9-1-2013

Adverse Childhood Experiences and Acute Coronary Syndrome in Adulthood

Kanchana D. Wijesekera
Loma Linda University

LOMA LINDA UNIVERSITY
School of Science and Technology
in conjunction with the
Faculty of Graduate Studies

Adverse Childhood Experiences and Acute Coronary Syndrome in Adulthood

by

Kanchana D. Wijesekera

A Dissertation submitted in partial satisfaction of
the requirements for the degree
Doctor of Philosophy in Clinical Psychology

September 2013

Each person whose signature appears below certifies that this dissertation in his/her opinion is adequate, in scope and quality, as a dissertation for the degree Doctor of Philosophy.

_____, Chairperson
Kelly R. Morton, Associate Professor of Family Medicine and Psychology

_____
Jerry W. Lee, Professor of Health Promotion Education

_____
David A. Vermeersch, Director of Clinical Training, Associate Professor of Psychology

_____
Kendal C. Boyd, Assistant Professor of Psychology

ACKNOWLEDGEMENTS

I would like to express my deepest gratitude to Dr. Morton who provided unwavering mentorship throughout the graduate training years. She challenged me and guided me, both have which have helped me grow personally and professionally. I would also like to thank my committee members for their time, advice and direction.

To my family and friends, your love and support through this long endeavor has been incredible, and I know this journey would not have been impossible without your support. Special thanks to my parents and brothers for always being there for me and for providing me with endless love and support. And to my husband, who I met one month prior to embarking upon the graduate school journey, your patience, understanding, love, and continuous support has been truly heartfelt, and I could not have accomplished this endeavor without you.

CONTENTS

TABLES

Tables Page

FIGURES

Figures Page

ABBREVIATIONS

ACEs	Adverse Childhood Experiences
ACS	Acute Coronary Syndrome

ABSTRACT OF THE DISSERTATION

Adverse Childhood Experiences and Acute Coronary Syndrome in Adulthood

by

Kanchana D. Wijesekera

Doctor of Philosophy, Graduate Program in Biochemistry
Loma Linda University, June 2013
Dr. Kelly R. Morton, Chairperson

Research studies demonstrate that exposure to adverse childhood experiences (ACEs) lead to physical and mental health consequences in adulthood, including cardiovascular disease (Dong, Giles, Felitti et al, 2004; Felitti, Anda, Nordenberg, et al, 1998). One potential mechanism linking ACEs to cardiovascular disease is impaired social competence necessary for successful relationships and adequate social support. In the current study, the impact of ACEs on social support and subsequent incidence of acute coronary syndrome (ACS) was examined in a cohort of 6,596 older Seventh-day Adventists. After controlling for sociodemographic, lifestyle, and psychological factors, ACE exposure predicted increased risk of ACS by 31% for males only. Similarly, exposure to 3 or more ACEs resulted in a more than 3-fold increased risk of ACS for males compared to no ACEs.

While ACE exposure had positive associations with negative social support and negative spouse/partner support, there were no associations with social integration. Social integration and support measures did not mediate the ACE-ACS relationship; even in supplementary analyses combining cardiac-related mortality and ACS. However, several gender and poverty moderation effects emerged. Church attendance decreased the risk of ACS in females in poverty, after controls and ACE exposure. Among females

in poverty, negative social support and negative spouse support increased the risk of ACS incidence after controls and ACE exposure. Among males not in poverty, suppression effects were found, suggesting that having low negative support and negative spouse/partner support decreased the risk of ACS incidence as well as combined ACS and cardiac-mortality. Although non-significant, suppression patterns for poverty subgroups revealed that any increase in negative support or spouse/partner support, leads to increase in ACS incidence and combined ACS and cardiac mortality.

Together, the findings highlight the long-term impact of ACEs on cardiac health, and the importance of considering non-traditional risk factors (such social support and church attendance), as critical pathways that can lead to cardiac health outcomes. Further examination of religiousness and emotion regulation pathways should follow to further elucidate on the mechanisms that underlie the ACE-ACS relationship.

CHAPTER ONE

INTRODUCTION

According to the Center for Disease Control (CDC) and Prevention (2013), nearly 600,000 people die of heart disease in the United States; that is 1 in every 4 deaths. Heart disease is also the leading cause of death for both men and women; though more than half of the deaths due to heart disease in 2009 were in men. These statistics highlight the need to understand possible underlying psychosocial factors related to cardiovascular disease (CVD). Evidence clearly indicates that risk factors such as smoking, physical inactivity, diabetes, BMI (Wielgosz & Nolan, 2000), as well as psychosocial risk factors like depression, anxiety, hostility, and chronic stress, can impact the etiology and prognosis of CVD (Lett et al., 2004; Rozanski, et al, 1999). More specifically, exposure to adverse childhood experiences (ACEs) is known to be a critical risk factor for the development of CVD (Batten et al. 2004; Dong et al., 2004; Felliti et al., 1998). In fact, epidemiological evidence demonstrates that childhood abuse and household chaos are associated with significantly higher risk of CVD in adulthood (Batten et al., 2004; Dong et al., 2004, Felitti et al., 1998, Goodwin & Stein, 2004, Lehman et al., 2005). However, few studies examine the mechanisms linking ACEs and CVD. Deficient social support emerges as a potential mediator as children exposed to harsh and chaotic families commonly show deficits in social skills, thus compromising social support systems throughout life (Repetti et al., 2002). A social support deficiency, in turn, has been recognized as a

significant risk factor for CVD outcomes (Seeman & Syme, 1987; Rosengren, Wilhelmsen, & Orth-Gomer, 2004).

In light of these considerations, this review will examine evidence of (a) the link between ACEs and CVD in adulthood, (b) the role of ACEs in the impairment of social relationships, and (c) the evidence regarding a possible social support mediator between ACEs and acute coronary syndrome (ACS) incidence in adulthood.

CHAPTER TWO

LITERATURE REVIEW

Adverse Childhood Experiences

Research indicates a relationship between ACEs and unfavorable health outcomes in adulthood. Some studies have investigated the specific types of childhood adversity exposure including sexual abuse (Briere, & Runtz, 1993; Paolucci & Violato, 2001), physical abuse and neglect (Fuller-Thomson, Brennenstuhl, & Frank, 2010; Malinosky-Rummell & Hansen, 1993), emotional abuse (Briere & Runtz, 1988; O'Hagan, 1995), and witnessing family violence (Parkinson & Humphrey, 1988). Unfortunately, most maltreated children experience more than one adverse experience exposure (Felitti et al., 1998; Kinard, 1994). Conditions such as drug abuse, spousal violence, and criminal activity in the household may co-occur with other forms of domestic abuse. Felitti and colleagues (1998) note that, without measuring other types of abuse and dysfunctional household factors, the long-term influence might be wrongly attributed to single types of abuse exposures. In addition, examining the cumulative influence of multiple categories of adverse childhood experience exposures may be a more accurate representation of precursors to health outcomes in adulthood.

The ACE studies are large-scale epidemiological studies that assess the impact of various forms of childhood abuse and household dysfunction on a wide variety of health behaviors and outcomes from adolescence to adulthood (Felitti et al., 1998; Anda, Croft,

Felitti, et al, 1999; Dong, Dube, Felitti, et al., 2003). Because abuse and dysfunction are interrelated, the ACE researchers employ a cumulative stressor assessment to examine the relationship between the total number of childhood exposures (ACE score) and health outcomes (e.g., Felitti et al., 1998; Dong et al., 2004). In the ACE research in particular, seven primary categories of childhood experiences are aggregated: (a) psychological abuse, (b) physical abuse (c) sexual abuse; (d) witnessing violence against your mother; and (e) living with household members who were substance abusers, (f) mentally ill or suicidal, or (g) ever imprisoned (Felitti et al., 1998). Recently, two additional categories were added to the original ACEs: (h) physical neglect, and (i) parent separation/divorce (Dong et al., 2004). Multiple ACEs represent a disordered social environment and stress exposures during childhood that negatively impact emotional and social development, and subsequently lead to a myriad of health problems. As such, all nine categories will be used in the present study to assess a total ACE score ranging from 0-9 during the first 18 years of life.

ACEs and Cardiovascular Disease Outcomes

Using the seven item ACE measurement, 13,494 adults who had completed a standardized HMO medical evaluation were investigated (Felitti et al., 1998). History of self-reported ischemic heart disease (IHD) was collected as part of the medical evaluation (e.g., had heart attack, pain or heavy pressure in chest with exertion, use nitroglycerine). A strong, graded relationship between exposure to ACEs and risk for IHD in adulthood was found (Felitti et al., 1998). Those who reported 4 or more ACE exposures were 2.2 times more likely to have IHD than those with none. Similarly, Dong et al. (2004) also

found after controls, exposure to the nine item ACE measure also predicted IHD incidence. The likelihood of having IHD increased after any ACE exposure other than parental separation/divorce. Additionally, persons reporting 7 or more ACEs were more than three times as likely to report IHD than those with no ACEs and this was even stronger in those over age 50 (Dong et al., 2004). The link between ACEs and heart disease is further supported by Batten and colleagues (2004) who found both a 9-fold increase in CVD (i.e., hypertension, heart attack, stroke) in females and a lifetime increase in depression in both males and females after ACE exposure.

Furthermore, causal models indicate that childhood SES predicted ACEs which in turn predict psychosocial functioning (depression, hostility, social interactions) and subsequent metabolic syndrome. However, given the study's combined assessment of psychosocial functioning (affective states and social interactions), it is unclear to what extent social interactions played a role in the link between ACE exposure and adult metabolic syndrome. As such, the aspects of social interactions that are of most importance need further delineation based on the potential link between social support, ACEs and health.

Adverse Childhood Experiences and Adult Relationships

The ability to form social relationships with others is an essential component of mental health. Unfortunately, ACEs create deficits in the ability to interact effectively with others (Repetti et al., 2002; Taylor et al., 2004). Children's needs are compromised when parents are preoccupied with marital conflicts, addiction, and household chaos; becoming physically and/or emotionally unavailable to offspring (Amato & Booth, 1996;

Repetti et al., 2002). As such, exposure to ACEs eliminates opportunities for adequate emotional and interpersonal regulatory coaching by caregivers, thereby hindering children's ability to acquire skills necessary for successful interactions with others. For example, those exposed to ACEs have difficulty in identifying and labeling and managing emotions in general and in challenging circumstances (Repetti et al., 2002); as such, they overreact to stressful circumstances and respond aggressively (Dishion, 1990; Dunn & Brown, 1994). Repetti and colleagues (2002) assert that ACE related deficits lead to children who are unpopular and unsuccessful in attracting and maintaining social relationships and who are aggressive, socially withdrawn, as well as rejected and victimized by peers.

Among adults, those who have experienced ACEs have smaller support networks, perceive their relationships as less supportive, and experience decreased satisfaction with their support networks (Gibson & Hartshorne, 1996; Harmer, Sanderson, & Mertin, 1999; Stroud, 1999). Extensive research has focused on exposure to childhood sexual abuse and subsequent interpersonal struggles such as dissatisfaction with couple relationships, marital discord, generalized fear, mistrust, hostility and conflict with partners (DiLillo, 2001). Moreover, victims of sexual abuse tend to be isolated and report loneliness and less social support than the non-abused (Gibson & Hartshorne, 1996).

Vranceanu, Hobfoll, and Johnson (2007) examined the effect of multiple ACEs (i.e., sexual abuse and physical abuse, psychological maltreatment, neglect, and witnessing family violence) and social support among young adult women. ACEs were associated with decreased satisfaction and perceived availability of social support, which in turn was related to depression and posttraumatic stress disorder (PTSD) symptoms

(Vranceanu et al., 2007) as well as avoidance of intimacy (Klohnen & Bera, 1998). Additionally, adult survivors of child neglect also reported high levels of anxiety, paranoia and hostility, which are all detrimental for relationship formation and maintenance (Gauthier, Stollak, Messe, & Aronoff, 1996).

Together, these findings suggest that exposure to ACEs creates emotion regulatory and social skill deficits that challenge relationship maintenance and success. Early fearful schemas about significant others served a protective function early in life when family life was threatening. Maintaining these negative schemas about others in adulthood becomes maladaptive and results in less social integration and functional support. ACEs cause a repertoire of ineffective skills that ultimately create interpersonal struggles. By adulthood, these emotional and social struggles impair physiological reactivity to stressors to such a degree that chronic cardiovascular dysregulation results in heart disease (Robles & Kiecolt-Glaser, 2003). These studies however have not examined late life effects of ACE exposure; the present investigation will extend this work into later adulthood.

