from the Texas City cohort. In this cohort, the findings were stronger in women
compared to men; however, the relationship between life events, daily hassles and AL was attenuated after controlling for smoking and exercise behaviour (Mair et al., 2011).

**Work Stress:** High levels of work stress have been repeatedly associated with higher AL in a variety of populations. In a study of female schoolteachers, women high on effort-reward imbalance had higher AL (Bellingrath et al., 2009). Similarly, two studies of industrial employees in China, found that lower job control and higher job demands were associated with higher levels of AL (Li et al., 2007; Sun, Wang, Zhang, & Li, 2007). Finish workers with more career instability between 27-36 years of age had a threefold increase in AL at age 41 (Kinnunen et al., 2005) and higher job demands have been associated with higher AL in a sample of industrial workers in Germany (Schnorpfeil et al., 2003).

Taken together, these studies suggest that chronic stress across a variety of domains is associated with AL; however, it remains unclear the extent to which specific stressors (acute versus chronic) and perceptions of stress contribute to the overall AL score. Given the myriad of different stressors that have been associated with AL, it has been recommended that studies examining stress and AL assess stress in multiple domains including: subjective ratings of the stressors, assessment of daily hassles, major life events in the past year, trauma, work stress and discrimination (Gersten, 2008).

**Non-Stress Psychological Variables and Allostatic Load**

In addition to stressors, there is some evidence that other psychological variables and personality factors are associated with AL.

**Depression.** Data from the SEBAS cohort has shown that higher depressive
symptoms are concurrently associated with higher AL (Goldman et al., 2005; Seplaki et al., 2005; Seplaki, Goldman, Weinstein, & Lin, 2006). Cross-sectional associations between AL and depression have been detected using data from the Douglas Hospital Longitudinal Study of Normal and Pathological Aging; however depression was not longitudinally predictive of AL increases over time in this sample (Juster et al., 2010).

**Hostility.** Using data from the Normative Aging Study, SES, as assessed through level of education, was inversely associated with AL; however the relationship was significantly attenuated with the inclusion of hostility (Kubzansky et al., 1999). Similarly, Hawkley et al. (2011) examined multiple potential mediators of the SES and AL relationship using data from the Chicago Health, Aging, and Social Relations Study. Of the personality variables examined, only hostility emerged as a significant mediator of SES differences in AL (Hawkley et al., 2011). Finally, Sun et al. (2007) found that hostility was associated with AL in a sample of older male Chinese industrial workers (Sun et al., 2007).

**Interpersonal Support.** Combined data from the MacArthur Study of Healthy Aging and the Wisconsin Longitudinal Study (WLS), showed that self-reported positive parental bonding and more positive current relationships were related to lower AL in men (Seeman et al, 2002). Similarly, results from the SEBAS study found that the presence of a spouse in men and close social ties with friends and neighbours in men and women was associated with lower AL among elderly men and women (Seeman et al., 2004).

**Health Practices and Allostatic Load**

Chronic stress is associated with worse self-care such as, decreases in exercise
and increases in negative health behaviours such as smoking and alcohol consumption. There has been some question about whether AL is affected primarily by psychological stress, or if other factors associated with stress such as a sedentary life style, smoking or alcohol consumption drive the relationship (Loucks et al., 2008). Smoking, alcohol use and inactivity have each been directly associated with AL (Crimmins, Kim, & Seeman, 2009; Hampson, Goldberg, Vogt, Hillier, & Dubanoski, 2009; Hu, Wagle, & Goldman, 2007). However, associations between SES and AL have persisted after controlling for health behaviours (Gustafsson, Janlert, Theorell, Westerlund, & Hammarström, 2011) and one study found that chronic stress was associated with AL beyond the influence of smoking, alcohol consumption, dietary and exercise behaviours (Gallo et al., 2011).

However, in another study, controlling for health practices attenuated the relationship between stress and AL (Mair et al., 2011). Sleep is another health practice that has been posited to impact AL (McEwen, 2006; Van Cauter & Spiegel, 1999); however, to date only one investigation has shown an association between sleep quality and AL, such that worse self-reported sleep quality was associated with higher AL (Hawkley et al., 2011).

**Limitations and Future Directions**

AA have higher levels of AL than their Caucasian counterparts and ethnic differences in AL appear to persist after controlling for indices of SES. Additionally, AA are exposed to numerous psychosocial stressors, often at levels beyond those experienced by CA (Williams, Yu, Jackson, & Anderson, 1997; Williams, Neighbors, & Jackson, 2008; Williams & Mohammed, 2009). Given the theoretical associations between stressors and AL and ethnic differences in exposure to psychosocial stressors, it follows
logically that differential exposure to psychosocial stressors may mediate ethnic differences in AL; however, to date there is no literature investigating this question.

Additionally, exposure to psychosocial stressors has been associated with increased prevalence of distress including depression, aggression and hostility (Banks, Kohn-Wood, & Spencer, 2006; Bynum, Burton, & Best, 2007; Davis & Stevenson, 2006; DuBois, Burk–Braxton, Swenson, Tevendale, & Hardesty, 2002), negative health practices (e.g. tobacco and alcohol use) (Bennett, Wolin, Robinson, Fowler, & Edwards, 2005; Gibbons et al., 2010; Landrine, Klonoff, Corral, Fernandez, & Roesch, 2006; Martin, Tuch, & Roman, 2003) and disrupted sleep quality (Kim & Dimsdale, 2007), each of which have been associated with increases in AL. The impact of these potential candidate mediators has also not been fully explored.

**Purpose of the Current Study**

The current study is an archival analysis from a larger project examining how ethnicity and adversity impact key mechanisms linked to cardiovascular pathophysiological regulation. A main hypothesis of the parent grant was, “After controlling for adversity, the ethnic effects on pathophysiology will no longer be significant”. The current project tested this hypothesis using the outcome of AL, a cumulative measure of ‘wear and tear’ on multiple physiological systems. The main purpose of the current study was to examine whether differential levels of stress variables, non-stress psychological variables and health practices mediated the relationship between ethnic group membership and AL (Aim 1). Given the well-documented associations between SES and AL and the ethnic differences in SES in the
sample, a measure of SES was held constant in all analyses to examine the independent contribution of the variables under study to AL. In line with previous recommendations, stress was assessed across multiple domains including: perceived stress, daily hassles, job stress and discrimination. Non-stress psychological variables (depression, hostility, and interpersonal support) and health practices (smoking, alcohol use, habitual exercise and subjective sleep quality) with theoretical associations to AL were also examined as potential mediators.

First, the question of whether ethnic differences in AL persisted after controlling for sample differences in age, gender and SES was examined. Second, the extent to which stress variables, non-stress psychological variables and health behaviours were associated with both ethnicity and AL was tested. Finally, meditational analyses were conducted to test the hypothesis that ethnic differences in stress variables, non-stress psychological variables and health practices mediated the relationship between ethnicity and AL (Aim 1). Consistent with previous investigations, it was hypothesized that AA would have higher AL than CA and that the association between ethnicity and AL would persist after controlling for differences in demographic and socioeconomic variables. It was also hypothesized that AA would experience greater exposure to stressors, have differential levels of self-reported non-stress psychological variables and would be more likely to engage in negative health practices than CA and that differences would mediate ethnic differences in AL (Aim 1).

The second and third aims of the dissertation explored conceptual issues associated with AL. Traditionally, AL scores have been calculated using an algorithm from the MacArthur Study of Healthy Aging. In this method, each biological parameter is
classified into quartiles based on the distribution of scores on the cohort (Seeman et al., 1997). An AL score is then calculated by aggregating the number of parameters for which the subject fell into the highest-risk quartile (the top quartile for all parameters except for HDL cholesterol). The second aim of the study was to examine if AL scores created from the traditional quartile scoring method or z-score method were associated in similar ways with predictor and mediator variables (Aim 2). Given that previous studies have found little predictive difference in AL calculated using different scoring methods, it was expected that AL scores calculated using both z-score and traditional quartile scoring methods would be associated in the same degree and magnitude to psychosocial mediators of ethnic differences in AL (Aim 2).

Finally, the AL score was deconstructed into its component parts in order to investigate associations between ethnicity, mediator variables and individual sub-factors of AL (Aim 3). The major theoretical AL pathways were explored, including subsets of biomarkers reflecting cardiovascular function, metabolism, SNS activity, HPA axis activity and inflammation. The extent to which sub-factors of AL were associated with mediators of the ethnicity-AL relationship in a similar fashion as the composite measure of AL was explored. Findings linking psychological variables to sub-factors of AL have been mixed, with some studies showing associations between psychological variables and sub-factors of AL and other studies failing to find an association. Given inconsistencies in the literature, no a priori hypotheses were made for this aim (Aim 3).
Methods

Participants

Participants were recruited from the San Diego, California area between 2005 and 2010. Recruitment occurred through referrals and advertisements. Exclusion criteria included diagnosis of a major clinical illness as determined by a physical evaluation, previous diagnosis of a sleep disorder, blood pressure $\geq 170/105$ mm Hg, history of psychosis, and BMI $\geq 40$ kg/m$^2$. Additionally, women taking hormonal contraceptives or hormone replacement therapy, women who were pregnant, or women with self-reported premenstrual syndrome were excluded from the study. All participants in the study reported working $\geq 20$ hours per week; this criterion was implemented because we were interested in investigating relationships between job stressors and cardiovascular functioning, requiring that participants be employed.

