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Child Adversity, Adult Intimate Partner Violence and Inflammation in Young Adults

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Child Adversity, Adult Intimate Partner Violence and Inflammation in Young Adults

A dissertation submitted in partial satisfaction of the

requirements for the degree of Doctor of Philosophy

in Public Health

by

Elinam Dzifa Dellor

2016

ABSTRACT OF DISSERTATION

Child Adversity, Adult Intimate Partner Violence and Inflammation in Young Adults

by

Elinam Dzifa Dellor

Doctor of Philosophy in Public Health

University of California, Los Angeles, 2016

Professor Dawn M. Upchurch, Chair

Negative childhood experiences and adult intimate partner violence may leave individuals vulnerable to poor mental and physical health in adulthood. Victims of emotional abuse and intimate partner violence have a higher risk of developing chronic medical conditions in adult life, including cardiovascular disease. Similarly, adults who lived in poverty as children have higher risk of cardiovascular disease. The immune system releases acute-phase proteins such as C-reactive protein (CRP) not only in response to infections but also in response to exposure to traumatic experiences. CRP has been linked to the incidence and progression of atherosclerosis, a precursor for cardiovascular disease. To understand potential pathways that link emotional abuse, intimate partner violence and exposure to poverty to chronic medical conditions in adulthood, this dissertation first investigates the association between emotional abuse and elevations in CRP. Second it examines the relationship between adult intimate partner violence and CRP. For emotional abuse and intimate partner violence gender interactions are tested. Lastly, the dissertation tests for an effect of poverty status in childhood and/or adulthood

on CRP and investigates the extent to which poverty may compound the effects of emotional abuse and intimate partner violence on CRP.

This dissertation uses data from the National Longitudinal Study of Adolescent to Adult Health (Add Health), a prospective observational study of a nationally representative sample of U.S adolescents enrolled in grades seven to twelve in the 1994-1995academic year. Funded by the National Institute of Child Health and Development (NICHD), the study is overseen by the Carolina Population Center at the University of North Caroline at Chapel Hill (UNC) (Harris, 2008). Add Health focuses on social factors that influence adolescent and young adult family and peer networks, health behaviors and health status. This dissertation uses data from three of the four waves of data: Wave I data was collected during 1994-1995 when respondents were enrolled in grades seven to twelve; Wave III data was collected from original Wave 1 respondents in 2001-2002 when these young adults were between the ages of 18 and 26 and Wave IV data was again collected from original Wave I respondents were between the ages 24 and 32 now settling into young adulthood. At Wave IV respondents provided blood samples in the form of dried blood spots (DBS), which were later analyzed for a variety of biological markers including C-reactive protein. All analyses for this dissertation used statistical methods adjusted for the complex sampling design of Add Health.

Repeated exposure to emotional abuse was significantly associated with CRP in fully adjusted models, although there was no evidence of a gender interaction. Similarly, experiencing chronic intimate partner violence in adulthood was significantly associated with elevated CRP levels in fully adjusted models. Young adults who lived in poverty as children had significantly higher CRP levels after adjusting for parents' education, young adults' own education level, demographic characteristics and known risk factors for CRP including smoking, BMI and

depressive symptoms. Respondents who lived in poverty as children and remained in poverty as adults also had significantly higher CRP levels, and the association appears to be mediated by BMI.

This dissertation finds support for the enduring effects of early life adversity and adult traumatic experiences on inflammation—a risk factor for cardiovascular disease. These findings also mirror population level disparities in the incidence of cardiovascular disease. Public health implications include health education efforts to increase knowledge about the physiologic consequences of emotional abuse and intimate partner violence. Secondary prevention efforts include routine screenings for exposure to emotional abuse, intimate partner violence and material adversity in primary care settings as well as increasing referrals to community based programs designed to help families cope with adversity.

The dissertation of Elinam Dzifa Dellor is approved.

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2016

DEDICATION

For my grandmother and first teacher, Irene “Mamavi” Dei.

Mamavi, I miss you every day.

I hope you’re proud of your student.

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Dellor, E. Freisthler, B. "Introducing Technology in Child Welfare Referrals". Poster presentation at CalSWEC Continuous Quality Improvement (CQI) Symposium. Davis, CA. July 15, 2015.