Disruption in the Physiological Regulation

Physiological dysregulation likely underlies the link between ACEs, social competence and health. Repetti et al. (2002) propose that children exposed to ACEs (i.e. risky families) have deficits in control and expression of emotions that impair social competence and disturb physiologic and neuroendocrine regulation (McEwen & Stellar, 1993). Specifically, ACEs produce chronic physiological activation, leading to wear and tear on organ systems like the cardiovascular system (Repetti et al., 2002). For example,

Luecken (1998) found college students who reported poor childhood family relationships had higher resting and task challenged blood pressures. Woodall and Matthews (1989) found that boys from less supportive families had stronger heart rate responses to laboratory stressors as well as more anger and hostility than those from more supportive families. Additionally, negative rather than positive family interactions are associated with abnormal cortisol response profiles, diminished immunity, and frequent illnesses (Flinn & England, 1997). Together, these studies emphasize ACE exposure leads to excessive cardiovascular reactivity to stressors (Smith & Ruiz, 2002) and an inability to recover quickly from these reactions (Robles & Kiecolt-Glaser, 2003).

Social Support and Cardiac Events

The term "social support" refers to both structural and functional support. Social integration refers to the structure of support (i.e. network size, contact frequency, group membership, marital status), while functional support involves subjective, perceived support quality from network members (i.e. emotional, informational, instrumental support, and companionship).

Impaired social support is recognized as a significant factor in cardiovascular disease outcomes (Hemingway & Marmot, 1999; Kuper, et al., 2002; Lett, et al., 2005). Within the domain of heart disease, most studies focus on the prognosis of individuals with *existing* CVD; both social integration and functional support significantly impact the progression of the disease and CVD-related mortality (see Lett et al., 2005; Barth et al., 2010 for reviews). However, given the paucity of studies and inconsistent results regarding heart disease incidence, the question remains whether lifelong social support

impairments after ACE exposure lead to CVD incidence. Both ACE and adult social support research indicates that negative social relationships heighten stress reactivity and lead to dysregulation of cardiovascular, endocrine, and immune functioning that makes one susceptible to heart diseases like acute coronary syndrome (Repetti et al., 2002).

Social Integration and Incidence of Cardiovascular Disease

Orth-Gomer, Rosengren, and Wilhelmsen (1993) analyzed the impact of social integration on new incidence of non-fatal acute myocardial infarction (AMI) and death attributed to coronary heart disease (CHD) in a 6-year follow-up in 736 Swedish men. CHD-related deaths were assessed by fatal MI, arteriosclerotic heart disease, and sudden coronary death via death certificates. Poor social integration was associated with an increased risk of an MI and death from CHD after controls (hyperlipidemia, overweight, hypertension, diabetes, and physical activity). Interestingly, the impact of social integration on CHD risk was of a similar magnitude as smoking. It is important to note that the number of new incident cases in the study was small (n=25). Rosengren, Wilhelmsen, and Orth-Gomer (2004) investigated the same cohort from Sweden in a 15-year follow-up of CHD morbidity (acute MI, coronary revascularization, and/or angina) or CHD-related mortality incidence in 96 men. Results once again found that, in comparison to those with high social integration, those with low social integration had the highest risk of new coronary events after controls (smoking, sedentary habits, systolic blood pressure, serum cholesterol and serum triglycerides, BMI, diabetes, and family history of coronary disease). In addition, a longitudinal investigation of 13,604 Swedish

participants revealed that a lack of social participation was predictive of first time AMI after controls (Ali, Merlo, Rosvall, Lithman, & Lindstrom, 2006).

In contrast, some studies have failed to demonstrate a relationship between social integration and CVD (Kawachi et al., 1996; Reed et al., 1983; Kaplan, 1988). For instance, Kawachi et al. (1996) examined 32,624 older, U.S., male, health professionals to investigate the link between social integration and incidence of stroke, MI, and CHD-related mortality (fatal MI, sudden cardiac death, and "other" deaths from CHD as determined by death certificates). Social integration was assessed via the Berkman-Syme social networks index (married, number of contacts, church member, community organization member). After controls, there was a dose-response gradient between social network and stroke incidence.

Functional Support and Cardiovascular Disease

Seeman and Syme (1987) provided one of the first direct comparisons of the importance of structural versus functional social support in relation to CVD in 30-70 years olds, with a diagnosis of angina pectoris, coronary artery disease, recent MI and/or asymptomatic coronary artery disease. The primary outcome was coronary atherosclerosis "total occlusion" score. After controls, instrumental and emotional support and not size of network were associated with atherosclerosis. Basic network size may not be the best proxy of social support as not all members of the network may provide positive support. Rosengren et al. (2004) also found that low emotional support increased the risk of coronary disease morbidity and mortality.

Others have demonstrated that functional support is not associated with CVD. Ikeda et al. (2008) assessed a Japanese cohort of 45,972 participants, aged 40-69 years association between functional social support and CVD morbidity and mortality incidence. Low emotional support and social isolation predicted a higher risk of mortality from stroke in men only; no associations were found between social support and incidence of stroke or MI.

While positive functional social support is not consistently related to CVD, negative functional social support is consistently deleterious to CVD outcomes. In fact, some argue that negative aspects of relationships influence health more than positive (Coyne & Bolger, 1990; Rook, 1984). For example, after controls, a dose-response association between excessive social network demands/worries and incidence of angina pectoris was found in middle aged Danes (Lund, Rod, Christensen, 2010). Additionally, angina pectoris incidence varied by intimacy such that excessive demands/worries from partner (OR= 3.53) and children (OR= 2.19) had stronger effects than demands from other family members (OR =1.91), friends (OR= 1.56), and neighbors (OR=1.86).

De Vogli, Chandola, and Marmot (2007) report the association between negative close relationships with CHD risk in the Whitehall II study. Although negative relationships were experienced more by women and those of lower SES, there were no gender or SES interactions with negative relationships predicting CHD (De Vogli et al., 2007). The authors posited that negative interactions in close relationships may be powerful predictors of cardiac risk due to heightened chronic physiological activation (De Vogli et al., 2007; Taylor, 1991).

Gender and Socio-Economic Status Differences

Lifetime risk of developing CHD after 40 is 49% for men and 32% for women (American Heart Association, 2011). Such disparities in the incidence of CVD emphasize the importance of considering gender differences.

Women tend to invest more time and energy in social relationships than men (De Vogli et al., 2007). In fact, women report higher psychological distress and negative social interactions, despite having more close relationships and giving and receiving more support than men (Fuhrer, Stansfeld, Chemali & Shipley, 1999). As such, increased interpersonal stress may effectively cancel out some of the benefits of social support for women (Shumaker & Hill 2001; Seeman, 1996). Moreover, when married couples were asked to discuss a problem in their relationship, women showed stronger stress responses than men (Kiecolt-Glaser et al., 1996).

Furthermore, evidence suggests that socio-economic status (SES) is an important factor in the etiology and progression of cardiovascular disease (Kaplan and Keil, 1993; Shaw et al., 2008). One of the classic studies of SES and coronary heart disease was the Whitehall Study (Rose & Marmot, 1981). Among 17, 530 civil servants aged 50-64 years, the lowest employment grade demonstrated a 53% higher prevalence of angina pectoris, 77% higher ischemic-type electrocardiogram abnormalities, and 75% higher prevalence of electrocardiographic abnormalities in comparison to the top (administrative) employment grade. At 10 year follow-up, coronary mortality rate was 3.6 times higher in the lowest grade in comparison to the top grade. Of note is also the study by Krause (2005) who found that greater criticism, demands, and being taken advantage of by network members, was associated with greater risk of heart disease in

low (but not high) income older adults. In light of these considerations, gender and SES differences are important to consider as they have important implications for understanding the link between ACE and ACS social support.

Covariates

Adjusting for confounding factors is an essential aspect of this investigation. In the ACE studies that investigated CVD outcomes (Felitti et al., 1998; Dong et al., 2003), only critical sociodemographic factors were controlled. However, in studies relating social support to CVD, many more health related covariates are controlled including smoking, alcohol use, exercise, BMI, and lifetime depression (Rosengren et al. 2003, Lund et al., 2005; De Vogli et al., 2002) and biological CVD risk factors such as hypertension (Orth-Gomer et al., 1996, Rosengren et al. 2003, Seeman & Syme, 1987, Lund et al., 2005; De Vogli et al., 2002) .

The focus of the current investigation is to predict acute coronary syndrome (ACS) incidence -- self-reported diagnosis of MI or medical procedures that prevented an MI after controls for sociodemographic factors (age, gender, ethnicity, education, difficulty meeting expenses in the past year) based on other ACE studies (Felitti et al, 1998; Dong et al., 2003), as well as lifestyle factors commonly controlled in social support and heart disease studies (i.e. BMI, smoking, alcohol use). In addition, given that negative emotionality and social skills/support are correlated, the current study controls for lifetime diagnosis of depression so that the impact of social support can be delineated more clearly.

Conclusion

Studies that focus on multiple exposures to adverse experiences highlight that childhood abuse, neglect, and household dysfunction are associated with adult CVD. Given the increased recognition for the co-occurrence of adverse experiences, the current study uses a cumulative stressor model to assess the relationships between the total number of ACE exposures, adult social support, and ACS incidence/survival. Social support may be one of the mediators responsible for the ACEs – ACS link; studies reveal social and emotion regulatory impairments among those exposed to ACEs, thereby hindering opportunities for acquiring effective support systems across the lifespan. Additionally, chronic negative relationships lead to physiological reactivity and cardiovascular diseases like ACS. The studies regarding the social support and CVD link have yielded mixed findings. In addition to the variability in covariates in the models tested, several other factors are also likely to play a role. Inconsistencies in the measurement of social support could provide one such explanation as some studies use measures of network size though they may not be used in positive ways; while others quantify functional support domains (i.e. emotional, instrumental, informational). Despite this variability, one central theme that emerges is the significant impact of negative rather than positive interactions on cardiac outcomes; this link is even more prominent within parental and spousal relationships. Evidence further suggests that chronic physiological activation (such as that which occurs with long term marital discord) may be one path that links negative interactions to CVD. For these reasons, the present investigation includes analyses to assess the impact of social integration as well as positive and negative interactions from network members. Additional analyses will

examine the impact of negative spousal/partner interactions on the link between ACE exposure and ACS incidence in those in such relationships.

The CVD definition across studies may also contribute to inconsistent findings; while some studies examine the impact of social support on MI alone, others use broader CVD definitions as an endpoint (i.e. MI, high blood pressure, and stroke). Other disease endpoints have included angina pectoris, atherosclerosis, ischemic heart disease, and coronary artery disease which are likely on a CVD severity continuum. For instance, while hypertension, angina pectoris, and atherosclerosis may represent the beginning stages of CVD, ischemic heart disease (characterized by reduced blood supply to the heart muscle) and MI (heart attack due to the blockage of an artery) are more severe.

With regards to disease outcomes, the current study recognizes the disadvantages of combining multiple endpoints (as in the case with CVD where high blood pressure, stroke, and MIs are grouped together). As such, the outcome of this investigation focuses on self-reported acute coronary syndrome (ACS); this not only captures self-reported diagnoses of MIs, but also medical procedures that prevent or stop an MI from occurring (such as the use of clot-busting medicine, balloons, or stents). Supplementary analyses will utilize combined ACS incidence and cardiac-related mortality to assess for potential differences in the outcomes.

Study Aims

The purpose of the study is to examine: 1) whether ACEs adversely impact the incidence of ACS via impaired social support, 2) whether effects of social support differ by gender and current poverty status, 3) the impact of ACE exposure and social support

on ACS incidence and cardiac-related mortality combined. Given these considerations, the following hypotheses were tested:

1) ACES will predict new incidence and survival of acute coronary syndrome (ACS) after controlling for sociodemographic factors (age, gender, ethnicity, education, and difficulty meeting expenses for basic needs in the last year), lifestyle factors (BMI, lifetime smoking, alcohol use), and psychological factors (lifetime depression diagnosis). These relationships will also predict ACS incidence and survival combined with cardiac-related mortality after 2006-7.
2) ACEs will be negatively related to social integration and positive social support, while positively related to negative social support and negative spouse/partner support, after adjusting for controls.
3) Social integration will mediate the relationship between ACEs and ACS incidence. Similar findings are expected when ACS incidence is combined with cardiac-related mortality.
4) Both positive and negative support from family and friends will mediate the relationship between ACEs and ACS incidence. Also, negative spouse/partner support will also mediate the relationship for participants who are currently in a relationship.
5) Current poverty will moderate the social support and ACS relationship: if in poverty, positive social support and social integration will reduce the likelihood of ACS incidence while negative social support and negative spouse/partner support (among those currently in a relationship) will increase ACS incidence. Similar

findings are expected when ACS incidence is combined with cardiac-related mortality.