The final sample consisted of 75 AA and 100 CA men and women. While 12% of the participants were diagnosed with hypertension, participants were otherwise free from chronic illness. Hypertensive medications can exert prominent effects on many variables included in the definition of AL (e.g., catecholamines, BP) and so patients who came into the study taking antihypertensive medications ($N = 3$) had their medications tapered slowly, maintaining BP $> 140/90$ but $< 170/105$ mm Hg for 3 weeks, under the supervision of a study physician. All patients tolerated tapering well and resumed antihypertensive medication use at the end of the study. The project was approved by the Institutional Review Board of the University of California, San Diego and all participants provided written informed consent before entering the study.
Procedures

After prescreening procedures confirmed eligibility, participants were invited to come to the Gillin Laboratory for Sleep and Chronobiology at the University of California, San Diego General Clinical Research Center. Questionnaires were given to participants upon their arrival in the hospital and were collected prior to discharge. Participants spent the evening in the hospital and a morning blood sample was obtained after wakening the subject at 6:30am the next morning. Blood samples for cholesterol and inflammatory biomarkers were drawn after an overnight fast. Blood samples were obtained through an intravenous catheter, which was inserted after hospital admission at around 4:30pm. The blood sample was taken from subjects while they were recumbent in bed to avoid the effects of movements or physical activities on immune system biomarkers or catecholamines. Blood samples were put on ice immediately after collection and were centrifuged. Plasma was stored at -80 degrees Celsius until assay. Urine samples were collected by the participants during the overnight hospital stay and were subsequently refrigerated. All assays, with the exception of cortisol, which is robust to free-thaw, were analyzed using samples that had not been previously freeze-thawed. Inflammatory biomarkers and neuroendocrine assays were conducted at the University of California San Diego (UCSD) General Clinical Research Center Core Laboratory.

Measures

Demographics. Ethnic group membership, gender and age were collected through self-report.


**Socioeconomic Status.** Socioeconomic status (SES) was assessed with the clinician-rated, Hollingshead 2-Factor Index of Social Position. The scale assesses participants’ highest level of formal education and current occupation. The two factors are summed, weighted and combined into a continuous measure of social index. Scores range from 11 to 77, lower scores are indicative of higher SES (Hollingshead, 1957).

**Stress Variables.** Stress was assessed across a variety of domains including perceived stress, daily hassles, perceived discrimination and job stress. The previously validated 10-item Perceived Stress Scale (PSS) was used to assess perceived stress in the past month. Respondents were asked to rate how frequently in the past month they have felt that their lives were unpredictable, uncontrollable or overloaded on a scale of 0 (never) to 4 (very often). Higher scores are indicative of more perceived stress (Cohen, Kamarck, & Mermelstein, 1983; Cohen, 1988). Cronbach’s alpha across the 10-items was .78.

**Daily Hassles.** Daily hassles were assessed using the Hassles portion of The Daily Hassles and Uplifts Scale. Hassles were assessed as a mean score of 53-items on a scale of 0-3, which asks subjects to respond as to how bothered they were by responsibilities across various domains such as relationships, work, finances and health. Higher values are associated with more distress from daily hassles (DeLongis, Coyne, Dakof, Folkman, & Lazarus, 1982; DeLongis, Folkman, & Lazarus, 1988). Cronbach’s alpha across the 53-items was .93.

**Perceived Discrimination.** Perceived discrimination was assessed using The Scale of Ethnic Experience, a 32-item, self-report questionnaire developed to measure the cognitive experience of ethnicity across multiple domains. The perceived discrimination
subscale assesses the extent to which an individual believes that members of his or her ethnic group have been discriminated against in society. Higher scores are indicative of greater endorsement of perceived discrimination (Malcarne, Chavira, Fernandez, & Liu, 2006). Cronbach’s alpha across the 8-item subscale was .89.

**Job stress.** Job stress was assessed using the self-report Job Content Questionnaire (JCQ). The JCQ is a widely validated measure of job stress, which assesses stressors across a variety of domains. Items range from 1 (strongly disagree) to 4 (strongly agree). In line with previous research showing associations between scales on the JCQ and AL, we investigated two subscales: (1) decision latitude (DL) or job control, assessed by summing the six item skill discretion (SD) subscale and the three item decision authority (DA) subscale ($\alpha = .85$) and (2) job demands, assessed using the five item psychological demands subscale ($\alpha = .53$) (R. Karasek et al., 1998; Li et al., 2007; Sun et al., 2007). Decisional control refers to an individual’s ability to control work activities and make important decisions. Job demands refers to task requirements or workload. Cronbach’s alpha across the 3-item decision authority subscale was .85 and across the 5-item job demands subscale was .63.

A measure of job strain was created using a modification of the Karasek Job Demand-Control model (Karasek, Baker, Marxer, Ahlbom, & Theorell, 1981). The model combines “job demands” and “decisional control”, to assess job strain. According to the model, high job demands and low decisional control are associated with job strain. Traditionally, job strain is calculated through a quadrant term approach, using a median split of job demands and decisional control as cutoffs; however, a strain by subtraction method, in which the decisional control score is subtracted from the job demands scores
has been shown to have more predictive utility and was utilized for these analyses (Courvoisier & Perneger, 2010).

**Non-Stress Psychological Variables**

**Hostility.** Hostility was assessed using the Buss-Durkee Hostility Scale. The scale is composed of 75 true/false items. A total hostility scale score is produced in addition to subscale scores that can be combined into Expression of Anger (indirect hostility, physical assaultiveness, verbal expression) and Experience of Anger (resentment, suspiciousness) subscales (Buss & Durkee, 1957). Cronbach’s alpha for 27-items composing the Expression of Anger subscale was .71. Cronbach’s alpha for the 17-items composing the Experience of Anger subscale was .79.

**Depression.** Depression was measured using the 20-item, Center for Epidemiological Studies – Depression Scale (CES-D). Respondents are asked to rate on a 4-point scale ranging from 1 (rarely/none of the time) to 4 (most/all of the time) the frequency of depressive symptoms in the past week. Responses are summed across items with higher responses indicative of higher depressive symptoms (Radloff, 1977). Cronbach’s alpha across the 20-item scale was .89.

**Social Support.** Social support was assessed with the 12-item, Interpersonal Support Evaluation List (ISEL). Respondents are asked to rate social support items on a 4-point scale ranging from 1 (definitely false) to 4 (definitely true). Responses are summed into three subscale scores, which assess tangible support, appraisal of support and belonging. The mean of the subscale scores was taken as a measure of social support (Cohen & Hoberman, 1983; Cohen, Mermelstein, Kamarck, & Hoberman, 1985). Higher
scores are indicative of more social support. Cronbach’s alpha across the 12-items was .87.

**Health Variables.** Smoking status, alcohol consumption, sleep quality and exercise were assessed. Participants were classified as smokers if they reported smoking cigarettes on a daily basis. Alcohol consumption was defined as the average number of alcoholic drinks consumed per week as reported by the participant. Data had a significant positive skew, was log transformed to estimate normality.

**Exercise.** Exercise was assessed using the Leisure Time Exercise Questionnaire (LTEQ). The LTEQ is a self-report questionnaire assessing regular physical activity. The questionnaire asks participants to rate how often in an average week they engage in mild, moderate or strenuous activity. A total score is calculated by multiplying the frequency of weekly exercise by differing intensities, strenuous exercise is multiplied by nine, moderate exercise by five, and mild exercise by three (Godin & Shephard, 1985). Higher scores reflect more regular physical activity. The total score has been shown to be a valid (Godin & Shephard, 1985; Godin, Jobin, & Bouillon, 1986; Jacobs, Ainsworth, Hartman, & Leon, 1993; Miller, Freedson, & Kline, 1994) and reliable (Godin & Shephard, 1985; Godin et al., 1986; Sallis, Buono, Roby, Micale, & Nelson, 1993) estimate of regular physical activity. Exercise data had a significant positive skew and was log transformed to approximate normality. Cronbach’s alpha across the 3-items was .77.

**Subjective Sleep Quality.** Sleep quality was assessed with the Pittsburgh Sleep Quality Index (PSQI). The PSQI is a 19-item self-report questionnaire that assesses habitual sleep quality across a seven domains. PSQI global scores range from 0-21,
higher scores are indicative of worse sleep quality (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Cronbach’s alpha across the 7-subscales was .70.

**Allostatic Load.** The AL score in this study was composed of 11 biological markers encompassing measures of cardiovascular function (SBP and DBP), metabolism (waist-hip ratio, fasting glucose, serum high-density lipoprotein cholesterol (HDL-c) and the ratio of HDL-c to total serum cholesterol), 8-hour SNS activity (urinary norepinephrine and epinephrine excretion levels), 8-hour HPA axis activity (urinary cortisol excretion) and markers of inflammation (C-Reactive Protein (CRP) and Interleukin-6 (IL-6)). The AL composite score was calculated by creating z-cores for each variable and averaging across all 11 variables (Aim 1).

An AL score was also calculated using the algorithm from the MacArthur Study of Healthy Aging. Each of the biological parameters included in the definition of AL were classified into quartiles based on the distribution of scores from the sample. The AL score was calculated by aggregating the number of parameters for which a subject fell into the highest-risk quartile (the top quartile for all parameters except for HDL cholesterol which the lowest quartile corresponds to highest risk) (Aim 2).