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Dellor, E. "Maltreatment and Inflammation in Young Adults: The Role of Resilience Protective Factors". Poster presentation at American Public Health Association (APHA) Annual Meeting, New Orleans, LA. November 15-19, 2014

Dellor, E. "The Impact of Child Maltreatment on the Mental Health of Transitioning Foster Youth". Poster presented at American Public Health Association (APHA) Annual Meeting, October 27-31, 2012

Dellor, E. "Images of Modernity and Women's Sexuality in Nigerian Film". Oral presentation at the UCLA African Activist Association (AAA) Meeting. May 21, 2011

CHAPTER 1

INTRODUCTION AND SPECIFIC AIMS

1.1 Introduction

Adverse childhood experiences have an enduring effect on physical and mental health. Two of the most commonly studied dimensions of negative childhood life experiences are child maltreatment and poverty. Childhood experiences of maltreatment and poverty are both linked to significant psychological and behavioral consequences including depression, obesity, smoking and all-cause mortality (Aber et al., 1997; Brieding et al., 2015; Campbell, 2002; Putnam, 2003; Shonkoff, 2012). Researchers have also found that maltreatment and poverty in childhood are key risk factors for chronic health conditions with inflammatory origin such as cardiovascular disease (Batten, 2004; Coker, 2000; Felitti, 1998). More recently, traumatic experiences in adulthood, such as adult intimate partner violence, have also been linked to higher incidence of chronic diseases including cardiovascular disease.

Although not all individuals with a history of negative childhood experiences and adult intimate partner violence go on to develop cardiovascular disease, studies have begun to investigate the biological mechanisms through which early life adversities influence a variety of health outcomes including cardiovascular disease (Miller et al, 2011). Most of this work is based on Cohen, Kessler and Underwood's (1995) definition of stress as a process involving a stimulus, an appraisal of it, followed by a response. When stressors are appraised as unmanageable or threatening, they elicit a psychological state that is experienced as stress followed by a host of behavioral changes (Lazarus & Folkman, 1984). Under this definition, child maltreatment, adult intimate partner violence and poverty are all viewed as stressors that are chronic in nature in that all three tend to occur over a long period of time and in the case of

child maltreatment and intimate partner violence, the sense of fear and danger they elicit remain long after the threat itself is gone. Chronic stress can alter important structures in the brain, the stress response system and ultimately, the immune system. Inflammation is part of the immune response to psychosocial stressors and inflammation is recognized as an early marker of risk for chronic diseases with inflammatory origin (McEwen, 1998). C-reaction protein (CRP), one biological marker used to detect systemic inflammation has been shown to predict future cardiovascular disease independent of traditional risk factors (Cesasri et al., 2003; Kuo et al., 2005; Pearson et al., 2003). Recent studies further suggest systemic inflammation is one mechanism through which psychosocial stressors may contribute to incident cardiovascular disease (Graham et al., 2006; Ridker et al, 2002).

Albeit limited, studies have demonstrated an association between child maltreatment and elevated CRP levels in healthy adult populations (Carpenter, 2012; Danese et al., 2007) though fewer have examined this relationship in young adult populations (Slopen et al., 2012). To date, most studies focus on a broad range of early life adversities (Taylor, Lehman, Kiefe & Seeman, 2006) or adverse childhood experiences ranging from losing a parent to witnessing substance use (Danese, 2009; Tietjen et al., 2012), typically in one summative score. There is an additional need to investigate the differential effects of specific sub-types of maltreatment, such as emotional abuse (Bertone-Johnson et al., 2012). Additionally, although emotional abuse is increasingly recognized as a developmentally damaging form of maltreatment it has received little attention in the literature (Hart, 2011). The inflammatory effects of traumatic experiences in adulthood including intimate partner violence is also not well investigated (Newton et al., 2011; Clark et al, 2016) and while the more extensive literature on the inflammatory effects of socioeconomic status consistently show an inverse relationship between socioeconomic status

and inflammation, few studies have specifically tested the effect of poverty generally, and the timing and duration of poverty stays specifically.

With the understanding that many age-related chronic conditions have origins in childhood and that traumatic experiences over the life course contribute to disease risk, the present study adds to this line of inquiry by examining the effects of child maltreatment, more proximal histories of adult intimate partner violence and childhood and/or adult poverty on inflammation in a nationally representative sample of young adults in the United States. The present study adds to the literature by examining the extent to which the inflammatory effects of emotional and intimate partner violence are contingent on gender and by using a dynamic measure of poverty that integrates both the timing and chronicity of poverty in investigating its effect on inflammation.