CHAPTER THREE

METHOD

Data Source

The data for this study were gathered in the 2003-6 Adventist Health Study-2 (AHS-2), a cohort study of approximately 96,000 Seventh-day Adventists (SDA) investigating cancer, diet, and lifestyle (Butler et al., 2008) and the 2006-11 Biopsychosocial Religion and Health Study (BRHS; Lee et al., 2008). For the BRHS, in 2006-7 a total of 20,000 participants were randomly selected from the AHS-2 cohort and mailed a 20-page questionnaire on stress, religiousness, and health (BRHS-wave 1) as well as to eligible participants in 2010-11 (BRHS-wave 2); a total of 6508 returned complete questionnaires for AHS-2 and both waves of the BRHS and were over age 50. In addition, Bi-annual Hospitalization Questionnaires wave I (2004-7), wave II (2007-9), and wave III (2009-10) were sent to all participants to track serious health problems requiring hospitalization. BRHS-wave 2 questionnaires were only mailed to participants who were SDAs, Black or White, and with complete data on other questionaires.

Data pertaining to ACE exposures were obtained from BRHS-wave 1 (2006-2007) and BRHS-wave 2 (2010-11). Information regarding general demographics, social integration, and functional social support were obtained from BRHS-wave I (2006-7). Data regarding ACS (heart attack, or treatment to stop a heart attack such as clot-busting medicines or the balloon or a stent) were gathered from the Bi-annual Hospitalization Questionnaires wave I (2004-7), wave II (2007-9), wave III (2009-2010), and BRHS-

wave 2 (2010-2011). AHS-2 (2003-2006) provides exclusion for ACS self-reports so that only *new* ACS incidences, or those after the 2006-7 BRHS survey return date when social support was assessed are examined here. Additionally, the causes of death were obtained from the mortality data (via death certificates) for the AHS-2 participants. In particular, those who experienced a cardiac-related mortality were included in supplementary analyses to predict ACS incidence or cardiac related mortality; in these analyses only the 2006-2007 survey data were utilized so there is less selection bias regarding only those who survived CVD for these analyses (see Table 1).

Missing data on scales were handled as follows: all scores were means of the completed scale questions; one missing item was allowed for scales with three to five items, two for scales with six to 10 items, and three if there were more than 10 items. Also, scale outliers more than 4.5 standard deviations from the mean were identified and excluded (n=38). Similar to Dong et al. (2004), those who had incomplete data for ACEs or ACS were considered to not have had the adverse event.

Table 1

Data Sources by Survey and Year

Data Source	Survey date	Data	Exclusions
AHS-2	2003-2006	Diagnosis of ACS.	Anyone reporting ACS excluded (*n*= 71).
BRHS-wave 1	2006-2007	Demographic data; social integration; positive and negative functional support; spouse support and strain, ACEs.	Participants <50 (*n*=2488), not SDA (*n*=120), missing data on variables of interest (*n*=1238); outliers (*n*=38). Among participants who are couples, one spouse from the dyad is randomly deleted to avoid data dependence. These subjects are excluded only when assessing spouse support (*n*=191)
Death Certificates	2006-2007	Cardiac-related mortality data as a primary cause of death.	No exclusions.
Hospitalization Questionnaires– Waves I, II, III	2004-2010	New incidence of ACS.	Those who reported ACS incidence *before* BRHS-wave I return date were excluded (*n*=59).
BRHS- wave 2	2010-2011	Additional ACEs not included in BRHS-wave 1and ACS incidence	No exclusions.

Participants

The study population included only members of the SDA church, an evangelical protestant denomination that includes a health message for congregants regarding nutrition (50% are vegetarian), lifestyle (6% drink alcohol, 1% currently smoke) and

church involvement (0.3% report never attending worship). The inclusion criteria for the present study were SDAs who are 50 years or older. A total of 8,101 participants completed the data for ACEs, social support, and ACS across the questionnaires. Upon completion of the data screening process, the current study included 6,596 participants. The excluded participants in the study (n=4,408) were younger (59.2 vs. 65.5 years old), had less education (5.6 vs. 6.0 on a 9 point scale), and were exposed to more ACEs (1.41 vs. 1.13; all p<.001) in comparison to those who were included. Difficulty meeting expenses for basic needs in the past year did not differ by inclusion vs. exclusion groups (p<.05).

Descriptive statistics of the demographic and measured variables by ACS survival group are offered in Table 2. The ACS group were more likely to be White, male, less educated, older, and widowed than the nonACS group.

Table 2

Sample Demographics

	No ACS (n = 6352)		ACS (n = 244)		*p*-value
	N	%	N	%	
Ethnicity					
1. White	4267	67.2	203	83.2	<.001
2. Black	2085	32.8	41	16.8	
Gender					
1. Males	2141	33.7	128	52.5	<.001
2. Females	4211	66.8	116	47.5	
Highest Education					
1. Grade School	97	1.5	8	3.3	
2. Some High School	266	4.2	6	2.5	
3. High School Diploma	804	12.7	37	15.2	
4. Trade School Diploma	305	4.8	12	4.9	.001
5. Some College	1449	22.8	63	25.8	

Table 2

Sample Demographics (Continued)

	No ACS (n = 6352)		ACS (n = 244)		
	N	%	N	%	*p*-value
6. Associate Degree	688	10.8	21	8.6	
7. Bachelors Degree	1402	22.1	40	16.4	
8. Masters Degree	944	14.9	28	11.5	
9. Doctoral Degree	397	6.3	29	11.9	
Difficulty Meeting Expenses for Basic Needs in the Past Year					
1. Not at all	4688	73.8	180	73.8	
2. A little	902	14.2	32	13.1	
3. Somewhat	350	5.5	14	5.7	>.05
4. Fairly	247	3.9	10	4.1	
5. Very	165	2.6	8	3.3	
Marital Status					
1. Never married	285	4.5	3	1.3	
2. First marriage	3160	50.4	124	52.1	
3. Remarried	1350	21.5	53	22.3	
4. Common Law	15	0.2	0	0.0	.001
5. Divorced/Separated	767	13.6	16	7.5	
6. Widowed	613	9.8	40	16.8	
Number of ACE Exposures					
1. 0	3291	51.8	113	46.3	
2. 1	1629	25.6	63	25.8	
3. 2	741	11.7	32	13.1	
4. 3	325	5.1	18	7.4	
5. 4	183	2.9	11	4.5	
6. 5	109	1.7	6	2.5	>.05
7. 6	54	0.9	0	0.0	
8. 7	12	0.2	1	0.4	
9. 8	6	0.1	0	0.1	
10. 9	2	0.0	0	0.0	
	Mean	SD	Mean	SD	*p*-value
Age	65.41	10.35	72.01	9.33	.001
Education*	5.76	1.98	5.68	2.15	<.05
Difficulty meeting expenses year*	1.47	0.95	1.50	1.00	>.05
Number of ACE Exposures*	0.92	1.31	1.07	1.36	>.05

** See values above for categories.*

Measures

Covariates

The analyses adjusted for the following demographic factors: age, gender, ethnicity, education, and difficulty meeting expenses in the past year. Gender was coded as 1 – Female and 2 – Male. Ethnicity was coded as 1 = White (non-Hispanic White) and 2 = Black (African-American, Caribbean Black, Biracial). Additional covariates included education and difficulty meeting expenses for basic needs last year (see Table 1). For the moderation analyses in this study, difficulty meeting expenses for basic needs in the past year was dichotomized: the responses "not at all" and "a little" were coded as 0 (i.e. no current poverty), while the responses "somewhat," "fairly," and "very" were coded as 1 (i.e. experiencing poverty).

The following lifestyle factors were also used as controls in the study: BMI, lifetime smoking, and alcohol use (obtained from BRHS- 2006-7). Lifetime smoking was assessed by the item "Have you ever smoked regularly?" 0 – No smoking; smoking cigars, pipe, or cigarettes coded as 1- Yes. Alcohol use was assessed by the question: "Have you used alcoholic beverages, even if only occasionally?" The responses were coded as 1 – No, and 2 – Yes. Life time diagnosis of depression (obtained from BRHS-2006-7) was assessed with "Have you ever been diagnosed by a physician for… depression?" The response yes was coded as 1 and no as 2.

Acute Coronary Syndrome (ACS)

Self-reported data for ACS incidence was gathered longitudinally, across AHS-2 2003-2006 survey, the Bi-annual Hospitalization Questionnaires in 2004-2007, 2007-

2009, 2009-2010, and BRHS survey 2006-2007 and 2010-2011. A response of yes to the following items were used to indicate ACS: "Please fill circles to show which conditions/diseases you have ever had diagnosed by a physician." Heart attack (myocardial infarction) rated as < 5 years ago, 5-9 years ago, 10-14 years ago, 15-19 years ago, >20 years ago. Additional question asked whether during any hospitalizations in the previous 2 years, "…did you have a heart attack, or have treatment to stop a heart attack such as clot-busting medicines or the balloon or a stent?" Anyone who reported an ACS incidence before the BRHS 2006-2007 survey were excluded; anyone reporting ACS after BRHS 2006-2007 was included as a new incidence. This means we are studying participants who survived an ACS event and reported it. Excluded would be those who died from the ACS event, though supplementary analyses on initial mortality statistics are offered as well.

Cardiac-Related Mortality

Information pertaining to the causes of one's death was obtained from death certificates for AHS-2 participants across all states up to 2011. For the supplementary analyses, mortality due to ischemic events was included along with the survived ACS event as an outcome – this means some later surveys were not completed by the mortality group and there are some changes in the ACES scoring for these supplementary analyses. The causes of death ranged from hypertensive diseases, ischemic heart diseases, pulmonary heart diseases, to other forms of cardiac diseases/events.

Adverse Childhood Experiences (ACEs)

The ACE categories in the current investigation were based on Felitti et al. (1998) and Dong et al. (2005). All ACE item responses were dichotomized: a score of 0 is given if there was no exposure to an ACE category; a score of 1 if exposed to the ACE category (Felitti et al., 1998; Dong et al., 2005; Haviland, Morton, Oda, & Fraser, 2010). The 9 categories for ACEs included: (a) psychological abuse, (b) physical abuse, (c) sexual abuse, (d) neglect, (e) substance abuse in the household, (f) domestic violence in the household (i.e., mother treated violently), (g) imprisonment of a family member, (h) mental illness or suicide of a family member, as well as (i) parental divorce (details to follow). The total ACE score ranges from 0 to 9 reported on either BRHS questionnaire [wave-1 (2006-2007) or wave-2 (2010-2011)] as all events occurred when the adult was under age 18 years and were retrospective reports.

Psychological Abuse

Psychological abuse was assessed with 2 items from the Ryff, Singer, and Palmersheim (2004) child abuse scale rated on a 4-point scale (not at all to a lot): "Between ages 5-15 years did the mother/woman or father/man who raised you… "insult, swear at, or ignore you?" Similar to Felitti and colleagues (1998), a response of "not at all" or "a little" was coded as a 0, all else 1. An additional item assessed the frequency of being fearful of being hit by a father or adult, and was rated on a 5-point scale (seldom or never to very often). A response of "seldom or never" and "once in a while" were coded as a 0, all else 1. As such, a sum of 0 on the three items equaled 0 for psychological

abuse and any other sum equaled 1. Those with missing data were considered to not have experienced this ACE category, and subsequently coded as 0.

Physical Abuse

Physical abuse was assessed with four items from the Ryff, Singer, and Palmersheim (2004) child abuse scale rated on a 4-point scale (not at all to a lot): "Between ages 5 and 15, did the mother/woman or father/man who raised you… "push, slap, or throw objects at you?" and/or "kick, bite, or strike you with an object?" Similar to Felitti and colleagues (1998), a response of "not at all" or "a little" was coded as 0, all else 1. An additional item was rated on a 5-point scale (seldom/never to very often): "…a parent or other adult in your household hit you so hard that you had marks or were injured?" Responses of "seldom or never" and "once in a while" were coded as 0, all else as 1. As such, a total of 0 on the five items equaled 0 for physical abuse and any other sum equaled 1. Those with missing data were considered to not have experienced this ACE category, and subsequently coded as 0.

Child Sexual Abuse

Child sexual abuse was assessed via three items. First, Cusack, Frueh, and Brady's (2004) trauma scale item: "…ever have sexual contact with anyone who was at least 5 yrs older than you before you reached the age of 13?" was marked if it occurred and scored as 1; else 0. The other two items assessed forced sexual contact before age 8, and between ages 8 to 18 years. If either (or both) of these items were marked as having occurred it was scored as 1 for each; else 0. As such, a sum of 0 on the three items

equaled 0 for child sexual abuse and any other sum equaled 1. Those with missing data were considered to not have experienced this ACE category, and subsequently coded as 0.

Childhood Neglect

Neglect occurring between ages 5-15 years was assessed with a risky family scale item (Taylor, Lehman, Kiefe, & Seeman, 2006) and rated on a 5-point scale (seldom/never to very often): "How often would you say you were neglected while you were growing up, that is left on your own to fend for yourself?". The responses of "seldom or never," and "once in a while," and "occasionally" were coded as a 0, while "often" and "very often" were coded as 1 (Dong et al., 2004). Those with missing data were considered to not have experienced this ACE category, and subsequently coded as 0.