Finally, subsets of biomarkers reflecting cardiovascular function (SBP and DBP), metabolism (waist-hip ratio, fasting glucose, HDL-c and the ratio of HDL-c to total serum cholesterol), SNS activity (urinary norepinephrine and epinephrine excretion levels), HPA axis activity (urinary cortisol excretion) and inflammation [C-Reactive Protein (CRP) and Interleukin-6 (IL-6)] were created from z-scores (Aim 3). All measurements were assessed at rest.
**Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP).** BP was calculated as the average of the second through fourth of four seated blood pressure readings that were assessed after 5-minutes of seated rest (Dinamap 1846× monitor. Critikon; Tampa, FL).

**Waist-hip ratio (WHR).** WHR was calculated as the ratio of the mid-waist measurement, assessed as midway between the lowest rib and the iliac crest and hip measurement assessed as the greatest extension of the gluteal muscle as seen from the right side by tape measure.

**Total Cholesterol, HDL-c and Fasting Glucose.** Cholesterol and fasting glucose samples were processed by the General Clinical Research Center. Lipids and fasting glucose were determined using a Roche Cobas 6000 (Roche Diagnostics, Indianapolis, IN).

**Norepinephrine (NE), epinephrine (EPI), Cortisol and Creatinine.** Samples were obtained after overnight urine collection. NE and EPI were determined using a COMT-based radioenzymatic assay (Kennedy, Ziegler, 1990). Intra-assay and inter-assay coefficients of variation were <7% and <11%, respectively.

Urinary cortisol was assessed using by commercial ELISA (Quantikine, R&D Systems, Minneapolis, Minnesota), with intra- and inter-assay coefficients of variation were both <10%.

**Inflammatory Markers.** C-reactive protein (CRP) was assayed using the high sensitivity Denka-Seiken method (Roberts, Moulton et al., 2001). Intra-assay and inter-assay coefficients of variation were <3%; assay sensitivity was <0.05 mg/L. Interleukin-6 was determined by commercial ELISA (Quantikine, R&D Systems, Minneapolis,
Minnesota). Intra-assay and inter-assay coefficient of variation were <5%; assay sensitivity was <0.72 pg/mL.

**Power Analysis**

Empirical estimates of the sample size necessary to detect mediation in the sample at .80 Power were determined by consulting power tables compiled by Fritz & Mckinnon (2007). The tables allow for estimation of the sample size necessary to obtain specific levels of power depending on the hypothesized magnitude of theorized alpha and beta paths (Fritz & MacKinnon, 2007). The sample sizes necessary to detect mediation in this dissertation were determined by estimating alpha path strength and beta path strength for theoretically linked groups of mediators.

Based on previous work in our group linking ethnicity and stress and non-stress psychological variables, the alpha paths for ethnicity to stress variables and ethnicity to non-stress psychological variables was estimated to be medium (accounting for approximately 10% of the variance according to Cohen estimates) (Thomas, Nelesen, & Dimsdale, 2004; Thomas, Nelesen, Ziegler, Bardwell, & Dimsdale, 2004; Thomas, Nelesen, Malcarne, Ziegler, & Dimsdale, 2006; Thomas, Bardwell, Ancoli-Israel, & Dimsdale, 2006). Considering the large discrepancies in physical activity levels between AA and CA and consistently noted poorer sleep quality in AA compared to CA (Haskell et al., 2007; Pate et al., 1995; Ruiter, DeCoste, Jacobs, & Lichstein, 2011; U.S. Department of Health and Human Services, 1996) we also estimated that alpha paths from ethnicity to health practices variables would also be medium in size.
Work linking stress to AL has not been conclusive; we therefore conservatively estimated that beta paths for all mediator variables to AL would be small. Given these estimates, it was determined that a sample size of N=368 would be necessary to have .80 Power to detect whether stress variables, non-stress psychological or health practice variables were significant mediators of the relationship between ethnicity and AL.

Statistical Analyses

Missing Data. In order to maximize statistical power, missing data was imputed using a regression approach. Age, gender and SES were each used as predictor variables. All reported analyses were conducted using imputed data. Results using only cases with complete data were also analyzed in order to ensure that the results did not differ substantively from results calculated from imputed data.

Biological Data. The mean and standard deviations of each biological variable were computed and each variable was examined for outliers.

Covariates. The first step of the analysis was to determine the extent to which age, gender and SES\(^1\) were associated with ethnicity and AL. Gender was coded 0 for men, and 1 for women. Ethnicity was coded 0 for AA and 1 for CA. Associations between each demographic covariate and ethnicity were examined using independent samples \(t\)-tests for continuous variables and Fisher exact tests for binary measures.

\(^{1}\) All analyses were repeated controlling for years of education as an alternate measure of SES. Findings were substantively similar across analyses.
Associations between each demographic covariate and AL were examined using correlations. Demographic variables were held constant in all further analysis.

**Aim 1.** The main aim of this dissertation study was to test the hypothesis that differences in stressors, non-stress psychological variables or health practices mediate the relationship between ethnicity and AL. Traditional guidelines for mediation state that three conditions must be met to establish a variable as a mediator. First, the predictor (ethnicity) must be related to the mediator. Second, the predictor variable must be related to the outcome variable. Third, when the outcome variable is regressed onto the mediator and predictor variable, a stronger relationship must exist between the mediator and the outcome variable than the predictor and outcome variable (Baron & Kenny, 1986). Notably, newer methods for conducting mediation do not impose the requirement that there be evidence of a simple association between the predictor and outcome variable in order to test hypotheses about indirect effects (Hayes, 2009).

The first step of the analysis was to determine whether ethnic differences in AL persisted after controlling for demographic covariates. To test this hypothesis, a univariate ANOVA with AL as the outcome variable and ethnicity as the predictor was conducted; the covariates age, gender and SES were held constant.

Next, partial correlations between specific psychosocial stressors, non-stress psychological variables or health practices and ethnicity and AL were explored.

Finally, mediation hypotheses were tested. Sets of variables were tested in a meditational model using a bootstrapping procedure \((n = 5000\) bootstrap resamples) of mediation (Preacher & Hayes, 2004; Preacher & Hayes, 2008). These analyses examine the indirect effects of independent variable (IV; ethnicity) on a dependent variable (DV;
Allostatic Load) through a mediator (e.g., discrimination, perceived stress, etc.) or set of mediators (e.g., stress variables). Using the multivariate, regression-based mediation strategy developed by Preacher & Hayes (2004, 2008), covariates can be held constant during the analysis and multiple mediators may be examined at the same time.

Regression coefficients are calculated for the effect of the IV on the mediating variable (path a), the mediating variable on the DV (path b), and the IV on the DV without the inclusion of mediators (path c), and finally, the IV on the DV after the mediator is considered (path c’). The product-of-coefficients (paths a*b) are calculated using a bootstrapping procedure to yield a point estimate for the indirect (mediating) effect.

Bootstrapping is a nonparametric statistical technique that can be used to gain an estimate of the sampling distribution of a statistic (Mooney, Duval, & Duval, 1993). Through randomly sampling subsets of data and calculating the desired statistic thousands of times, a sampling distribution for the statistic is obtained. The desired statistic and accompanying confidence interval can then be derived as well as a point estimate which provides a test of significance. In this case, the point estimate is associated with the indirect effect of the mediator and the confidence interval of the point estimate provides a test of evaluating if a theorized mediator or set of mediators statistically accounts for a significant part of IV’s prediction of DV (Efron, Tibshirani, & Tibshirani, 1993; Preacher & Hayes, 2004).

In order to minimize the number of statistical tests which were conducted, mediation analysis proceeded in two steps. Per recommendations from Preacher and Hayes (2004), first sets of conceptually meaningful variable sets (e.g., stressors, non-stress psychological variables, and health behaviours) were examined for the potentially
mediating effect of a group of variables. Next, the contribution of individual variables as potential mediators was examined separately.

**Aim 2.** The second aim of the dissertation was to investigate if AL scores calculated using the traditional scoring algorithm performed in a similar fashion to those created using the z-score method. To test this aim, an AL score was calculated using the algorithm from the MacArthur Study of Healthy Aging. The statistical analyses described in Aim 1 were conducted using the new AL score as the outcome variable.

**Aim 3.** The third, exploratory aim, of the dissertation was to determine whether mediators of the ethnicity-AL-association persisted across the sub-factors of AL. In addition to the sum score of AL, subsets of biomarkers reflecting cardiovascular function, metabolism, SNS activity, HPA axis activity and inflammation were created. Mediation analyses were then conducted using each of the sub-factors of AL as outcome variables.
Results

Missing Data

Table 1 reports number of data points available for each variable. Missing data ranged from 0-10.2% across individual variables. Cases that were missing data on at least one variable were not associated with age, gender, SES, ethnicity or AL ($p$s > 0.14).

Associations between Demographic Variables, AL and Ethnicity

Table 1 reports sociodemographic and clinical sample characteristics. The first step of the analysis was to determine the extent to which age, gender and SES were associated with AL and ethnicity. Two-way ANOVA showed that ethnicity was associated with age, such that AA participants in the sample were younger than CA participants ($M_{AA} = 37.41, SD = 9.08$ versus $M_{CA} = 33.49, SD = 9.84, p = 0.01$). Chi-square analysis suggested that ethnicity was not associated with gender (chi-square = 0.29, $p = 0.58$). Two-way ANOVA showed that ethnicity was associated with SES, such that AA participants had higher Hollingshead Total scores, indicative of lower SES ($M_{AA} = 45.43, SD = 13.67$ versus $M_{CA} = 38.06, SD = 15.18, p = 0.01$).