This dissertation is guided by theories relating psychosocial processes as well as social conditions to biological changes in childhood and in adulthood. The primary objectives of the dissertation are to: 1) test whether young adult survivors of emotional abuse are characterized by elevated inflammation levels, 2) test whether a history of adult intimate partner violence is characterized by elevated levels of inflammation, 3) investigate the inflammatory consequences of experiences of poverty in childhood and/or adulthood and the extent to which the effects of emotional abuse and intimate partner violence are magnified by poverty status.

1.2 Specific Aims

Aim 1A: To examine the extent to which levels of inflammation differ by childhood experiences of emotional abuse independent of physical and sexual abuse, young adult socioeconomic status, demographic and familial characteristics and other known risk factors for inflammation.

Aim 1B: To examine the extent to which the effect of emotional abuse on inflammation is contingent on gender.

Description: Research suggests that child maltreatment affects the development of cardiovascular disease and that dysregulation of the immune system as measured by systemic inflammation may play a role. While there are many forms of child maltreatment, prior research has focused almost exclusively on physical abuse, sexual abuse and neglect. Although emotional abuse is quite common, it is seldom assessed and its inflammatory effects, above and beyond other subtypes of maltreatment is ill-understood. Nested weighted ordinary least squares regression models with an interaction between emotional abuse and gender are used to predict whether the inflammatory effect of emotional abuse is different for men and women.

Hypotheses:

1A. Levels of inflammation are expected to be higher among young adults with a history of emotional abuse.

1B. The association between emotional abuse and inflammation is expected to be contingent on gender such that women who have experienced emotional abuse have higher CRP levels compared to men who have experienced emotional abuse.

1C. The association between emotional abuse and inflammation is expected to persist after taking demographic and familial characteristics (e.g. age, race/ethnicity, parental education and parental income), young adult socioeconomic status, health behaviors and health status into account.

Aim 2A: To examine the extent to which levels of inflammation differ by history of adult intimate partner violence, independent of physical, sexual and emotional abuse in childhood, young adult socioeconomic status, demographic and familial characteristics, and other known risk factors for inflammation.

Aim 2B: To examine the extent to which the effects of intimate partner violence on inflammation are contingent on gender.

Description: Having tested the effect of childhood experiences of emotional abuse on inflammation in aim 1, this aim explores the inflammatory consequences of more proximal experiences of trauma: adult intimate partner violence. Weighted bivariate associations and Wald

tests are used to examine differences in inflammation (measured by CRP levels) by history of adult intimate partner violence. Nested weighted ordinary least squares regression models with an interaction between intimate partner violence and gender are used to predict CRP, and test whether the inflammatory effect of intimate partner violence is different for men and women.

Hypotheses:

2A. Levels of inflammation are expected to be higher among young adults with a history of intimate partner violence.

2B. The association between intimate partner violence and inflammation is expected to be contingent on gender such that female respondents with a history of intimate partner violence have higher CRP levels compared to male respondents.

2C. The relationship between intimate partner violence and inflammation is expected to persist after taking into account physical and sexual abuse, demographic and familial characteristics (e.g. age, race/ethnicity, parental education and parental income), young adult socioeconomic status, health behaviors and health status.

Aim 3A: Examine how exposure to poverty in childhood and/or adulthood influence inflammation independent of physical, sexual and emotional abuse in childhood, young adult socioeconomic status, demographic and familial characteristics, and other known risk factors for inflammation.

Aim 3B: Examine the extent to which the effects of poverty on inflammation are moderated by experiences of emotional abuse.

Aim 3C: Examine the extent to which the effects of poverty on inflammation are moderated by experiences of intimate partner violence.