Household Substance Abuse

Exposure to an alcoholic or drug-abusing household member was assessed with the risky family scale item (Taylor, Lehman, Kiefe, & Seeman, 2006) and rated on a 5-point scale (seldom/never to very often): "In your childhood, did you live with anyone who was a problem drinker or alcoholic, or who used street drugs?" A response of "seldom or never" or "once in a while" were coded as 0, all else 1. Those with missing data were considered to not have experienced this ACE category, and subsequently coded as 0.

Mother Treated Violently

Exposure to one's mother being treated violently during childhood was assessed with four items: 1) The frequency of mother/stepmother being pushed, grabbed, or slapped; 2) The frequency of mother/stepmother being bitten or kicked; 3) The frequency of mother/stepmother being repeatedly hit for at least a few minutes; and, 4) frequency of mother/stepmother being threatened with a gun or knife. All items were rated on a 5-point scale (seldom/never to very often). Similar to Dong et al. (2004), for items 1 and 2, the responses "seldom or never" or "once in a while" were coded as 0, all else as 1. For items 3 and 4, only "seldom or never" was coded as 0, all else as 1. Given the greater severity of the violence indicated in items 3 and 4, a response of "once in a while" is contributes to the ACE score. As such, a sum of 0 on the 4 items equaled 0 for the mother violence category, and any other sum equaled 1. Those with missing data were considered to not have experienced this ACE category, and subsequently coded as 0.

Mental Illness of Household Members

Mental illness was assessed with the Felitti et al., (1998) items: "Was a household member depressed or mentally ill?" and "Did a household member attempt suicide?" Respondents who indicate these occurrences when "younger than 8" and/or "8-18 years" were coded as 1, while those who did not were coded as 0. As such, a sum of 0 on the two items equaled 0 for mental illness in the household, and any other sum equaled 1. Those with missing data were considered to not have experienced this ACE category, and subsequently coded as 0.

Imprisonment of Household Members

The imprisonment of a household member was assessed with a single item: "Did a household member go to prison?" Respondents who indicate this occurred when they were "younger than 8" or "8-18 years" was coded as 1, while those who did not were coded as 0. Those with missing data were considered to not have experienced this ACE category, and subsequently coded as 0.

Parental Divorce

The ACE category for parental divorce was assessed via the BRHS- wave 2 item: "Parents got divorced?" Respondents who indicate this occurred when they were "younger than 8" or between "8-18 years" were coded as 1, all else 0. Therefore, a total of 0 on the two items equaled 0 for parental divorce, all else equaled 1. Those with missing data were considered to not have experienced this ACE category, and subsequently coded as 0.

Social Support

Social support was assessed as 1) social integration (structure and quantity of social network), 2) positive or negative functional support received from family and friends, and 3) functional spouse support.

Social Integration

Social integration was assessed by 3 items: "About how many people are your close friends?" and "About how many people do you regularly socialize with?" These

two items were rated on an 8-point scale: None, 1 or 2, 3 or 4, 5 or 6, 7 or 8, 9 or 10, 11 to 15, and > 15. Given the importance placed on church involvement among SDAs, church attendance was examined as a major source of social participation for these participants. Church attendance was rated on a 6 point scale (never to > weekly) with this item: "How often do you attend church or religious meetings?" Each of the social integration items were assessed independently.

Positive Functional Support

Positive social support was assessed with the Newsom, Rook, Nishishiba, Sorkin, and Mahan (2005) 8-items regarding positive support received in the past month from spouse, family, friends, relatives, etc. These included informational support, instrumental support, emotional support, and companionship rated on a 5-point scale (never to very often).

Negative Functional Support

Negative social support was assessed with Newsom, Rook, Nishishiba, Sorkin, and Mahan (2005) 8-items regarding negative support received in the past month from spouse, family, friends, relatives, etc. These included unwanted advice or intrusion, failure to provide help, unsympathetic or insensitive behavior, and rejection or neglect. The 8 items were rated on a 5-point scale ranging from never to very often, and was summed together to create a single scale. The higher the score, the more negative functional support reported. Support from current spouse/partner was assessed with Ryff, Singer, and Palmersheim's (2004) 12-item negative spouse/partner scale, rated on a 4-

point scale (not at all to a lot). Of these 12 items, 6 were originally positive items; these items were re-coded and subsequently summed together with the 6 negative items. A higher score represents more negative support from partner/spouse if in a current intimate relationship.

Data Analyses

To examine the relationship between the variables of interests, partial correlations were examined between ACEs and social support variables, after adjusting for sociodemographic, lifestyle, and psychological factors. For all statistical significance, the alpha was set at 0.05.

To examine the ACS group differences for ACE exposure and negative spouse/partner support (for those currently in a relationship), two ANCOVAs were conducted. To examine the ACS group differences for social support (i.e. social integration and functional support), a MANCOVA was conducted.

Our dependent variable was self-report for new incidence of ACS (yes-no; and in supplementary analyses this included ischemic mortality as well), and our main independent variables were total ACEs, and the mediators were social integration (number of people close friends, number of people one socializes with, and frequency of church attendance), positive functional support, and negative functional support. An addition, for those currently in a relationship (married, remarried, or in a common law marriage), negative spouse/partner support was tested as a mediator. While gender differences were examined in the ACE-ACS analyses, moderation effects by both gender and current poverty status were examined when assessing the role of social integration

and support. In the models, the ordinal odds ratios (*OR*) were examined to evaluate the relationship between ACEs, social support, and the risk of ACS.

CHAPTER FOUR

RESULTS

ACE Exposure and ACS Incidence

Table 3 shows ACE exposure and ACS incidence in the study group. Nearly half the cohort experienced one or more ACEs, and 3.5% of the study group reported a new incidence of ACS during the study period. Among all subjects and for females, the most common ACE category was sexual abuse, followed by substance abusing family member, and psychological abuse. For males the most common was substance abusing household member, followed by sexual and psychological abuse. The least common ACE category was imprisonment of a household member. In addition, Chi-square analyses revealed gender differences in ACE exposures. In particular, sexual abuse, substance abusing household member, mental illness of a family member, mother beaten violently/domestic violence, and parental divorce were experienced more often by females than males.

Independent t-tests revealed that ACS incidence was more common among older (72.0 vs. 65.3 years; $p < .001$), less educated (5.65 vs. 5.78; $p < .05$) and White rather than Black participants (ACS = 4.5% vs. 1.9%; $p < .001$; ACS/cardiac mortality = 6.1% vs. 2.9%) and males rather than females (5.4% vs. 2.6%; $p < .001$). This ethnic difference indicates the Blacks who responded to our questionnaires differ from the U.S. population, making ethnic comparisons less valid as ACS typically occurs at a higher rate in Blacks than Whites.

Analysis of Covariance (ANCOVA) was conducted to compare ACE total by ACS groups, after controls. Among all participants, there were more ACEs in those that reported an ACS than those who did not for all participants (M_{ACS}= 1.07, SD= 1.36; $M_{no\text{-}ACS}$=0.92, *SD*=1.31; *p*=.001). Also, there were more ACEs reported in the ACS group than the nonACS group in males (M_{ACS}= 1.25, SD= 1.49; $M_{no\text{-}ACS}$=0.72, *SD*=1.15; *p*<.001) but not females.

Table 3

Frequency of ACE Exposure and ACS incidence

	All subjects % (N)	Males % (N)	Females % (N)	Chi-Square
ACE Exposure by Category				
Psychological Abuse	13.0 (872)	12.6 (290)	13.3 (582)	0.56
Physical Abuse	11.0 (732)	11.5 (264)	10.7(468)	1.08
Sexual Abuse	20.9 (1376)	11.3 (260)	25.8 (1116)	199.4**
Substance Abusing Household Member	19.2(1259)	16.5 (363)	20.6 (896)	17.2**
Mental Illness of a Family Member	5.3 (357)	4.2 (99)	5.8 (258)	7.63*
Mother Violently Beaten (Domestic Violence)	5.5 (367)	3.9 (90)	6.4 (277)	17.8**
Imprisonment of a Household Member	1..2 (79)	0.9 (21)	1.3 (58)	2.20
Childhood Neglect	10.0 (650)	9.4 (214)	10.3 (436)	1.50
Parent Divorce	6.0 (407)	4.9 (115)	6.5 (292)	6.82*
Total ACE Exposure				
0	51.8 (3404)	58.2 (1322)	48.5 (2082)	
1	25.5 (1692)	23.3 (521)	26.7 (1171)	
2	11.7 (773)	10.3 (237)	12.5 (536)	
3	5.3 (343)	5.7 (99)	4.4 (244)	
4	2.8 (194)	2.0 (47)	3.3 (147)	73.1**
5	1.7 (115)	1.3 (30)	2.0 (85)	
6	0.8 (54)	0.3 (8)	1.0 (46)	
7	0.2 (13)	0.1 (2)	0.3 (11)	
8	0.1 (6)	0.1 (3)	0.1 (3)	
9	<0.1 (2)	0 (0)	0.1 (2)	
New Incidence of ACS				
Yes	3.5 (244)	5.6 (128)	2.7 (116)	37.1**
No	96.5 (6352)	94.6 (2256)	97.4 (4475)	

*$p < .05$. **$p < .01$. ***$p < .001$.

Hypothesis 1: ACEs and ACS

Adjusted odds ratios (OR) and 95% confidence intervals were obtained from multivariate logistic regressions to test the significance of the graded relation between the total ACE score and the incidence of ACS. The socio-demographics (age, ethnicity, gender, education, and difficulty meeting expenses in the last year), lifestyle factors (BMI, lifetime smoking, and alcohol use), and psychological factor of lifetime depression were inserted in step 1 as controls. The total ACE score (0-9) was entered into the logistic models as an ordinal variable in step 2. The analyses were first conducted for all subjects, males and females. Gender and poverty status were explored as potential effect moderators.

Among all subjects, there was an ordinal *OR* of 1.17 (95% *CI*: 1.07, 1.29), indicating that every point increase in ACE exposure increases the likelihood of having a new incidence of ACS by 17% ($p = .001$), after controls. A subsequent analysis to assess an interaction effect with gender revealed significant findings as ACEs predicted the incidence of ACS for males, but not females ($p < .01$). For males, the ordinal *OR* was 1.31 (95% *CI*: 1.16-1.49), suggesting that every point increase in the ACE score increases the likelihood of having a new incidence of ACS by 31% ($p < .01$). Current poverty status did not yield a significant interaction effect with ACEs ($p > .05$).

Each ACE category was examined as an independent predictor of ACS incidence for all subjects, males, and females, after controls. Among all participants, psychological abuse (*OR* = 1.89, 95% *CI*: 1.35, 2.65, $p < .001$) and physical abuse (*OR* = 2.04, 95% *CI*: 1.44, 2.89, $p < .001$) independently predicted ACS incidence after controls. Gender analyses demonstrated that psychological abuse predicted ACS incidence independently

for males only (*OR* = 2.12, CI: 1.35, 3.32, *p* = .001), while physical abuse predicted ACS incidence for both males (*OR* = 1.96, 95% *CI*: 1.22, 3.13, *p* < .01) and females (*OR* = 2.17, 95% *CI*: 1.29, 3.67, *p* < .01). Additionally, sexual abuse (*OR* = 1.74, 95% *CI*: 1.03, 2.82; *p* < .05), mental illness of a family member (*OR* = 2.35, 95% *CI*: 1.50, 4.81; *p* < .05), and mother beaten violently (*OR* = 2.87, 95% *CI*: 1.12, 4.65; *p* < .05) significantly predicted ACS incidence for males only.