AL was positively correlated with age ($r = 0.26, p < 0.001$). A two-way analysis of variance (ANOVA) revealed that AL was associated with gender, such that males in the sample had significantly higher AL than females ($M_{males} = 0.14, SD = 0.46$ versus $M_{females} = -0.18, SD = 0.44, p <.001$). A second ANOVA showed that AL was associated with ethnicity, such that AA had higher AL than CA ($M_{AA} = 0.12, SD = 0.48$ versus $M_{CA} =$ - .
0.09, $SD = 0.46, p < 0.01$). AL was not correlated with the Hollingshead Total ($r = -0.02, p = 0.80$).

All subsequent analyses were conducted with gender, age and SES included as covariates.

**Aim 1**

The first aim explored whether differences in stressors, non-stress personality variables or health practices mediated the relationship between ethnicity and AL.

**Ethnic Differences in AL.** Univariate ANOVA showed that ethnicity was associated with AL after controlling for covariates, such that AA participants in the sample had higher AL than CA participants ($M_{AA} = 0.110, SE = 0.050$ versus $M_{CA} = -0.091, SE = 0.043, p < 0.01; \eta^2 = 0.05$)

**Correlations between Individual Mediators, Ethnicity and AL.** Next, partial correlations between potential mediator variables, ethnicity and AL were explored. AL was positively associated with the experience of anger subscale ($r = 0.20, p < 0.01$) and subjective sleep quality ($r = 0.22, p < 0.01$) and negatively associated with self-reported exercise ($r = -0.16, p < 0.05$). AA’s reported significantly more perceived stress ($M_{AA} = 14.57, SE = 0.75$ versus $M_{CA} = 12.52, SE = 0.65, p < 0.05$), perceived discrimination ($M_{AA} = 3.41, SE = 0.09$ versus $M_{CA} = 2.57, SE = 0.08, p < 0.01$) and experience of anger ($M_{AA} = 26.22, SE = 2.00$ versus $M_{CA} = 16.33, SE = 1.73, p < 0.01$) than their Caucasian counterparts. AA’s were also significantly more likely to report being smokers ($M_{AA} = 21\%$ versus $M_{CA} = 7\%, p < 0.01$) and having worse subjective sleep quality ($M_{AA} = 5.96$, $M_{CA} = 4.68, p < 0.05$).
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\[ SE = 0.34 \text{ versus } M_{CA} = 4.50, SE = 0.29, p < 0.01 \]. Only the experience of anger subscale and the subjective sleep quality demonstrated significant associations with both ethnicity and AL. See Table 2.

**Psychosocial Stressors as Mediators of the Relationship between Ethnicity and AL.**

Examination of correlations between stress variables showed that perceived stress had low, but significant, correlations with daily hassles, perceived discrimination and job strain. No other significant correlations were observed between stress variables. See Table 3a.

Estimates of indirect effects of ethnicity on AL through each set of mediators is shown in Table 4. The first mediation analysis tested the extent to which perceived stress, daily hassles, perceived discrimination and job strain mediated the relationship between ethnicity and AL. The total effect of ethnicity on AL was not reduced after accounting for the combined mediational effects of perceived stress, daily hassles, perceived discrimination or job strain. The total indirect effect was not significant, indicating that, together the psychosocial stressors did not mediate the effect of ethnicity on AL (Point estimate = -0.002, BCa = -0.081 - 0.081). An examination of the indirect effects of ethnicity through each of the specific meditators indicated that none were significant independent mediators.

**Non-Stress Psychological Variables as Mediators of the Relationship between Ethnicity and AL.** Examination of relationships between non-stress psychological variables showed that the potential mediators were intercorrelated. Expression of anger had significant, moderate correlations with experience of anger and depression.
Expression of anger and interpersonal support had a low, but significant association. Experience of anger was highly correlated with depression and moderately correlated with interpersonal support. Finally, interpersonal support was moderately correlated with depression. (Table 3b)

The second mediation analysis tested the extent to which depression, hostility and interpersonal support served as mediators of the relationship between ethnicity and AL. As shown in Figure 2, non-stress psychological variables significantly mediated the relationship between ethnicity and AL. In other words, the total effect of ethnicity on AL was significantly reduced (path c to path c’) after accounting for the combined mediational effect of non-stress psychological variables (Point Estimate = -0.057, BCa = -0.137 – -0.012). However, non-stress psychological variables were only partial (rather than full) mediators with the direct effect (path c’) remaining significant, indicating that additional variables mediate this relationship. An examination of the indirect effects of ethnicity through each of the individual mediators indicated that only experience of anger was as an independent mediator of the association.

**Health Practices as Mediators of the Relationship between Ethnicity and AL.**

The third mediation analysis tested the extent to which smoking, alcohol consumption, habitual exercise and subjective sleep quality mediated the relationship between ethnicity and AL. Subjective sleep quality had a low, but significant correlation with exercise, such that increased exercise was associated with better subjective sleep quality. Subjective sleep quality was also significantly correlated with smoking, such that participants who endorsed smoking also reported worse sleep quality. No other significant correlations between variables were observed.
As shown in Figure 3, health practices were a significant mediator of the relationship between ethnicity and AL. In other words, the total effect of ethnicity on AL was significantly reduced (path c to path c’) after accounting for the combined mediational effect of health practice variables (Point Estimate = -0.062, BCa = -0.148 - -0.010). Again, health practices variables were only a partial (rather than full) mediator with the direct effect (path c’) remaining significant, indicating that additional variables mediate this relationship. An examination of the indirect effects of ethnicity through individual mediators indicated that only subjective sleep quality served as an independent mediator of the association between ethnicity and AL.

**Aim 2**

The second set of analyses explored whether results were substantively different when AL was calculated using the traditional quartiles method versus z-scores. The statistical steps utilized for Aim 1 were repeated using the AL scores calculated using quartiles.

**Correlations between Individual Mediators with Ethnicity and AL (quartiles).** The first step was to determine the extent to which age, gender and SES were associated with AL (quartiles). The correlation between AL (quartiles) and age approached significance ($r = -0.15, p = 0.053$). A two-way analysis of variance (ANOVA) revealed that AL (quartiles) was associated with gender, such that males in the sample had significantly higher AL (quartiles) than females ($M_{males} = 0.28, SD = 0.19$, versus $M_{females} = 0.21, SD = 0.16, p = 0.01$). A second ANOVA showed that AL (quartiles) was associated with ethnicity, such that AA had higher AL (quartiles) than CA
\(M_{\text{AA}} = 0.12, SD = 0.48 \text{ versus } M_{\text{CA}} = -0.09, SD = 0.46, p < 0.01\). AL (quartiles) was not correlated with the Hollingshead Total \((r = -0.02, p = 0.80)\). All subsequent analyses were conducted with gender, age and SES included as covariates.

**Correlations of Individual Mediators, Ethnicity and AL (quartiles).** Next, associations between specific psychosocial stressors, non-stress psychological variables, health practices, ethnicity and AL (quartiles) were explored. Similar to AL, calculated using the z-scoring method, AL calculated using quartile scoring was positively associated with the experience of anger subscale \((r = 0.19, p < 0.05)\) and subjective sleep quality \((r = 0.18, p < 0.05)\) and was negatively associated with habitual exercise \((r = -0.17, p < 0.05)\). No other significant associations were detected. See Table 2.

**Ethnic Differences in AL (quartiles).** Again, univariate ANOVA showed that ethnicity was associated with AL calculated using quartiles. After controlling for covariates, AA participants in the sample had higher AL than CA participants \((M_{\text{AA}} = 0.28, SE = 0.02 \text{ versus } M_{\text{CA}} = 0.22, SE = 0.02, p < 0.01; \eta^2 = 0.04)\).

**Psychosocial Stressors as Mediators of the Relationship between Ethnicity and AL (quartiles).** Estimates of indirect effects of ethnicity on AL (quartiles) through each of the mediators are shown in Table 5. The first mediation analysis tested the extent to which perceived stress, daily hassles, perceived discrimination and job strain mediated the relationship between ethnicity and AL (quartiles). Results using the AL calculated using the quartile scoring method were substantively the same as those using the z-scoring method. Again, the total indirect effect was not significant, indicating that, together the psychosocial stressors did not mediate the effect of ethnicity on AL (quartiles) \((\text{Point estimate} = -0.003, \text{BCa} = -0.0360 - 0.0259)\). An examination of the
indirect effects of ethnicity through each of the specific mediators indicated that none were significant independent mediators.

**Non-Stress Psychological Variables as Mediators of the Relationship between Ethnicity and AL (quartiles).** The second mediation analysis tested the extent to which depression, hostility and interpersonal support served as mediators of the relationship between ethnicity and AL (quartiles). Again, non-stress psychological variables emerged as significant mediators of the relationship between ethnicity and AL (quartiles). In other words, the total effect of ethnicity on AL (quartiles) was significantly reduced (path c to path c') after accounting for the combined mediational effect of non-stress psychological variables (Point Estimate = -0.020, BCa = -0.046 - 0.003). Non-stress psychological variables fully mediated ethnic differences in AL (quartiles), with the direct effect (path c') becoming non-significant. An examination of the indirect effects of ethnicity through each of the individual mediators indicated that only expression of anger emerged as a significant individual mediator of the association.