Description: While past work links low socioeconomic status in childhood to elevated inflammation levels, few directly examine the effect of living in poverty. This aim uses measures of poverty that incorporate two dimensions of poverty, timing and chronicity, as well as changes in income mobility to test the effect of living in poverty on inflammation. Weighted bivariate associations and Wald tests are used to examine differences in inflammation (measured by CRP levels) by experiencing poverty from childhood to adulthood. Two nested weighted ordinary least squares regression models are used: 1) with an interaction between emotional abuse and experiencing poverty from childhood to adulthood and 2) with an interaction between intimate partner violence experiencing poverty from childhood to adulthood to predict whether the inflammatory effects of emotional abuse and of intimate partner violence varies by experiencing poverty from childhood.

Hypotheses:

3A. Young adults who experienced poverty in childhood and adulthood (persistent poverty) will have the highest inflammation levels while those who never experienced poverty will have the lowest.

3B. The association between persistent poverty and inflammation is expected to be contingent on emotional abuse such that the impact of childhood poverty on inflammation is greater for young adults with a history of emotional abuse compared to those with no exposure to emotional abuse.

3C. The association between persistent poverty and inflammation is expected to be contingent on intimate partner violence such that the impact of persistent poverty on inflammation is greater for young adults who also experienced intimate partner violence compared to those with no history if intimate partner violence.

1.2 Dissertation Overview

The dissertation is organized as follows. Chapter 2 describes the background, significance and implications of chronic stressors in childhood and adulthood. Chapter 3 provides an overview of the theoretical models used to guide the dissertation and presents a conceptual model. Chapter 4 reviews the research design and analytic methods used to model each research question. In Chapters 5, 6 and 7, I present results for nested weighted ordinary least squares regression models of inflammation on emotional abuse (Aim 1), intimate partner violence (Aim 2) and childhood and/or adult poverty (Aim 3) respectively. Lastly, Chapter 8 presents a discussion of findings along with the strengths, limitations and public health implications of the present study.

CHAPTER 2

BACKGROUND AND SIGNIFICANCE

2.1 Overview

This chapter presents a review of the literature on the background and significance for the present dissertation. The chapter begins with an overview of the classification and prevalence of child maltreatment and intimate partner violence, followed by an overview of poverty in the United States. In the second section, epidemiologic studies linking child maltreatment and adult intimate partner violence to cardiovascular disease are reviewed. In the third section the literature on inflammation as a risk marker for cardiovascular disease is presented along with available empirical literature linking child maltreatment and intimate partner violence to inflammation. A discussion of the physiologic mechanisms linking child maltreatment and intimate partner violence to inflammation is then presented. Next, an overview of gender differences in exposure and consequence of child maltreatment and intimate partner violence is discussed followed by a discussion of key covariates including depression, socioeconomic status, smoking, body mass index, age and race/ethnicity. Lastly, I present the gaps in existing literature testing physiologic dysregulation as a consequence of traumatic experiences.

2.2 Classification and Prevalence of Child Maltreatment and Intimate Partner Violence

2.2.1 Traumatic Stressors

Child maltreatment and intimate partner violence are both classified as traumatic stressors (Wheaton, 1994; Tjaden & Thoennes, 2000). Victims of child maltreatment and intimate partner violence are often exposed to repeated incidents of violence on multiple occasions over a prolonged period of time. Thus, victims typically live with constant anticipation of assault within families characterized by high levels of aggression and are unable to seek

comfort from others. Chronic traumatic experiences are generally distinguished from other stressful life events by the timing, magnitude and abnormality of the event (Shaw, 2002). Although traumatic experiences are broad in scope, they can be categorized as events that are "outside the range of human experience" that have the capacity to elicit feelings of "intense fear, helplessness, or horror" (4th ed., DSM-IV; American Psychiatric Association (APA); pp. 427-428). These physical, sexual or threatening acts may not only cause injury, but also intense fear and perceived threat of life, all of which are elements of traumatic stress that may remain long after the assault itself has ended. It is this prolonged fear, hyper-vigilance and powerlessness that carries with it health compromising effects over the life course (Newton et al., 2011).

2.2.2 Child Maltreatment

Child maltreatment is defined as any non-accidental "act by a parent or caregiver that results in harm, potential harm or threat of harm to children under the age of 18" (Leeb, 2008). This definition, however, covers a broad spectrum of abuse and the congressionally mandated assessment of maltreatment, the National Incidence Study (NIS), further identifies four common sub-types of maltreatment: physical abuse, sexual abuse, emotional abuse and neglect (Sedlak et al., 2010). Physical abuse is defined as acts of commission by a caregiver that causes actual physical harm or potential for harm. Sexual abuse occurs when a caregiver uses a child for sexual gratification. Emotional abuse includes acts such as denigration, ridicule and intimidation that have adverse effects on the emotional health and development of a child and neglect includes inadequate supervision of children, inattention to the emotional needs of children, denying children physical access to the home and failure to seek medical attention for children (Sedlak et al., 2010).