Table 4

Adjusted Odds Ratios for the Total ACE Score on ACS

	All Subjects		**Males**		**Females**	
	OR	95% CI	OR	95% CI	OR	95% CI
Step 1: Controls						
Age	1.06***	1.05, 1.09	1.06***	1.04, 1.08	1.06***	1.04, 1.08
Education	1.01	0.95, 1.08	1.05	0.96, 1.15	0.97	0.88, 1.08
Current Poverty	1.13	0.99, 1.29	0.88	0.68, 1.16	1.29**	1.10, 1.52
Black Ethnicity+	0.48***	0.35, 0.65	0.42***	0.25, 0.66	0.54**	0.36, 0.82
Male Gender++	1.95***	1.48, 2.55	--	--	--	--
BMI	1.05***	1.03, 1.08	1.07**	1.03, 1.12	1.04*	1.01. 1.08
Smoking	1.85***	1.28, 2.68	2.00**	1.19, 3.35	1.74*	1.00, 3.01
Alcohol usage	1.01	0.72, 1.44	1.15	0.70, 1.93	0.87	0.53, 1.41
Depression	0.94	0.64, 1.39	0.63	0.37, 1.10	1.29	.74, 2.26
Step 2: ACE Exposure						
Age	1.07***	1.05, 1.08	1.07***	1.05, 1.09	1.06***	1.04, 1.09
Educ.	1.01	0.95, 1.08	1.04	.95, 1.14	0.97	0.88, 1.08
Current Poverty	1.12	0.98, 1.28	0.87	.67, 1.13	1.29***	1.10, 1.52
Black Ethnicity+	0.49***	0.36, 0.66	0.42	.26, .68	0.55**	0.36, 0.82
Male Gender++	2.01***	1.53, 2.65	--	--	--	--
BMI	1.05***	1.03, 1.08	1.07**	1.02, 1.11	1.04**	1.01, 1.08
Smoking	1.79**	1.23, 1.60	1.81*	1.08, 3.06	1.74*	1.00, 3.00
Alcohol usage	0.96	0.68, 1.37	1.06	.63, 1.76	0.86	0.52, 1.40
Depression	1.00	0.68, 1.48	0.66	.38, 1.15	1.32	0.75, 2.31
Total ACE score	1.17***	1.06, 1.24	1.31***	1.16,1.49	1.04	0.90, 1.21

Table 4

Adjusted Odds Ratios for the Total ACE Score on ACS (Continued)

	All Subjects	
	OR	95% CI
Gender Moderation		
Age	1.07***	1.05, 1.08
Education	1.01	0.95, 1.08
Current Poverty	1.12	0.98, 1.29
Black Ethnicity+	0.48***	0.35, 0.66
Male Gender++	1.57*	1.12, 2.18
BMI	1.05**	1.03, 1.08
Smoking	1.74**	1.20, 2.53
Alcohol usage	0.97	0.68, 1.38
Depression	0.96	0.68, 1.42
Total ACE	0.80	0.59, 1.09
Gender* Total ACE	1.28**	1.07, 1.54

+ Referent is White
++ Referent is female
*$p < .05$. **$p < .01$. ***$p < .001$.

Given the relatively small sample sizes in the groups with ACE totals of 3-9, these categories were combined. The analysis was re-run with the following 4 ACE categories: (a) 0 ACEs, (b) 1 ACE, (c) 2 ACEs, and (d) 3 or more ACEs (see Table 5). Among all participants, an ordinal *OR* of 1.60 (95% *CI*: 1.06, 2.42) for those who reported 2 ACEs, and an *OR* of 2.05 (95% *CI*: 1.37, 3.09) for those who reported 3 or more ACEs. This suggests that in comparison to no ACEs, those who reported 2 ACEs had a 60% increased risk of ACS and those who reported >3 ACEs had a 205% increased risk of ACS ($p < .05$ and $p = .001$, respectively). Exposure to 1 ACE did not significantly increase the likelihood of having an ACS compared to no ACEs. Furthermore, gender interaction analyses revealed that this effect was significant for males only when exposed

to 3 or more ACEs. In particular, males had over a three-fold increased risk of ACS incidence after 3 or more ACEs (*OR* = 3.02; $p < .001$). There were no significant interaction effects for current poverty level ($p > .05$).

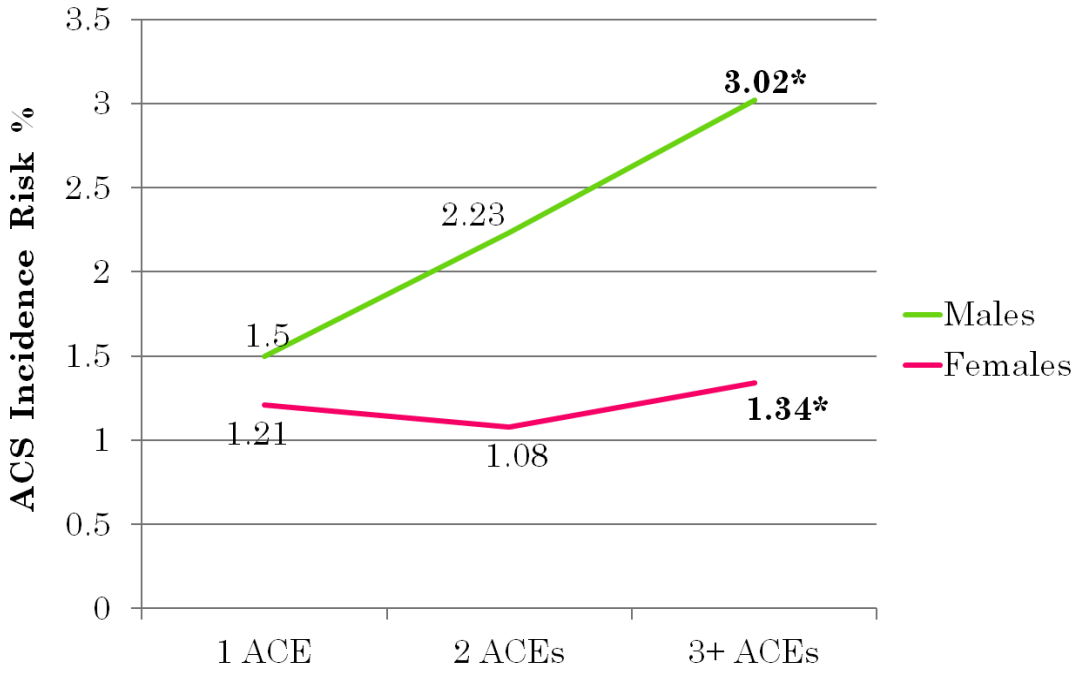

Figure 1. Exposure to 3+ ACEs increases the risk of ACS incidence for males.

Table 5

Adjusted Odds Ratios for ACEs on ACS

	All Subjects		**Males**		**Females**	
	OR	95% CI	OR	95% CI	OR	95% CI
Step 1: Controls						
Age	1.06***	1.05, 1.08	1.06***	1.04, 1.08	1.06***	1.04, 1.08
Education	1.01	0.95, 1.08	1.05	0.96, 1.15	0.97	0.87, 1.08
Current Poverty last yr	1.13	0.98, 1.29	0.88	0.68, 1.14	1.30**	1.09, 1.50
Black Ethnicity+	0.48***	0.35, 0.65	0.41***	0.26, 0.67	0.54*	0.36, 0.82
Male Gender++	1.94***	1.48, 2.57	--	--	--	--
BMI	1.05***	1.03, 1.08	1.07**	1.03, 1.11	1.04**	1.01, 1.08
Smoking	1.85**	1.28, 2.68	2.00**	1.19, 3.35	1.74	1.00, 3.01
Alcohol usage	1.01	0.71, 1.43	1.15	0.69, 1.90	0.86	0.53, 1.41
Depression	0.94	0.64, 1.39	0.63	0.37, 1.10	1.29	0.74, 2.26
Step 4: ACE Exposure (Categorical)						
Age	1.07***	1.05, 1.08	1.07***	1.05, 1.09	1.07***	1.05, 1.09
Education	1.01	0.95, 1.08	1.45	0.96, 1.15	0.97	0.88, 1.08
Current Poverty	1.12	0.98, 1.29	0.87	0.68, 1.14	1.30**	1.09, 1.50
Black Ethnicity+	0.48***	0.35, 0.65	0.42***	0.26, 0.67	0.55*	0.36, 0.82
Male Gender++	2.02***	1.53, 2.66	--	--	--	--
BMI	1.05***	1.03, 1.08	1.07**	1.03, 1.11	1.04**	1.01, 1.08
Smoking	1.79**	1.24, 2.60	1.86**	1.11, 3.12	1.74	1.00, 3.01
Alcohol usage	0.96	.67, 1.36	1.04	0.63, 1.73	0.85	0.52, 1.39
Depression	1.00	0.67, 1.49	0.70	0.40, 1.22	1.32	0.74, 2.31
0 ACE	(referent)		(referent)		(referent)	
1 ACE	1.36	.99, 1.89	1.50	0.94, 2.40	1.21	0.77, 1.89
2 ACEs	1.60*	1.06, 2.41	2.23**	1.28, 3.87	1.08	0.57, 2.02
3+ACEs	2.05***	1.37, 3.09	3.02***	1.74, 5.25	1.34	0.72, 2.51
Gender Moderation						
Age	1.07***	1.05, 1.08				
Education	1.01	0.95, 1.08				
Current Poverty	1.12	0.98, 1.28				
Black Ethnicity+	0.48***	0.36, 0.66				
Male Gender++	1.54*	1.05, 2.27				
BMI	1.05***	1.03, 1.08				
Smoking	1.77**	1.22, 2.56				
Alcohol usage	0.96	0.67, 1.36				
Depression	0.98	0.66, 1.45				

Table 5

Adjusted Odds Ration on ACS Incidence (Continued)

	All Subjects	
	OR	95% CI
0 ACE	(referent)	--
1 ACE	0.99	0.37, 2.69
2 ACEs	0.51	0.13, 1.95
3+ ACEs	0.52	0.14, 1.96
Gender* 0 ACE	(referent)	
Gender* 1 ACE	1.23	0.65, 2.32
Gender* 2 ACEs	2.12	0.93, 4.81
Gender* 3+ ACEs	2.45*	1.10, 5.48

+ Referent is White
++ Referent is female
*$p < .05$. **$p < .01$. ***$p < .001$.

Hypothesis 2: ACEs Predict Social Integration and Support

Bivariate correlations were first conducted to examine the relationship between ACEs and social integration and support. None of the social integration variables (number of close friends, number of people one socializes with, and frequency of church attendance) were associated with ACE exposure after controls ($p > .05$). While positive functional support was also not correlated with ACE exposure, negative functional support was positively correlated with ACE exposure ($r = .12$, $p < .001$). In addition, among those who are currently in a relationship, negative spouse/partner support was positively associated with ACE exposure ($r = .09$, $p < .001$). Together, these results suggest that the greater the ACE exposure, the more negative social support and negative spouse/partner support in adulthood.

Given the association between ACEs and negative support variables, two regression models were tested to determine whether negative support and negative spouse/partner support could be predicted by ACEs exposure after controls. The results suggest that a proportion ($R^2 = .09$) of the total variation in negative social support was predicted by the controls and ACEs combined, F (10, 6562) = 71.96, $p < .001$. Furthermore, after controls, ACEs alone predicted negative social support (R^2 change = .01, $p < .001$).

Similarly, the second multiple regression analysis revealed that among those currently in a relationship, a proportion ($R^2 = .08$) of the total variation in negative spouse/partner support was predicted by the controls and ACEs combined, F (10, 4385) = 44.22, $p < .001$. After controls, ACEs independently predicted negative spouse/partner support (R^2 change = .01, $p < .001$).

Hypothesis 3: Mediation Test of Social Integration

A MANCOVA was performed to compare mean scores on social integration (number of close friends, number of people one socializes with, and frequency of church attendance) and functional support (positive and negative social support) between the ACS and non-ACS groups, after adjusting for the controls. Only church attendance was lower in the ACS group compared to the non-ACS group for all subjects ($_{ACS}M = 5.18$, $_{No\text{-}ACS}M = 5.32$ respectively, $p<.05$) and for females ($_{ACS}M = 5.12$, $_{No\text{-}ACS}M = 5.31$ respectively, $p<.05$). Although the gender interaction (church attendance X gender) was non-significant – a subgroup analysis indicated that in females, church attendance protected against ACS incidence after controls and ACEs ($OR = .80$; $p < .05$). For every

unit increase in church attendance, the incidence of ACS decreased by 20% for females (see Table 6). Although poverty interaction term (church attendance X poverty) and the 3-way interaction term (church attendance X poverty X gender) were non-significant, the post-hoc subgroup analyses: males in poverty, males not in poverty, females in poverty, and females not in poverty showed that for females in poverty, church attendance protected against ACS incidence after controls and ACEs (p = .05). For every unit increase in church attendance, the incidence of ACS decreased by 32% for females in poverty. No significant findings were found for the other subgroups.