**Health Practices as Mediators of the Relationship between Ethnicity and AL (quartiles).** The third mediation analysis tested the extent to which smoking, alcohol consumption, habitual exercise and subjective sleep quality served as mediators of the relationship between ethnicity and AL (quartiles). Health practices were a full mediator of the relationship between ethnicity and AL. In other words, the total effect of ethnicity on AL was significantly reduced (path c to path c') after accounting for the combined mediational effect of health practice variables (Point Estimate = -0.019, BCa = -0.052 - 0.002). An examination of the indirect effects of ethnicity through each of the individual
mediators indicated that none of the health practices were significant independent mediators of the association.

**Aim 3**

The third set of analyses explored whether sub-factors of AL were associated with ethnicity and mediators in a similar fashion as the composite score. Mediation analysis was performed with each of the sub-factors of AL as an outcome variable. Sets of stress variables, non-stress psychological variables and health practices described above were explored as potential mediators.

Univariate ANOVA showed that ethnicity was associated with some sub-factors of AL after controlling for covariates, such that AA participants in the sample had higher cardiovascular AL ($M_{AA} = 0.35, SE = 0.04$ versus $M_{CA} = 0.18, SE = 0.04, p < 0.001$) and inflammatory AL ($M_{AA} = 0.30, SE = 0.03$ versus $M_{CA} = 0.20, SE = 0.03, p < 0.01$) than CA participants. Ethnic differences in metabolic AL ($M_{AA} = 0.27, SE = 0.04$ versus $M_{CA} = 0.22, SE = 0.04, p = 0.90$), SNS AL ($M_{AA} = 0.35, SE = 0.04$ versus $M_{CA} = 0.18, SE = 0.04, p = 0.26$) and HPA AL ($M_{AA} = -0.11, SE = 0.12$ versus $M_{CA} = 0.07, SE = 0.10, p =0.18$) were not detected.

**Psychosocial Stressors as Mediators of the Relationship between Ethnicity and Sub-Factors of AL.**

The first set of mediation analyses tested the extent to which perceived stress, daily hassles, perceived discrimination and job strain mediated the relationship between ethnicity and sub-factors of AL. Psychosocial stressors did not mediate cardiovascular (Point estimate = -0.013, BCa = -0.158 – 0.147), metabolic (Point estimate = 0.011, BCa
= -0.084 – 0.113), SNS (Point estimate = 0.034, BCa = -0.156 – 0.244), inflammatory (Point estimate = -0.027, BCa = -0.185 – 0.077) or HPA (Point estimate = -0.059, BCa = -0.237 – 0.130) sub-factors of AL. An examination of the indirect effects of ethnicity through each of the specific mediators indicated that none were significant independent mediators. See Table 6.

**Non-Stress Psychological Variables as Mediators of the Relationship between Ethnicity and Sub-Factors of AL.** The second set of mediation analyses tested the extent to which depression, hostility and interpersonal support mediated the relationship between ethnicity and sub-factors of AL. Psychosocial and psychological variables mediated differences in the cardiovascular sub-factor of AL (Point estimate = -0.100, BCa = -0.265 - -0.008), but not metabolic (Point estimate = -0.043, BCa = -0.163 – 0.021), SNS (Point estimate = -0.081, BCa = -0.219 – 0.001), inflammatory (Point estimate = -0.029, BCa = -0.113 - 0.045) or HPA Axis (Point estimate = -0.034, BCa = -0.163 – 0.091) sub-factors of AL. See Table 7.

An examination of the indirect effects of ethnicity through each of the specific mediators indicated that the expression of anger subscale mediated ethnic differences in SNS functioning (Point estimate = -0.087, BCa = -0.245 - -0.003).

There are well established associations between hostility and BP. Given psychosocial and psychological variables mediated only the cardiovascular sub-factor of AL, the question was raised as to whether the contribution of BP driving main effects. To test this question the cardiovascular component of AL was removed from the total AL score and mediation analysis was again conducted. After removing the cardiovascular sub-factor of AL from the composite score, there remained significant ethnic differences
in AL ($M_{AA} = 0.073, SE = 0.049$ versus $M_{CA} = -0.063, SE = 0.042, p < 0.05$). Again, non-stress psychological variables emerged as significant mediators of the relationship between ethnicity and AL (minus cardiovascular factors) (Point Estimate = -0.047, BCa = -0.115 – -0.006). Non-stress psychological variables fully mediated ethnic differences in AL (quartiles), with the direct effect (path c’) becoming non-significant. An examination of the indirect effects of ethnicity through each of the individual mediators indicated that again, only expression of anger emerged as a significant individual mediator of the association (Point Estimate = -0.051, BCa = -0.124 – -0.010).

**Health Practices Variables as Mediators of the Relationship between Ethnicity and sub-factors of AL.** The final set of mediation analyses tested the extent to which smoking, alcohol consumption, habitual exercise and subjective sleep quality mediated the relationship between ethnicity and sub-factors of AL. Health practices mediated differences in the metabolic sub-factor of AL (Point estimate = -0.080, BCa = -0.206 - -0.010), but not cardiovascular (Point estimate = -0.076, BCa = -0.288 – 0.032), SNS (Point estimate = -0.036, BCa = -0.187 – 0.056), inflammatory (Point estimate = -0.054, BCa = -0.168 - 0.044) or HPA Axis (Point estimate = -0.069, BCa = -0.217 – 0.019) sub-factors. An examination of the indirect effects of ethnicity through each of the specific mediators indicated that none were significant independent mediators. See Table 8.
Discussion

In doing this work, it has become clear that the constituents of AL and the analytic techniques that serve to create the variable are not uniform or clearly defined. Additionally, questions remain about the theory underpinning the model. That being said, variables that were most representative of the major categories thought to encompass AL were selected for this dissertation and consistent with previously published work, AA had higher AL than CA. Ethnic differences in AL persisted after controlling for differences in age, gender and SES and emerged regardless of the scoring method employed. Importantly, differences in AL appeared to be associated with ethnic differences in hostility and subjective sleep quality, variables which emerged as partial mediators of the association between ethnicity and AL. Interestingly, stress variables contributed very little. These findings contribute to a long tradition in behavioural medicine, linking hostility to health outcomes. They also highlight a relatively new, but important area, demonstrating relationships between sleep and health.

Aim 1

Stress Variables. AA have consistently been shown to have higher AL than their Caucasian counterparts (Geronimus et al., 2006; Seeman et al., 2010). A main tenet of the AL theory is that chronic exposure to stressors results in ‘wear and tear’ on the body, culminating in increases in AL. It has been suggested that differential exposure to chronic stressors, such as discrimination, may mediate ethnic differences in AL.
(Geronimus et al., 2006; Mays et al., 2007). Although variables assessing levels of perceived stress and discrimination were higher in the AA group in our sample, no stress variables were associated with AL, nor did stressors emerge as mediators of the relationship between ethnicity and AL.

It is unclear why an association between stress and AL was not observed in our sample. Results from the SEBAS cohort and the Texas City cohort have linked indices of perceived stress and hassles to increases in AL (Gersten, 2008; Glei et al., 2007; Goldman et al., 2005; Mair et al., 2011). Additionally, stress in the domains of work and social functioning have been associated with increased AL (Bellingrath et al., 2009; Gallo et al., 2011; Juster et al., 2011; Kinnunen et al., 2005; Li et al., 2007; Schnorpfeil et al., 2003; Sun et al., 2007); however, other studies have failed to detect positive associations between measures of stress and AL (Clark et al., 2007; Hawkley et al., 2011).

Potentially, null findings in this dissertation are the result of having controlled for SES in each of the analyses. SES has strong and consistent associations with AL and studies that have reported associations between perceived stress and AL have not always included SES as a covariate (Gersten, 2008; Glei et al., 2007; Goldman et al., 2005; Seeman et al., 2010). Alternatively, it may be important to more finely distinguish the types of stressors that mediate the ethnicity-AL association. Specific stressors such as having spouse in poor health, financial difficulty and widowhood have each been individually linked to higher levels of AL (Weinstein, Goldman, Hedley, Yu-Hsuan, & Seeman, 2003). Additionally, one study reported that stress in the areas of work, finances and caregiving were associated with increased AL whereas stress in the areas of health and relationship problems were not (Gallo et al., 2011). Perhaps, more specific
identification of stressors would have revealed stress variables to be mediators of the ethnicity-AL relationship in this sample. It is also possible that this study was simply underpowered to detect associations between stressors and AL and use of a larger sample size may have elucidated undetected relationships.

**Non-Stress Psychological Variables.** Non-stress variables emerged as significant mediators of ethnic differences in AL and the experience of anger subscale on the Buss-Durkee Hostility Inventory was a significant independent mediator of the association. The emergence of hostility as a mediator of the ethnicity-AL connection adds to a small body of literature showing associations between hostility and AL. For example, Sun et al. (2007) found that hostility was associated with AL in a sample of older male Chinese industrial workers. Additionally, two studies have reported that hostility partially explained associations between SES and AL (Hawkley et al., 2011; Kubzansky et al., 1999).