Table 6

Adjusted Odds Ratios for Social Integration on ACS

	All Subjects		**Males**		**Females**	
	OR	95% CI	OR	95% CI	OR	95% CI
Step 1: Controls (Details not shown)						
Step 2: ACE Exposure						
Age	1.06***	1.05, 1.08	1.06***	1.04, 1.08	1.06***	1.04, 1.08
Education.	1.01	0.95, 1.08	1.05	0.96, 1.15	0.97	0.88, 1.08
Current Poverty	1.13	0.99, 1.29	0.88	0.69, 1.16	1.29**	1.10, 1.52
Black Ethnicity+	0.48***	0.35, 0.65	0.42***	0.25, 0.66	0.54**	0.37, 0.83
Male Gender++	1.95***	1.48, 2.55	--	--	--	--
BMI	1.05***	1.03, 1.08	1.07**	1.03, 1.12	1.04**	1.01, 1.08
Smoking	1.85**	1.28, 2.68	2.00**	1.19, 3.35	1.74	1.01, 3.01
Alcohol usage	1.01	0.72, 1.44	1.15	0.69, 1.90	0.87	0.53, 1.42
Depression	0.94	0.64, 1.39	0.63	0.37, 1.10	0.63	0.37, 1.10
ACE Exposure	1.17***	1.07, 1.29	1.31***	1.16, 1.49	1.04	0.90, 1.21
Step 3: Social Integration						
Age	1.06***	1.05, 1.08	1.06***	1.04, 1.08	1.06***	1.04, 1.08
Education	1.01	0.95, 1.08	1.05	0.96, 1.15	0.97	0.88, 1.08
Current Poverty	1.12	0.99, 1.29	0.87	0.67, 1.14	1.28**	1.09, 1.52
Black Ethnicity+	0.49***	0.36, 0.65	0.43***	0.26, 0.69	0.57**	0.37, 0.83
Male Gender++	1.95***	1.48, 2.55	--	--	--	--
BMI	1.05***	1.03, 1.08	1.07**	1.03, 1.11	1.04**	1.01, 1.08
Smoking	1.88**	1.28, 2.68	2.01**	1.19, 3.38	1.75	1.01, 3.04
Alcohol usage	1.00	0.70, 1.44	1.15	0.69, 1.91	0.85	0.52, 1.38
Depression	0.97	0.65, 1.44	0.64	0.37, 1.12	1.35	0.77, 2.36
ACE Exposure	1.17***	1.07, 1.29	1.31***	1.16, 1.49	1.04	0.89, 1.21
N people socialize with	1.04	0.96, 1.11	0.98	0.87, 1.09	1.01	0.99, 1.22
N close friends	1.00	0.92, 1.09	1.06	0.94, 1.18	0.95	0.83, 1.08
Church attendance	0.86	0.73, 1.00	0.93	0.75, 1.17	0.80*	0.64, 0.97

+ Referent is White

++ Referent is female

*$p < .05$. **$p < .01$. ***$p < .001$.

The procedure proposed by Baron and Kenny (1986) was utilized to test for mediation effects of social integration. Accordingly, the significant association between the ACE exposure (the independent variable) and ACS incidence (dependent variable) was first established. Then, for complete mediation, three conditions need to be met: (a) The ACE exposure must be significantly associated with the social integration variable, (b) social integration must be significantly associated with ACS incidence, and (c) the relationship between ACE exposure and the ACS must become non-significant (or significantly reduced for partial mediation) when social integration is controlled.

Accordingly, none of the social integration measures meet the above criteria. Although church attendance in females predicted ACS incidence, it could not be tested as a mediator given the non-significant association between ACEs and church attendance [i.e. condition (a) was not met].

Hypothesis 4: Mediation Test of Functional Social Support

The same procedure to test mediation of functional social support was used (positive social support, negative social support, and negative spouse/partner support). According to the previous linear regression analyses, Condition (a) was met for two of the variables as ACEs predicted negative social support and negative spouse/partner support for all participants. Condition (b) was not met for any of these variables for all participants or by gender, but did so for poverty status. In particular, current poverty emerged as a moderator as the interaction terms of poverty X negative social support as well as poverty X negative spouse/partner support both predicted ACS incidence (*p*s < .01). Post-hoc analyses revealed that, among those in poverty, negative social support

from family/friends increased the risk of ACS incidence by 78% (*OR* = 1.78, $p < .05$). Similarly, for those in poverty, negative spouse/partner support increased ACS incidence by 9% (*OR* = 1.09; $p < .05$), after controls.

Given the poverty moderation, binomial regressions were conducted to more closely examine the mediations across 4 subgroups (gender x poverty). Controls were inserted into block 1, ACEs into block 2, and negative social support into block 3 (see Table 7). Findings revealed that ACEs predicted ACS incidence after controls only for males not in poverty (*B* = 0.27, *OR* = 1.31, $p < .001$). When the negative social support (mediator) was entered in Block 3, the coefficient (i.e. standardized beta weight) for the ACEs increased, and remained significant (*B* = 0.29, *OR* = 1.34, $p < .001$), thereby suggesting a suppressor effect. As described by MacKinnon, Krull, and Lockwood (2000), "…a situation in which the magnitude of the relationship between an independent variable and a dependent variable becomes larger when a third variable is included would indicate suppression." Findings in the other subgroups were not significant though demonstrated a similar pattern. Furthermore a noteworthy finding is that among females in poverty, negative social support predicted ACS incidence after controls and ACE exposure (*OR* = 1.94, $p < .05$). For every increase in negative social support, the risk of ACS increased by 94% for females in poverty. Given these findings, a 3-way interaction term was tested (negative social support X poverty X gender), it was not - significant ($p > .05$).

Given that negative spouse/partner support predicted ACS incidence after controls and ACEs (*OR* = 1.09; $p < .05$), mediation effects were examined across the 4 subgroups among those who are currently in a relationship (see Table 8). Results demonstrated that

ACEs predicted ACS incidence after controls only for males not in poverty (B = 0.27, OR = 1.31, $p < .001$). When the negative spouse/partner support (mediator) was entered in Block 3, the coefficient for the ACEs increased, and remained significant (B = 0.28, OR = 1.32, $p < .001$), once again demonstrating a suppression effect. In addition, a similar pattern of suppression was observed across all other subgroups, although findings did not reach statistical significance (ps > .05). Furthermore, among females in poverty, negative spouse/partner support predicted ACS incidence after controls and ACEs (OR = 1.10, $p <$ .05). For every unit increase negative spouse/partner support, the risk of ACS increased by 10% for females in poverty. Given these findings, a 3-way interaction term was tested (negative spouse/partner support X poverty X gender), and it yielded non-significant results (p > .05).

Table 7

Adjusted Odds Ratios for Negative Social Support on ACS by Gender and Poverty Status

	Males in Poverty (n = 198)		**Males Not in Poverty** (n = 2069)		**Females in Poverty** (n = 594)		**Females Not in Poverty** (n = 3712)	
	B	*OR*	*B*	*OR*	*B*	*OR*	*B*	*OR*
Step 1: Controls (Details not shown)								
Step 2: ACE Exposure								
Age	0.11	1.11**	0.06	1.07	0.03	1.03	0.69	1.07***
Education	-0.01	0.99	0.07	1.07	-0.06	0.94	-0.02	0.98
Black Ethnicity+	-0.33	0.72	-0.89	0.41*	0.24	1.28	-0.93	0.40**
BMI	0.15	1.17*	0.06	1.06*	0.04	1.04	0.45	1.05*
Smoking	-0.52	0.60	0.71	2.02	0.16	1.18	0.68	1.97*
Alcohol usage	-0.38	0.96	0.08	1.09	-0.16	0.86	0.13	0.88
Depression	0.36	1.44	-0.40	0.67	-0.19	0.82	0.46	1.58
ACE Exposure	0.33	1.39	0.27	1.31***	-0.15	0.89	0.08	1.09
Step 3: Negative Social Support								
Age	0.12	1.13**	0.06	1.07***	0.04	1.04	0.07	1.07
Education	-0.01	0.99**	0.66	1.07	-0.08	0.92	-0.02	0.98
Black Ethnicity+	-0.58	0.56	-0.89	0.41**	0.28	1.32	-0.92	0.40
BMI	0.15	1.16	0.06	1.07**	0.04	1.04	0.05	1.05
Smoking	-0.70	0.50	0.71	2.04**	0.18	1.20	0.69	1.99
Alcohol usage	-0.13	1.14	0.06	1.06	-0.15	0.86	0.12	0.88
Depression	0.53	0.65	-0.45	0.64	-0.07	0.94	0.42	1.53
ACE Exposure	0.36	1.43	0.29	1.34***	-0.19	0.85	0.09	1.09
Negative Social Support	0.62	1.86	-0.34	0.71	0.66	1.94*	-0.18	0.83

+ Referent is White
++ Referent is female
*p<.05. **p<.01. ***p<.001.

Table 8

Adjusted Odds Ratios for Negative Spouse/Partner Support on ACS by Gender & Poverty Status

	Males in Poverty (n=142)		**Males Not in Poverty** (n=1732)		**Females in Poverty** (n=264)		**Females Not in Poverty** (n=3358)	
	B	*OR*	*B*	*OR*	*B*	*OR*	*B*	*OR*
Step 1: Controls (Details not shown)								
Step 2: ACE Exposure								
Age	0.11	1.12**	0.63	1.07***	0.03	1.03	0.08	1.08
Education.	-0.12	0.88	0.05	1.06	0.02	1.02	0.02	1.02
Black Ethnicity+	0.16	1.17	-0.95	0.39**	1.20	3.31	-1.12	0.33
BMI	0.13	1.14	0.06	1.06*	0.08	1.08	0.07	1.07
Smoking	0.50	1.66	0.69	2.00*	0.95	2.58	0.82	2.28
Alcohol usage	-1.10	0.33	0.17	1.18	-0.21	0.81	-0.40	0.67
Depression	-0.30	0.74	-0.62	0.54*	-1.31	0.27	0.81	2.25
ACE Exposure	0.28	1.32	0.27	1.31***	-0.24	0.79	0.17	1.18
Step 3: Negative Spouse/Partner Support								
Age	0.12	1.13*	0.06	1.06**	0.04	1.04	0.07	1.08***
Education	-0.13	0.88	0.06	1.06	-0.05	0.95	0.02	1.02
Black Ethnicity+	0.03	1.03*	-0.91	0.40**	0.97	2.63	-1.07	0.34*
BMI	0.14	1.15*	0.06	1.06*	0.09	1.10	0.07	1.08**
Smoking	0.31	1.36*	0.71	2.04*	0.89	2.44	0.89	2.44
Alcohol usage	-1.10	0.33	0.17	1.19	-0.12	0.89	-0.39	0.67
Depression	-0.11	0.90	-0.69	0.50	-1.23	0..29	0.73	2.08
ACE Exposure	0.29	1.34	0.28	1.32***	-0.33	0.72	0.18	1.20
Negative Spouse/Partner Support	.008	1.08	-0.03	0.97	0.09	1.10*	-0.05	0.96

+ Referent is White
++ Referent is female
*p<.05. **p<.01. ***p<.001

Supplementary Analyses: ACS Morbidity and CVD Mortality Combined

Given that the literature has demonstrated an impact of poor social support on the increased risk of death related to CVD (Orth-Gomer et al., 1993; Rosengren et al., 2004), this study combined new incidence of ACS incidence/cardiac-related mortality (obtained from the mortality data of BRHS 2006-2007 participants'' death certificates) to examine any changes in the findings noted above. These analyses included a total 332 participants who had either ACS (n = 244) or cardiac-related mortality (n = 94); 6 participants met criteria for both. To examine combined ACS and cardiac-mortality outcome, only ACEs from AHS-2 and BRHS-2006-2007 were included, which resulted in the inclusion of 6 of the previous 9 ACE categories: psychological abuse, physical abuse, sexual abuse, substance abusing family member, childhood neglect, and parent divorce.

The findings were similar to those of ACS incidence alone. There was a significant gender interaction as ACE exposure increased ACS incidence/cardiac-related mortality for males by 22% (*OR*: 1.22, 95% *CI*: 1.06, 1.40; p =.006), but not females. This is a reduction from the previously reported 31% increased risk of ACS incidence found in males. Among the social integration items, church attendance was found to significantly decrease the risk of ACS incidence/cardiac-related mortality for all subjects (*OR* = 0.85, 95% *CI*: 0.74, 0.97; $p < .05$). Although the gender interaction (gender X church attendance) was not significant, church attendance significantly decreased ACS incidence/cardiac mortality in females by 20% (*OR* = 0.80, 95% *CI*: 0.66, 0.96; $p < .05$); the same as the ACS incidence analysis. No significant findings were found by poverty status, or the 4 gender-poverty subgroups. This differs from the previous findings which

demonstrated that church attendance protected against ACS incidence for females in poverty.

In addition, regressions revealed that both negative social support and negative spouse/partner support were predicted by the modified ACEs after controls [F (10, 6562) = 67.5, $p < .001$ and F (10, 4385) = 43.3, $p < .001$ respectively), thus, these functional support measures were examined as mediators. The tests of mediation were conducted across the 4 poverty x gender subgroups previously noted. Similarly, ACEs predicted ACS incidence/cardiac mortality after controls only for males who were not in poverty (B = 0.20, OR = 1.22, $p < .01$). When negative social support was entered, the coefficient for the modified ACEs increased, and remained significant (B = 0.22, OR = 1.24, $p <$ .01), suggesting a suppression effect. Although non-significant, this pattern of suppression was evident in all subgroups. Furthermore, negative social support did not yield an increased risk of ACS incidence and cardiac mortality for women in poverty, after adjusting for controls and ACEs. This is in contrast to the 94% increased risk of ACS incidence previously found among women in poverty. Given these findings, a 3-way interaction term was tested (negative spouse/partner support X poverty X gender), and it yielded non-significant results ($p > .05$).