The experience of anger subscale captures a construct defined as an individual’s chronic internal experience of anger, irritation, suspicion and annoyance (Musante, MacDougall, Dembroski, & Costa, 1989; Riley & Treiber, 1989). Notably, many of the items that make up the experience of anger subscale on the Buss-Durkee tap an individual’s belief that they have been mistreated by others (e.g., “I don’t seem to get what’s coming to me”, “other people always seem to get the breaks”) or the need to remain hypervigilant to surroundings (e.g., “I tend to be on guard with people who are sometimes more friendly than I expected” and “My motto is “never trust strangers”). AA in our sample had significantly higher scores on the experience of anger subscale than their CA counterparts. Potentially, higher levels of internalized anger stem from chronic
exposure to unfair treatment. It has been established that race-related maltreatment evokes anger and anger suppression has been cited as coping mechanisms in response to exposure to interpersonal racism (Brondolo, Brady ver Halen, Pencille, Beatty, & Contrada, 2009; Brondolo et al., 2008; Broudy et al., 2007). Although path analyses are outside of the scope of this dissertation, it is notable that in our sample, discrimination was positively associated with the experience of anger subscale ($r = 0.27, p < 0.001$).

Despite years of investigation, the question still remains as to how hostility “gets under the skin” to influence AL. In addition to well-established relationships with BP and cardiac function, hostility has been associated with physiologic functioning across several systems encompassed in the definition of AL. Hostility in adolescents and the middle-aged predicts the emergence metabolic syndrome, a construct composed of variables highly associated with AL (Elovainio et al., 2011; Nelson, Palmer, & Pedersen, 2004; Räikkönen, Matthews, & Salomon, 2003; Räikkönen, Matthews, Sutton-Tyrrell, & Kuller, 2004). Hostility and anger have also been shown to predict impairments in glucose metabolism, fasting plasma insulin and plasma lipid levels (Goodman, Must, Daniels, & Dolan, 2010; Richards, Hof, & Alvarenga, 2000; Shen, Countryman, Spiro III, & Niaura, 2008; Surwit et al., 2002; Weidner, Sexton, McLellarn, Connor, & Matarazzo, 1987). Additionally, hostility has been associated with increases in markers of inflammation both cross-sectionally and longitudinally (Elovainio et al., 2011; Shivpuri et al., 2011). Finally, levels of hostility have been directly associated with NE (Sherwood, Hughes, Kuhn, & Hinderliter, 2004). The AL model posits chronic exposure to stressors results in alternations of primary mediators (e.g., SNS variables), yielding changes in secondary mediators (e.g., BP) and eventually higher AL. Sub-factor analyses in this
dissertation showed that the expression of anger subscale from the Buss Durkee Hostility Inventory was a mediator of ethnic differences in SNS and cardiovascular AL. Potentially, changes in neuroendocrine markers linked to hostility, and not stressors, lead to increased resting levels of downstream systems such as BP. Clearly, longitudinal work is necessary to further elucidate the pathways through which hostility may influence AL.

Health Practice Variables. In addition to non-stress variables, health practices also helped to explain the relationship between ethnicity and AL, specifically, poor sleep quality emerged as a partial mediator of the relationship. It has been theorized that poor sleep quality may negatively impact AL (McEwen, 2006; Van Cauter & Spiegel, 1999). One investigation found that in a sample of 51- to 69-year-old White, Black, and non-Black Hispanic men and women subjective sleep quality was a partial mediator of the relationship between SES and AL (Hawkley et al., 2011; Kubzansky et al., 1999).

Traditional theories of AL suggest two types of negative physiological responses to stimuli result in elevations of AL. The first is frequent activation of physiological systems in response to stress and the second is failure to shut down a response after stressors are removed (McEwen, 1998). Preliminary evidence linking sleep to AL suggests that we should consider not only the effects of challenges to physiological systems, but also the importance of restorative processes such as those that occur during sleep (Hawkley et al., 2011). It is also plausible that physiological dysregulation associated with AL leads to poorer sleep quality. Ample evidence shows the deleterious effects of sleep deprivation and poor sleep quality on health outcomes; however, longitudinal studies investigating this question are necessary to determine causality.
**Aim 2**

In our sample, ethnicity accounted for an equivalent amount of variance in AL, regardless of whether the traditional quartile scoring method or the z-score method was used. Additionally, the same non-stress variables and health practices emerged as mediators for both AL outcome variables. AL has been scored numerous ways and variable inclusion has been diverse. Findings from our sample mirrored previous finding showing that differential scoring methods appear to have little impact on results (Karlamangla et al., 2002; Seeman et al., 2010; Seplaki et al., 2005).

Each method of scoring the data has advantages and disadvantages. Using a z-score method of calculating AL allows for greater variation in the data and may more accurately reflect the continuous nature of the variable (Hawkley et al., 2011; Mair et al., 2011). In contrast, the simplicity of the quartile method may be more appealing to clinicians. Continued discussion and study of the best approaches with which to score AL is clearly necessary; however, it is likely that larger, epidemiological datasets, with longitudinal follow-up will be necessary to answer questions related to optimal scoring.

**Aim 3**

Findings from the sub-factor analyses conducted in this dissertation were largely null. Similar to the main analyses, stress variables did not emerge as mediators in any sub-factor analyses. Non-stress variables mediated ethnic differences in a cardiovascular measure of AL, with the experience of anger emerging as an independent variable of the association. As a group, non-stress variables did not mediate ethnic differences in SNS AL; however, the individual variable, experience of anger was a significant mediator.
Additionally, health practices mediated ethnic differences in a metabolic component of AL.

Meta-analyses have consistently identified positive relationships between experiential anger and BP (Jorgensen, Johnson, Kolodziej, & Schreer, 1996; Schum, Jorgensen, Verhaeghen, Sauro, & Thibodeau, 2003; Suls, Wan, & Costa, 1995). Given the well-established associations between anger and BP, we investigated whether the associations between expression of anger and AL remained after components of cardiovascular functioning were removed from the model. Findings indicated that the experience of anger variable continued to mediate differences in AL, even after markers of BP were removed from the composite score, suggesting that the association was not solely driven by the relationship between hostility and BP.

AL is hypothesized to represent moderate dysregulation across multiple systems, resulting in significant health risks (Seeman et al., 1997). According to the AL model, alterations in stress hormone functioning results in dysregulation of other physiological systems, leading to the development of illness (Juster et al., 2011); however the theoretical model has come under criticism for lack of consistent evidence identifying associations between psychosocial stressors and theoretical primary mediators in the AL model. Some authors have questioned whether the crude distinction of ‘primary’ and ‘secondary’ mediator categories adequately capture and dynamic, nonlinear and adaptive nature of the interactions that occur between biomarkers included in the AL variable (Buckwalter et al., 2011). Additionally, the question as to whether AL as a concept has any additive or predictive value beyond that of traditionally identified pathways linking psychology and health has yet to be conclusively determined. Findings from this
dissertation showed that hostility was associated with alterations in SNS. Theoretically, SNS alternations could result in higher set points in associated physiologic variables; however, these findings must be interpreted with caution, as the analyses were exploratory. Again, longitudinal work is necessary to elucidate whether differential levels of psychological variables actually result in changes to specific physiologic systems, leading to increases in AL.

**Limitations**

In doing this work, it has become clear that the constituents of AL are not uniform and that the ways of analyzing AL are under-explored. In contrast to the MacArthur studies (Seeman et al., 1997; Seeman et al., 2004), our measure of AL did not include DHEA-S; furthermore, we substituted waist circumference for waist-hip ratio and fasting glucose for glycosylated hemoglobin. Deciding which variables to include in the creation of an AL score is a continuing evolution in the literature and it remains to be established what the most important and critical markers of AL are. Many post-hoc analyses, such as those from this dissertation, use markers from previously established data sets to “piece-together” a variable reflecting allostasis. To address this weakness, we selected AL variables from categories that are constant within the field (Seeman et al., 2010). Our conceptualization of the model approximates that used in other studies, and our findings replicate those reported in the literature (e.g. that AA had higher AL than CA). Still, differential variable inclusion could make it hard to replicate results from studies using alternative conceptualizations of AL, for example, the construction of the AL variable in this dissertation may have obscured the ability to detect associations between stress
variables and AL that have been observed using different conceptualizations of the model.

The study is also limited by cross-sectional assessment. Further research will be necessary to determine the temporal nature of the relationships between stressors, non-stress psychological variables and health practices and AL. Also, longitudinal studies will be necessary to adequately track the sequence of physiologic changes theorized to lead to increased overall AL.

Additionally, the measure of SES in the study is composed of an assessment of occupation and education, which may not accurately reflect other indices of SES related to AL such as wealth, financial stress, and perceived adequacy of resources. Also, participants were all working adults from the San Diego, California area and results may not generalize to individuals from a wider socioeconomic range or to groups from other parts of the country.

Finally, the study is limited by issues of power. Non-stress psychological variables and health practices significantly mediated ethnic differences in AL; however, it is possible that other significant findings were obscured by our small sample size.

Conclusions

Researchers have been discussing relationships between hostility and health since the inception of behavioural medicine. In the first episode of Psychosomatic Medicine, Franz Alexander theorized that “inhibited aggressive impulses” and “inhibited hostile tendencies” put acute strain on the heart, resulting in rises in BP (Alexander, 1939). Since then, tomes of research have followed, showing that anger and hostility exert a toll on
cardiovascular health. Findings from this dissertation continue a long trajectory of research about the impact of hostility on health, extending the findings to suggest that hostility is associated with a measure of widespread physiologic dysregulation. Results from this dissertation also highlight a newer body of behavioural medicine research investigating the impact of nighttime functioning on physiological states.