The findings relating to negative spouse/partner support were similar to those found earlier with ACS incidence. Among males who are not in poverty, the addition of negative spouse/partner support resulted in a slight increase in the ACEs coefficient (B = -0.21, OR = 1.23, $p < .01$); thereby suggesting a suppression effect. Although not statistically significant, this pattern was seen across all poverty x gender subgroups. In addition, among females in poverty, negative spouse/partner support increased the risk of

ACS incidence/cardiac mortality by 12% after controls and ACEs (*OR* = 1.12, $p < .05$); this is a slight increase from the 11% risk of ACS incidence previously reported for women. Given these findings, a 3-way interaction term was tested (negative spouse/partner support X poverty X gender), and it yielded non-significant results ($p > .05$).

CHAPTER FIVE

DISCUSSION

Summary of Findings

The study revealed that exposure to ACEs had a significant impact on ACS incidence/cardiac mortality in mid to late adulthood after adjusting for sociodemographic, lifestyle, and psychological risk factors. Although women had significantly more ACEs than men, men experienced higher ACS incidence. Thus, men may have fewer coping strategies than women after ACE exposures. As such, in males, psychological abuse, physical abuse, sexual abuse, mental illness of a family member, and mother beaten violently independently predicted ACS incidence. Only physical abuse significantly predicted ACS incidence in females. As in other studies (Batten et al., 2004, Felitti et al., 1998; Dong et al., 2004) the cumulative ACE exposures demonstrated a dose-dependent relationship with ACS incidence: the more childhood adversities experienced, the greater the ACS risk.

Our ACE and ACS relationship is consistent with the dose-dependent association after controls reported by Felitti et al. (1998) and Dong et al. (2004). Our additional gender analyses did reveal, however, a much stronger ACE and ACS relationship in men than in women. In particular, having experienced three or more adverse childhood experiences resulted in a more than 2-fold increased risk of ACS in all subjects and over 3-fold increase in males. The risk for males seen in our study is higher than the 2-fold risk demonstrated in the Felitti et al. (1998) study among all participants.

We concur that multiple ACEs likely represent a disordered social environment and chronic stress exposures during a critical period of development that irrevocably

change stress reactivity and ultimately impact health via this impairment. Our finding that men had higher ACE-ACS incidence differs from Korkeila et al. (2010) who report an increased risk of cardiovascular disease among women after ACE exposure than men in a prospective cohort study. However, Korkeila et al. examined only young and middle aged adults whereas we examined middle aged and late life adults when there is a higher prevalence of CVD. Additionally, Korkeila et al. only assessed long term financial difficulties, divorce/separation of parents, serious conflicts in the family, severe illness of a family member, frequent fear of a family member, or alcohol problem of a family member and not severe, early life adversities including physical and sexual abuse as well as neglect, domestic violence and criminal activity and substance abuse in the household. Furthermore, gender differences were assessed by dichotomizing ACE exposure: exposed to ACEs and not exposed to ACEs, whereas our study examined ACE exposure on a continuum. In addition, only age appears to have been utilized as a control, while our study included a broader range of sociodemographic factors, as well as lifestyle and psychological factors that are consistent with the literature thus ruling out many other known risk factors for ACS.

In the current sample, men were more vulnerable to the long-term impact on ACE exposure on ACS incidence; women though exposed to more ACEs were somehow protected from ACS incidence, either because of psychological buffers or because ACS incidence happens at later ages in females than males. In addition, church attendance had a protective effect against ACS incidence in females in poverty. These SDA women did have significant church involvement and other studies have shown that religious activity does improve longevity (Hummer, Ellison, Rogers, Moulton, & Romero, 2004; Maselko,

Kubzansky, Kawachi, Seeman, & Berkman, 2007; Pargament, Koenig, Tarakeshwar, & Hahn, 2004) due to social support, better coping or healthier lifestyle (less smoking, alcohol use. Of note, our study controlled for significant cardiovascular risk factors (such as smoking, alcohol use, BMI, depression), and still demonstrated protective effects of church attendance above and beyond these significant risk factors. Furthermore, because both males and females in this sample attended church at the same rate, the reason for the protective effect for women and not men requires further study. It may be a social support effect or it may be another aspect of religiousness that co-occurs with church attendance. Also, women with financial struggles may be turning to worship and religious faith as cost-effective coping mechanisms to overcome hardships, whereas higher incomes may lead to other ACS protections (i.e. access to healthcare, afford purchase of healthier food, safer neighborhoods for exercise). Such possibilities warrant further exploration.

ACE exposure was associated with more negative social support from family and friends, as well as from partners/spouses, yet neither of these negative social supports mediated the relationship between ACEs and ACS incidence when assessed across the 4 gender-poverty status subgroups. Instead, negative social support and negative spouse/partner support demonstrated statistically significant suppression effects for males not in poverty. This suppression effect pattern was observed across all subgroups – the inclusion of negative social support resulted in an increased magnitude of the effect of ACEs on ACS incidence. Although further exploration is needed, our findings suggest that negative social support explains variability in the impact of ACE exposure, such that having less negative social support is protective from ACS in males with financial

resources. Also, the general pattern further suggests that for those in poverty, any level of negative social support leads to an increase one's risk for ACS incidence.

Our results are also consistent with Krause (2005), which demonstrated increased risk of cardiac events for those in low-SES. However, our findings extend Krause's results in two ways. First, we found negative support remained significant after adjusting for a comprehensive list of known risk factors and ACE exposure. Second, although it did not reach statistical significance, our study demonstrated a pattern of decreased ACS risk with less negative social interactions for those not in poverty. However, for individuals who are financially struggling to meet basic needs, any amount of negative support from family, friends, partners/spouses increases their ACS risk.

Contrary to our hypotheses, ACE exposure was not associated with positive social support or social integration after adjusting for controls. We adjusted for more controls than most ACE studies because we wanted our controls to be similar to those of other cardiovascular disease studies. As such, it may be that controlling for lifestyle and psychological factors decreased the relationship between social integration, positive social support, and ACEs. In addition, positive social support as well as number of close friends and number of people one socializes with, did not demonstrate a protective effect on ACS incidence. However, these findings are consistent with the literature that demonstrated that negative interactions have a stronger impact on negative health outcomes than positive interactions.

The supplementary analyses that combined cardiac-related mortality data with ACS incidence demonstrated similar findings to those found with survived ACS incidence alone, with a few exceptions. In particular, the 6-item short version of ACE

exposure predicted ACS incidence/mortality outcome for males. Of note, the more extreme ACE exposures were omitted as they were only measured in 2010, and those who died between 2007–2010 could not complete this survey. Given that imprisonment of family member, mental illness of family member, and mother beaten violently were not included in the ACE score, the findings are likely to be an underestimate of the ACE effects. Furthermore, church attendance independently predicted ACS incidence/mortality for females after adjusting for controls and ACE exposure, thereby highlighting the increased ability for females to obtain support via church attendance.

Similar to ACS findings, the ACS/cardiac-mortality results showed that if you are male with financial resources, less negative support is protective. A pattern of suppression effects was also observed across all subgroups, and further suggests that for those currently living in poverty, any negative social support increased the risk of ACS incidence/cardiac mortality. This emphasizes the idea that those who are not currently facing financial strain may have other additional resources to cope with stress reactivity that buffers ACE exposure.

Study Strengths

While previous studies have revealed associations for an ACEs-social competence deficit link, and for poor social support-cardiovascular disease link, this study examined all three relationships. Given the strong associations found between ACEs and ACS incidence, it is particularly important to examine and better understand what mechanisms may be contributing to this relationship; as such, the current study added to the literature that social integration and functional support do not mediate the ACE-ACS link in the

traditional way. Instead, the suppression effects suggested that among those with financial resources, having less negative social support and spouse/partner support protected from ACS incidence, even after accounting for social-demographics, traditional cardiovascular risk factors, and ACE exposure. This finding highlights the importance of considering non-traditional risk factors for poor cardiac health.

Additionally, the current study generated consistent cumulative effects of ACE exposure on health outcomes in adulthood. In fact, one of the clear strengths of this study is the large numbers of older adults to test the impact of ACEs on ACS. These data provide strong evidence of the longstanding nature of adverse childhood events over time on cardiac health. Furthermore, having a sample large enough to conduct gender and poverty analyses enabled us to see a trend that males and low socioeconomic status groups are particularly vulnerable to long term health effects of ACEs. Similarly, though women had greater ACE exposure they had less ACS incidence perhaps because of social integration via their church communities. Lastly, an additional strength includes the utilization of validated measures in this study.

Study Limits

A number of limitations in this study should be noted. In particular, this cohort was quite educated (with the majority falling in the range of some college or higher) and financially stable (with 74% not experiencing any difficulty meeting basic expenses in the past year). Consequently, the generalizability of these findings is limited to those of similar socioeconomic status in the United States and may therefore underestimate the ACE and ACS effects.

Another limitation of this study includes the retrospective nature of the data gathered for the ACEs. Since the majority of the cohort are middle aged or older, a great deal of time has elapsed since their exposure to their family of origin; thus, we recognize that not only does the deterioration of memory play a role, but also the present life circumstances and events can heavily color how they view and report on their family of origin. However, we support the argument presented by Lehman and colleagues (2005) with respect to this limitation as they assert that the instrument on which the ACEs have demonstrated a dose-response relationship to a broad array of health outcomes (e.g. depression, cancer, coronary heart disease), thereby permitting the authors to conclude that a response bias is highly unlikely to yield such effects (Lehman et al., 2005, 2009; Taylor et al., 2006a, 2006b).

Additionally, all the spouse/partner support analyses included those who are currently in a relationship. The exclusion of divorced participants highlights a selection bias for our study, as the participants included are those who have achieved and maintained a relationship; as such, the negative spouse/partner support effects are likely to be an underestimate. Furthermore, the response-bias for Blacks as evidenced by the lower ACS incidence in comparison to Whites in inconsistent with the current patterns of heart disease cited in the literature, made ethnic comparisons untenable.

Despite the intriguing poverty and gender moderation effects found in this study for social integration and support, one of the limitations is the non-significant 3-way interaction terms. As a result, the moderation effects are small and require further study to draw conclusions. The non-significant terms are likely due to the majority of the cohort being highly educated and having financial resources (i.e. 86.9% had little or no

difficulty meeting expenses for basic needs in the past year). Subsequently, the study had limited power to detect the group differences that was needed to achieve statistical significance.

Furthermore, the current study primarily assessed only how social integration and support can operate on the ACE and ACS relationship, and other pathways were not examined. Additional indirect effects as well as moderators were not examined in this study and warrant further exploration. Lastly, while mortality data were examined in the study, the assessment of ACE exposure was compromised. Ongoing assessment of cardiac and all-cause mortality are forthcoming in the present cohort study.

Future Directions and Conclusion

We demonstrated the ACE-ACS link was stronger for men, despite women reporting significantly more exposure to ACEs. This information could be utilized in future studies to further investigate factors leading to this gender difference. In addition, our study demonstrated that social integration and functional support do not mediate the ACE-ACS link in a traditional way. Given the suppression effects described above, additional studies that investigate gender and SES differences are needed to further explore these results in more depth. Due to consistent findings that poor social support leads to fatal-heart attacks, future studies should investigate larger samples to further assess the role of ACE exposures and social support and its contribution to mortality by gender and SES.

Overall, the study emphasizes the importance of early risk factors in the identification and treatment of risk groups for poor health outcomes. In fact, results from

the current study and related research should be used to inform prevention and intervention with children and families. In particular, early interventions and resources that can promote a safe and loving family environment for children are likely to negate some of the long term consequences that are shown here. Moreover, research should attempt to identify resilient features (personal and environmental) which enhance the likelihood that individuals exposed to ACEs in the early years of life can lead a healthy life. Such efforts could help break the possible perpetuation of the toxic effects of ACEs from one generation to the next.

REFERENCES

Amato, P. & Booth, A. (1996). A prospective study of divorce and parent-child relationships. *Journal of Marriage and Family, 58*(2), 356-365. DOI: 10.230735-3501.

Anda RF, Croft JB, Felitti VJ, et al. (1999). Adverse childhood experiences and smoking during adolescence and adulthood. *JAMA, 282*, 1652–1658. DOI: 10.1001/jama.282.17.1652.

Batten, S., Aslan, M., Maciejewski, P., Mazure, C. (2004). Childhood maltreatment as a risk factor for adult cardiovascular disease and depression. *Journal of Clinical Psychiatry*, *2*, 249-254. DOI: 10.4088/JCP.v65n0217.

Berkman, L., Leo-Summers, L., Horwitz., R. (1992). Emotional support and survival after myocardial infarction. A prospective, population-based study of the elderly. *Annals of Internal Medicine, 117*, 1003–1009. DOI: 10.7326/0003-4819-117-12-1003.

Berkman, L., Vaccarino, V., Seeman, T. (1993). Gender differences in cardiovascular morbidity and mortality: the contribution of social networks and support. *Annals of Behavioral Medicine, 75*, 112-118.

Briere, J. & Runtz, M. (1988). Multivariate correlates of childhood psychological and physical maltreatment among university women. *Child Abuse & Neglect,12*, 331-341. DOI: 10.1016/0145-2134(88)90046-4.

Briere, J. & Runtz, M. (1993). Childhood sexual abuse: Long-term sequelae and implications for psychological assessment. *Journal of Interpersonal Violence, 8,* 312–330. DOI: 10.1177/088626093008003002.

James C. Coyne and Niall Bolger (1990). Doing Without Social Support as an Explanatory Concept. *Journal of Social and Clinical Psychology*, *9*(1), 148-158. DOI: 10.1521/jscp.1990.9.1.148.

National Center for Chronic Disease Prevention and Health Promotion, Division for Heart Disease and Stroke Prevention. Retrieved from http://www.cdc.gov/heartdisease/facts.htm.

National Center for Chronic Disease Prevention and Health Promotion. Office on Smoking and Health. Retrieved from http://www.cdc.gov/tobacco/data_statistics/fact_sheets/adult_data/cig_smoking/

De Vogli, R., Chandola, T., Marmot, M. (2007). Negative aspects of close relationships and heart disease. *Archives of Internal Medicine, 167*(18), 1951-1957. DOI: 10.1001//archinte.167.18.1951.

DiLillo (2001). Interpersonal functioning among women reporting a history of childhood sexual abuse: empirical findings and methodological issues. *Clinical Psychology Review, 21*(4), 553-576. DOI: 10.1016/SO272-7358(99)00072-0.

Dishion, T.J. (1990). The family ecology of boys peer relations in middle childhood. *Child Development, 61,* 874-891. DOI: 10.23.07/1130971.

Dong, M., Dube, S., Felitti, V., et al. (2003). Adverse childhood experiences and self-reported liver disease: New insights into the causal pathway. *Archives of Internal Medicine,163,* 1949–1956. DOI: 10.1001/archinte.163.16.1949.

Dong, M., Giles, W., Felitti, V., Dube, S., Williams, et al. (2004). Insights into causal pathways for ischemic heart disease: Adverse Childhood Experiences Study. *Circulation, 110,* 1761-1766. DOI: 10.1161/.01.CIR.0000143074.54995.7F.

Felitti, V., Anda, R., Nordenberg, D., Williams, F., Apitz, A., Edwards, V., …Marks, J. (1998). Relationship of Childhood Abuse and Household Dysfunction of Many of the Leading Causes of Death in Adults. *American Journal of Preventive Medicine, 14*(4), 245-258. DOI: 10.1016/s0749-3797(98)00017-8.

Flinn, M. & England, B. (1997). Social Economics of Childhood glucocorticoid stress response and health. *American Journal of Physical Anthropology, 102*, 33-53. DOI: 10.1002/(SICI)1096-8644(199701)102:1<33::AID-AJPA4>3.0.CO;2-E.

Fuhrer, R., Stansfeld, S., Chemali, J., & Shipley, M. (1999). Gender, social relations and mental health: prospective findings from an occupational cohort (Whitehall II study). *Social Science and Medicine, 48*(1), 77-87.

Fuller-Thomson, E., Brennenstuhl, S., Frank, J. (2010). The association between childhood physical abuse and heart disease in adulthood: Findings from a representative community sample. *Child Abuse & Neglect, 34*(9), 689-698.

Gauthier, L., Stollak, G., Messe, L., & Aronoff, J. (1996). Recall of childhood neglect and physical abuse as differential predictors of current psychological functioning. *Child Abuse & Neglect, 20*, 549-559

Gibson, R., Hartshorne, T. (1996). Childhood sexual abuse and adult loneliness and network orientation. *Child Abuse & Neglect, 20*(11), 1087-1093.

Goodwin, R. & Stein, M. (2004). Association between childhood trauma and physical disorders among adults in the United States. *Psychological Medicine, 34*(3), 509-520.

Harmer, A., Sanderson, J., Mertin, P. (1999). Influence of negative childhood experiences on psychological functioning, social support, and parenting for mothers recovering from addiction. *Child Abuse & Neglect, 23*(5), 421-433.

Hemingway, H. & Marmot, M. (1999). Psychosocial factors in the aetiology and prognosis of coronary heart disease: systematic review of prospective cohort studies. *British Medical Journal, 318*(7196), 1460-1467.

Hummer, R., Ellison, C., Rogers, G., Moulton, B., et al. (2004). Religious Involvement and Adult Mortality in the United States: Review and perspective. *Southern Medical Journal, 97*(21), 1223-1230.

Ikeda, A., Iso, H., Kawachi, I., Yamagishi, K et al. (2008). Social Support and Stroke and Coronary Heart Disease: The JPHC Study Cohorts II. *Journal of the American Heart Association, 39, 768-775.*

Kawachi, I., Colditz, G., Ascherio, A., Rimm, E. et al. (1996). A prospective study of social networks in relation to total mortality and cardiovascular disease in men in the USA. *Journal of Epidemiology and Community Health, 50,* 245-251.

Kinard, E., (1994). Methodological issues and practical problems in conducting research on maltreated children. *Child Abuse & Neglect, 18*(8):635–656.

King, D., Mainous, A., Taylor, M. (2004). Clinical use of C-reactive protein for cardiovascular disease. *Southern Medical Journal* 97, pp. 985–988.

Klohnen, E. C., & Bera, S. (1998). Behavioral and experiential patterns of avoidantly and securely attached women across adulthood: A 31-year longitudinal perspective. *Journal of Personality and Social Psychology, 74,* 211–223.

Korkeila, J., Vahtera, J., Korkeila, K., Kivimaki, M. et al. (2010). Childhood adversities as predictors of incident coronary heart disease and cerebrovascular disease. *Heart (British Cardiac Society),* 96*(4*), 298-303. DOI: 10.1136/hrt.2009.188250.

Krause, N. (2005). Negative interaction and heart disease in late life: exploring variations by socioeconomic status. *Journal of Aging and Health,* 17*(1)*, 28-55.

Kuper, H., Adami, H., Theorell, T., Weiderpass, E. (2006). Psychosocial determinants of coronary heart disease in middle-aged women: a prospective study in Sweden. *American Journal of Epidemiology*, 164, 349 –357.

Lehman, B., Taylor, S., Kiefe, C., Seeman, T. (2005). Relation of Childhood Socioeconomic Status and Family Environment to Adult Metabolic Functioning in the CARDIA Study. *Journal of Psychosomatic Medicine, 67*, 846-854.

Lett, H., Blumenthal, J., Babyak, M., Sherwood, A., Strauman, T., Robins, C., Newman, M. (2004). Depression as a risk factor for coronary artery disease: evidence, mechanisms, and treatment. *Journal of Psychosomatic Medicine 66*(3), 305-15.

Lett, H., Blumenthal, J., Babyak, M., Strauman, T., Robins, C. et al (2005). Social support and coronary heart disease: epidemiologic evidence and implications for treatment. *Journal of Psychosomatic Medicine, 67*, 869–87

Lindblad, U., Langer, R., Wingard, D., Thomas, R. & Barrett-Connor, E. (2001). Metabolic syndrome and ischemic heart disease in elderly men and women. *American Journal of Epidemiology, 153,* 481-489.

Luecken, L., Kraft, A., & Hagan, M. (2009). Negative relationships in the family-of-origin predict attenuated cortisol in emerging adults. *Hormone Behavior, 55*(3), 412-417. DOI: 10.1016/j.yhbeh.2008.12.007.

Lund, R., Rod, N., Christensen, U. (2010). Are negative aspects of social relations predictive of angina pectoris? A 6-year follow up study of middle-aged Danish women and men. *Journal of Epidemiology and Community Health*, *66*, 359-365. DOI: 10.1136/jech.2009.106153.

Malinosky-Rummell R, Hansen, D. (1993). Long-term consequences of childhood physical abuse. *Psychological Bulletin, 114*, 68–79.

Maselko, J., Kubzansky, L., Kawachi, L, Seeman, T. & Berkman, L. (2007).Religious service attendance and allostatic load among high-functioning elderly. *Journal of Psychosomatic Medicine, 69*(5), 464-472.

McEwen, B., & Stellar, E. (1993). Stress and the individual: mechanisms leading to disease. *Archives of Internal Medicine, 153*(18), 2093-2101.

Newsom, J., Rook, K., Nishishiba, M., Sorkin, D., and Mahan, T. (2005). Understanding the relative importance of positive and negative social exchanges: examining specific domains and appraisals. *Journals of Gerontology. Series B, Psychological Sciences and Social Sciences, 60*, 304–331.

Orth-Gomer, K., Rosengren, A., and Wilhelmsen, L.(1993). Lack of social support and incidence of coronary heart disease in middle-aged Swedish men. *Psychosomatic Medicine*, 55, 37–43.

O'Hagan, K. (1995). Emotional and psychological abuse: problems of definition. *Child Abuse & Neglect, 19*, 449-461.

Paolucci, *E.*, Violata, C. (2001). A meta-analysis of the published research on the effects of child sexual abuse. *The Journal of Psychology, 135*, 17-36.

Pargament, K., Koenig, H., Tarakeshwar, N. & Hahn, J. (2004). Religious coping methods as predictors of psychological, physical and spiritual outcomes among medically ill elderly patients: a two-year longitudinal study. *Journal of Health Psychology, 9*(6), 713-730.

Parkinson, P., Humphrey, C. (1988). Children who witness domestic violence: The implications for child protection. *Child and Family Law Quarterly, 10*, 147-159.

Reed, D., McGee, D., Yano, K., Feinleib, M. (1983). Social networks and coronary heart disease among Japanese men in Hawaii. *American Journal of Epidemiology, 117*; 384-96.

Repetti, R., Taylor, S., Seeman, T. (2002). Risky families: family social environments and the mental and physical health of offspring. *Psychological Bulletin, 128*, 330 – 366.

Rose G, Marmot MG. Social class and coronary heart disease *1981*). British Heart Journal, 45*:13-19.

Ross R. (1999). Atherosclerosis: an inflammatory disease. *New England Journal of Medicine*, 340, 115–126.

Robles, T. F., & Kiecolt-Glaser, J. K. (2003). The physiology of marriage: pathways to health. *Physiology & Behavior 79*, 409-416.

Rosengren, A., Wilhelmsen, L., Orth-Gomer, K. (2004). Coronary disease in relation to social support and social class in Swedish men: a 15 year follow-up in the study of men born in 1933. *European Heart Journal, 25*, 56–63.

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

Ryff, C. D., Singer, B. H., & Palmersheim, K. A. (2004). Social inequalities in health and well-being: The role of relational and religious protective factors. In O. G. Brim,

C. D. Ryff & R. C. Kessler (Eds.), *How healthy are we? : A national study of well-being at midlife* (pp. 90-123). Chicago: University of Chicago Press.

Seeman, T. & Syme, S. (1987). Social networks and coronary artery disease: a comparison of the structure and function of social relations as predictors of disease. *Journal of Psychosomatic medicine, 49*(4), 341-254.

Shaw, L., Merz, C., Bittner, V., Kip, K., et al . (2008). Importance of socioeconomic status as a predictor of cardiovascular outcome and costs of care in women with suspected myocardial ischemia. Results from the National Institutes of Health, National Heart, Lung and Blood Institute-sponsored Women's Ischemia Syndrome Evaluation (WISE). *Journal of Women's Health, 17*(7), 108-192. DOI: 10.1089/jwh.2007.0596.

Smith, T. & Ruiz, J. (2002). Psychosocial influences on the development and course of coronary heart disease: Current status and implications for research and practice. *Journal of consulting and Clinical Psychology, 70*(3), 548-568.

Smith, T. & Ruiz, J. (2002). Psychosocial influences on the development and course of coronary heart disease: Current status and implications for research and practice. *Journal of Consulting and Clinical Psychology, 70*(3), 548-568.

Stroud D. (1999). Familial support as perceived by adult victims of childhood sexual abuse. *Sexual Abuse: Journal of Research & Treatment, (2),* 159–175

Taylor, S., Lehman, B., Kiefe, C., Seeman, T (2006). Relationship of early life stress and psychological functioning to adult C-reactive protein in the Coronary Artery Risk Development in Young Adults Study. *Biological Psychiatry 60*, 819–824.

Vranceanu, A., Stevan, H., Johnson, R. (2007). Child multi-type maltreatment and associated depression and PTSD symptoms: The role of social support and stress. *Child Abuse & Neglect, 31*(1), 71-84.

Wielgosz A. and Nolan R. (2000). Biobehavioral factors in the context of ischemic cardiovascular diseases. *Journal of Psychosomatic Research, 48,* 339–345.

Woodall, K. L., & Matthews, K. A. (1989). Familial environment associated with Type A behaviors and psychophysiological responses to stress in children. *Health Psychology, 8,* 403-426.