Between 1971 and 1972, the Life Change Events Study (LCES) investigated anger-coping responses and physiologic health. Subjects were exposed to two hypothetical, 'unjustified' anger-provoking situations and scores of anger, guilt and protest were created to capture each participant’s response to the hypothetical situations. In the early studies, suppression of anger was associated with increased BP (Harburg et al., 1973). In later work, Harburg and colleagues (2003) examined the health outcomes of respondents from the initial study. Findings suggested that suppression of anger, as assessed in 1971 was associated with cardiac death and deaths linked cancer in the years following (Harburg, Julius, Kaciroti, Gleiberman, & Schork, 2003).

The AL model suggests that dysregulation across physiologic systems predicts the emergence of multiple disease states and eventually mortality. Interpretation of the findings from this dissertation, in the context of Harburg’s work, leads one to theorize that potentially, suppressed anger was disrupting more than BP regulation in those early studies. Perhaps wide scale dysregulation of multiple systems, similar to that hypothesized to occur in AL was occurring and potentially that dysregulation lead to early mortality. While this remains speculation, the concept of AL may allow for better understanding of how psychological variables can influence the progression and emergence of multiple disease states.
## Tables and Figures

### Table 1: Sociodemographic and Clinical Sample Characteristics

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#### Psychosocial Stressors

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#### Health Practices

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<td>C-Reactive Protein (mg/L)</td>
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<td>Interleukin-6 (pg/mL)</td>
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Note. Gender and smoking data are n (%). All others are Mean ± SD. HDL = High Density Lipoprotein; SEM = Standard Error of the Mean
*p < .05, **p < .01
Table 2: Partial Associations between Posited Mediators with Ethnicity and Allostatic Load

<table>
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<tr>
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<th>Allostatic Load (z-score)</th>
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<td>3.41 (.09)</td>
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<td>Non-Stress Psychological Variables</td>
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<td>Expression of Anger</td>
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<td>&gt; -0.01</td>
<td>38.84 (1.77)</td>
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<td>Experience of Anger</td>
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<td>0.19*</td>
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Table 2: Continued

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<th>Allostatic Load (z-score)</th>
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<td>Exercise</td>
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<td>Smoke (% yes)</td>
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<td>0.18*</td>
<td>5.96 (0.34)</td>
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All analyses controlled for Age, Gender and Socioeconomic Status

*p < .05, two-tailed; ** p < .01, two-tailed
Table 3a: Intercorrelations between Psychosocial Stressors

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<td>1. Perceived Stress</td>
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<tr>
<td>2. Daily Hassles</td>
<td>.28***</td>
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</tr>
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<td>3. Perceived Discrimination</td>
<td>.18*</td>
<td>.07</td>
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<td>4. Job Strain</td>
<td>-.21**</td>
<td>-.12</td>
<td>-.11</td>
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Table 3b: Intercorrelations between Non-Stress Psychological Variables

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<td>2. Experience of Anger</td>
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</tr>
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<td>3. Depression</td>
<td>.30***</td>
<td>.54***</td>
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</tr>
<tr>
<td>4. Interpersonal Support</td>
<td>-.17*</td>
<td>-.38**</td>
<td>-.35***</td>
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Table 3c: Intercorrelations between Health Practices

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<td>2. Smoke</td>
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<td>3. Alcohol Use</td>
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<td>4. Sleep Quality</td>
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<td>.23**</td>
<td>.06</td>
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*p < .05, two-tailed; ** p < .01, two-tailed, ***p < .001, two-tailed
Table 4: Summary of mediation results (standard errors in parentheses, 5000 bootstrap samples) with Allostatic Load using z-scores as the outcome variable.

<table>
<thead>
<tr>
<th>Independent Variable (IV)</th>
<th>Mediating Variable (MV)</th>
<th>Effect of IV on M (a)</th>
<th>Effect of M on DV (b)</th>
<th>Direct effects (c')</th>
<th>Indirect effect (a*b)</th>
<th>BCa Confidence Interval</th>
<th>Total effects (c)</th>
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<td></td>
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<tr>
<td>Race</td>
<td>Perceive d Stress</td>
<td>-2.053 (1.014)*</td>
<td>.001</td>
<td>-0.198</td>
<td>-0.001</td>
<td>-0.029 – 0.201</td>
<td>-0.201</td>
</tr>
<tr>
<td>Hassles</td>
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<td>-0.108 (-0.094)</td>
<td>-0.094</td>
<td>0.010</td>
<td>-0.004</td>
<td>(0.005) (0.077)*</td>
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<tr>
<td>Discrimination</td>
<td>(0.091)</td>
<td>-0.831 0.019</td>
<td>0.019</td>
<td>-0.016</td>
<td>-0.095</td>
<td>(0.126)* (0.042)</td>
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<tr>
<td>Job</td>
<td>(2.085)</td>
<td>1.354 0.003</td>
<td>0.003</td>
<td>0.004</td>
<td>-0.006</td>
<td>(0.003)</td>
<td></td>
</tr>
<tr>
<td>Strain</td>
<td></td>
<td>(2.085) (0.003)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>-0.033</td>
</tr>
<tr>
<td><strong>Non-Stress Psychological Variables</strong></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Race</td>
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<td>-2.647 (2.388)</td>
<td>-0.002</td>
<td>-0.144</td>
<td>0.005</td>
<td>-0.006 – 0.201</td>
<td>-0.201</td>
</tr>
<tr>
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<td>-9.891 0.007</td>
<td>-0.064</td>
<td>-0.147</td>
<td>(0.004)</td>
<td></td>
<td>-0.039 (0.039)</td>
</tr>
<tr>
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<td>-0.432 -0.005</td>
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<td>-0.010</td>
<td>(0.004)</td>
<td>-0.019</td>
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<tr>
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<td>-0.012</td>
<td>-0.056</td>
<td>-0.020</td>
<td>-0.201</td>
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<td>-0.005</td>
<td>-0.038</td>
<td>(0.070)*</td>
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<td>-0.033 (0.068)*</td>
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<td>-0.039</td>
<td>(0.101)</td>
<td>-0.012</td>
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<tr>
<td></td>
<td>(0.051) (0.103)</td>
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<td></td>
<td>-0.004</td>
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Table 4: Continued

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<th>Direct effects (c')</th>
<th>Indirect effect (a'b)</th>
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<th>Total effects (c)</th>
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*p < .05, two-tailed
Table 5: Summary of mediation results (standard errors in parentheses, 5000 bootstrap samples) with Allostatic Load using quartiles as the outcome variable.

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<th>Independent Variable (IV)</th>
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<th>Direct effects (c')</th>
<th>Indirect effect (ab)</th>
<th>BCa Confidence Interval</th>
<th>Total effects (c)</th>
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<td>-0.005</td>
<td>-0.070</td>
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<td></td>
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<td>(0.002)</td>
<td>(0.030)*</td>
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<td>(0.016)</td>
<td>(0.026)</td>
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<td>-0.005</td>
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<td>(0.036)</td>
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<tr>
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<td>0.010</td>
<td>-0.008</td>
<td>-0.039</td>
<td>-0.013</td>
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<td>(0.016)</td>
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<td>0.001</td>
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<td>(0.001)</td>
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<tr>
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<td>(2.701)*</td>
<td>(0.001)*</td>
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<tr>
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<td>0.007</td>
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<td>-0.007</td>
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<td>(0.002)</td>
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<td>&lt; 0.001</td>
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<td>-0.005</td>
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<td></td>
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<td>(0.309)</td>
<td>(0.007)</td>
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<tr>
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<td>-0.006</td>
<td>-0.023</td>
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<td>(0.028)</td>
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<td>(0.026)</td>
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<td></td>
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<td>-0.103</td>
<td>0.011</td>
<td>-0.001</td>
<td>-0.012</td>
<td>-0.012</td>
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<tr>
<td></td>
<td></td>
<td>(0.052)*</td>
<td>(0.040)</td>
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Table 5: Continued

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<th>Effect of M on DV (b)</th>
<th>Direct effects (c')</th>
<th>Indirect effect (a'b)</th>
<th>BCa Confidence Intervals</th>
<th>Total effects (c)</th>
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<td>(0.040)</td>
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<td>(0.005)</td>
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*p < .05, two-tailed
Table 6. Summary of mediation results (standard errors in parentheses, 5000 bootstrap samples) with Stress Variables and Sub-Factors of Allostatic Load

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<th>Indepentent Variable (IV)</th>
<th>Mediating Variable (MV)</th>
<th>Dependent Variable (DV)</th>
<th>Effect of IV on M (a)</th>
<th>Effect of M on DV (b)</th>
<th>Direct effect (ab)</th>
<th>Indirect effect (a'b)</th>
<th>BCa Confid. Interval</th>
<th>Total effects (c)</th>
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<tr>
<td>Race Perceived Stress CVD</td>
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<td>-2.053</td>
<td>-0.016</td>
<td>-0.003</td>
<td>-0.047</td>
<td>(1.014)</td>
<td>(0.134)*</td>
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<td>Hassles</td>
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<td>-0.331</td>
<td>0.036</td>
<td>-0.032</td>
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<td>0.064</td>
<td>-0.054</td>
<td>-0.184</td>
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<td>0.087</td>
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<td>-0.016</td>
<td>-0.008</td>
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<td>(0.096)</td>
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<td>0.017</td>
<td>-0.014</td>
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<td>-0.061 (0.146)</td>
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<td>Mediating Variable (MV)</td>
<td>Dependent Variable (DV)</td>
<td>Effect of IV on M (a)</td>
<td>Effect of M on DV (b)</td>
<td>Direct effects (c')</td>
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<td>BCa Confidence Intervals</td>
<td>Total effects (c)</td>
</tr>
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<td>0.012</td>
<td>-0.001</td>
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<td>0.002</td>
<td>0.003</td>
<td>-0.011</td>
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<td>Hassles</td>
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<td>-0.112</td>
<td>0.012</td>
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<td>0.003</td>
<td>0.004</td>
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Table 6: Continued

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<tr>
<th>Independent Variable (IV)</th>
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<th>Dependent Variable (DV)</th>
<th>Effect of IV on M (a)</th>
<th>Effect of M on DV (b)</th>
<th>Direct effect (a*b)</th>
<th>Indirect effect (c')</th>
<th>BCa Confidence Intervals</th>
<th>Total effects (c)</th>
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<td>(2.085) (0.006)</td>
<td>-0.067</td>
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*p < .05, two-tailed
Table 7: Summary of mediation results (standard errors in parentheses, 5000 bootstrap samples) with Non-Stress Psychological Variables and Sub-Factors of Allostatic Load

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<tr>
<th>Independent Variable</th>
<th>Mediating Variable</th>
<th>效果 of IV on M</th>
<th>Effect of M on DV</th>
<th>Direct effects (c’)</th>
<th>Indirect effects (a*b)</th>
<th>BCa Confidence Interval</th>
<th>Total effects (c)</th>
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<tr>
<td>Race Expression of Anger</td>
<td>CVD</td>
<td>-2.647</td>
<td>-0.007</td>
<td>0.392</td>
<td>0.019</td>
<td>-0.010</td>
<td>-0.492</td>
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<tr>
<td>Race Expression of Anger</td>
<td>Metabolic</td>
<td>-2.647</td>
<td>-0.003</td>
<td>0.042</td>
<td>0.009</td>
<td>-0.009</td>
<td>-0.085</td>
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<tr>
<td>Race Expression of Anger</td>
<td>SNS</td>
<td>-2.647</td>
<td>-0.001</td>
<td>0.090</td>
<td>0.001</td>
<td>-0.024</td>
<td>-0.171</td>
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* p < 0.05
Table 7: Continued

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<tr>
<th>Independent Variable (IV)</th>
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<th>Dependent Variable (DV)</th>
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<th>Effect of M on DV (b)</th>
<th>Direct effects (c')</th>
<th>Indirect effects (a'b)</th>
<th>BCa Confidence Intervals</th>
<th>Total effects (c)</th>
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<tbody>
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<td>0.005</td>
<td>0.023 -</td>
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<td></td>
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<td>(0.010)</td>
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<td>Interpersonal Support</td>
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<td>Inflammation</td>
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<td>0.006</td>
<td>-0.334</td>
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<td>-0.363</td>
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<tr>
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<td></td>
<td>(2.388)</td>
<td>(0.004)</td>
<td>(0.123)</td>
<td>0.008</td>
<td>(0.107)*</td>
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</tr>
<tr>
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<td>-9.891</td>
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<td></td>
<td>(2.701)*</td>
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<td>-0.013 -</td>
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<td>(1.412)</td>
<td>(0.007)</td>
<td>0.039</td>
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<tr>
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<td>0.007</td>
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<td>(0.029)</td>
<td>0.000</td>
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<td>(0.163)</td>
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<td>(0.157)</td>
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</tr>
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<td></td>
<td>(2.701)*</td>
<td>(0.006)</td>
<td>0.039</td>
<td>0.046</td>
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<td></td>
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<td>(0.010)</td>
<td>0.008</td>
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<td></td>
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<td>-0.026</td>
<td>0.001</td>
<td>-0.023 -</td>
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<td>(0.042)</td>
<td>0.035</td>
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*p < .05, two-tailed
Table 8: Summary of mediation results (standard errors in parentheses, 5000 bootstrap samples) with Health Practices and Sub-Factors of Allostatic Load

<table>
<thead>
<tr>
<th>Independent Variable (IV)</th>
<th>Mediating Variable (MV)</th>
<th>Dependent Variable (DV)</th>
<th>Effect of IV on M (a)</th>
<th>Effect of M on DV (b)</th>
<th>Direct effects (c)</th>
<th>Indirect effects (a'b)</th>
<th>BCa Confidence Intervals Total effects (c)</th>
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<tr>
<td>Health Practice AL Sub-Factor</td>
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<td></td>
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</tr>
<tr>
<td>Race</td>
<td>Smoke</td>
<td>CVD</td>
<td>-0.103 (0.052)*</td>
<td>-0.023 (0.203)</td>
<td>0.416</td>
<td>0.002</td>
<td>-0.048 – 0.059 (0.14 1) -0.492</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Metabolic</td>
<td>-0.103 (0.052)*</td>
<td>0.032 (0.141)</td>
<td>0.005</td>
<td>-0.003</td>
<td>-0.047 – 0.026 (0.09 8) -0.085</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SNS</td>
<td>-0.103 (0.052)*</td>
<td>-0.196 (0.221)</td>
<td>0.135</td>
<td>0.020</td>
<td>-0.012 – 0.094 (0.15 4) -0.171</td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
<td></td>
<td>0.056 (0.051)</td>
<td>-0.420 (0.144)</td>
<td>-0.023</td>
<td>-0.088</td>
<td>-0.088 – 0.009</td>
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<tr>
<td>Exercise</td>
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<td></td>
<td>0.136 (0.076)</td>
<td>-0.146 (0.096)</td>
<td>-0.020</td>
<td>-0.086</td>
<td>-0.086 – 0.004</td>
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<tr>
<td>Sleep</td>
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<td>-1.631 (0.456)*</td>
<td>0.046 (0.023)</td>
<td>-0.074</td>
<td>-0.236</td>
<td>-0.236 – 0.007</td>
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<tr>
<td>Quality</td>
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<td>0.056 (0.051)</td>
<td>-0.065 (0.225)</td>
<td>-0.3004</td>
<td>0.025</td>
<td>-0.050– 0.025</td>
</tr>
<tr>
<td>Independent Variable (IV)</td>
<td>Mediating Variable (MV)</td>
<td>Dependent Variable (DV)</td>
<td>Effect of IV on M (a)</td>
<td>Effect of M on DV (b)</td>
<td>Direct effects (c')</td>
<td>Indirect effects (a'b)</td>
<td>BCa Confidence Interval (c)</td>
</tr>
<tr>
<td>---------------------------</td>
<td>-------------------------</td>
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<td>----------------------</td>
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</tr>
<tr>
<td>Exercise</td>
<td></td>
<td></td>
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<td>0.080</td>
<td>0.011</td>
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<td>-1.467</td>
<td>0.039</td>
<td>-</td>
<td>-0.235 –</td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>Smoke</td>
<td>Inflammation</td>
<td>-0.103</td>
<td>0.292</td>
<td>-0.309</td>
<td>-0.093 – 0.363</td>
<td>0.004 (0.107)</td>
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<tr>
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<td>0.002</td>
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<tr>
<td>Race</td>
<td>Smoke</td>
<td>HPA</td>
<td>-0.103</td>
<td>0.292</td>
<td>0.251</td>
<td>-0.156 – 0.182</td>
<td>0.008 (0.157)</td>
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<tr>
<td>Exercise</td>
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<td>0.136</td>
<td>0.086</td>
<td>0.012</td>
<td>-0.022</td>
<td></td>
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<tr>
<td>Sleep</td>
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<td></td>
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<td>0.025</td>
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<td>(0.456)*</td>
<td>0.027</td>
<td>0.040</td>
<td>0.026</td>
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*p < .05, two-tailed
Figure 1: Frequency of variables included in the definition of AL in 14 studies assessing the relationship between stress and AL.

AL = Allostatic Load; SBP = systolic blood pressure; DBP = diastolic blood pressure; HDL = high-density lipoprotein; LDL = low density lipoprotein; HbA1c = glycosylated hemoglobin; Epi = epinephrine; DHEA-S = Dehydroepiandrosterone; CRP = C-Reactive protein; IL = Interleukin; HSV-1 = Herpes simplex virus 1; IGF-1 = Insulin-like growth factor-1
Figure 2: Non-stress psychology variables and health practices variables significantly mediated ethnic differences in Allostatic Load (covariates age, gender and SES were controlled for in the model). Path values are unstandardized regression coefficients with SE in parentheses. The point estimates represent the mediated (indirect) effects derived from the bootstrapped product of paths a and b. Panel A illustrates the direct relationship between ethnicity and Allostatic Load. Panel B shows the indirect relationship between ethnicity and AL through non-stress psychological variables (Point Estimate of the Total Effects = -0.057; BCa = -0.137 - -0.014).

*p < 0.05, ** p < 0.01
Figure 3: Non-stress psychology variables and health practices variables significantly mediated ethnic differences in Allostatic Load (covariates age, gender and SES were controlled for in the model). Path values are unstandardized regression coefficients with SE in parentheses. The point estimates represent the mediated (indirect) effects derived from the bootstrapped product of paths a and b. Panel A illustrates the direct relationship between ethnicity and Allostatic Load. Panel B shows the indirect relationship between ethnicity and AL through health practice variables (Point Estimate of the Total Effects = -0.062; BCa = -0.0149 - -0.010).

*p < 0.05, ** p < 0.01
References


Centers for Disease Control and Prevention, National Center for Health Statistics, National Vital Statistics System, 57(14), 1-135.


The results of this dissertation, in part, have been submitted for publication of the material. Tomfohr, Lianne; Dimsdale, Joel. The dissertation author was the primary investigator and author of this material.