In 2014, US Child Protective Services (CPS) received approximately 3.6 million reports alleging child maltreatment (USDHHS, 2013). Of these, an estimated 702,000 children (9.4 per 1,000) were determined to be victims of maltreatment (CDC, 2014). Perhaps owing to stringent legal requirements needed to substantiate maltreatment allegations, there is a large discrepancy between official CPS reports of maltreatment and rates reported by victims and parents in community samples. The National Survey of Children's Exposure to Violence (NatSCEV) conducted between January 2008 and May 2008 found that among children ages zero to seventeen living in the contiguous United States, ten percent were exposed to some form of maltreatment. An estimated six percent experienced sexual abuse in their homes while twenty-five percent reported ever witnessing violence within the home (Finkelhor, 2009).

The National Comorbidity Study Replication (NCS-R), found twenty percent of adult males and twenty-five percent of adult females report experiencing or witnessing violence in the home by age thirteen. They also found that one in three adults report exposure to physical or sexual abuse as children (Koenen, 2010). The second National Survey of Children's Exposure to Violence (NatsCEV II), conducted in 2011 provides more recent prevalence estimates showing that more than one in ten children experienced at least one form of maltreatment by a caregiver with nearly four percent reporting physical abuse (Finkelhor, 2013). Overall, an estimated two percent reported sexual abuse although the rate among girls fourteen to seventeen years was over ten percent (Finkelhor, 2015). In a review of the community-based surveys reports of physical abuse ranged from four to sixteen percent, approximately ten percent of children were neglected or emotionally abused with five percent reporting sexual abuse (Gilbert et al., 2009). The gap between official estimates and the much higher rates reported in community surveys support the

idea that a substantial number of children exposed to maltreatment do not come to the attention of official agencies (Finkelhor, 2009; Fergusson, 2008).

Moreover, maltreatment is not consistently captured, or measured in research. There is great heterogeneity in the ways in which child maltreatment is assessed in empirical studies (Fallon, 2010; Schilling, Aseltine & Gore, 2008). With some exceptions, including direct access to official case records, child maltreatment is typically assessed using retrospective self-administered questionnaires. Under the umbrella of retrospective reports, one approach is to use instruments that measure specific subtypes of child maltreatment. Of these, the Child Trauma Questionnaire (CTQ) is the most widely used instrument. The CTQ is a 28-item self-administered instrument consisting of five subscales: emotional abuse, emotional neglect, physical abuse, physical neglect and sexual abuse (Bernstein et al., 1994). The scores on the CTQ are based on a 4-point likert scale, ranging from 1 (absence of abuse) to 4 (severe abuse). The CTQ yields a total score as well as subscale scores for emotional abuse, emotional neglect, physical abuse, physical neglect and sexual abuse. A short form CTQ version with eight questions is also commonly used (Bernstein et al., 2003). As one of the first instruments developed for measurement of child abuse, the CTQ has been extensively tested among in ethnically diverse samples (Scher et al., 2001), healthy (Carpenter et al., 2012; Bernstein & Fink, 1998) and clinical samples (Lu et al, 2013; Pace et al., 2012) with reported Cronbach's alpha of 0.79-0.94 and reliability coefficients of 0.80-0.83 (Roy & Perry, 2004).

The Early Trauma Inventory (ETI) (Bremmer & Vermetten, 2000) and the Childhood Experience of Care and Abuse Questionnaire (CECAQ) (Brown et al., 2007) are also commonly used in the literature. The ETI (Cronbach's alpha=0.75-0.95) is similar to the CTQ in that it yields subscores for four subtypes of abuse. Unlike the CTQ however, the ETI includes domains

for witnessing violence, separation from parents and experiencing loss. The underlying constructs and measures of the CECAQ (Cronbach's alpha=0.86-0.91) are similar to the CTQ and ETI. However, the administration of it differs from the others, as it is a semi-structured interviewer-administered instrument.

The second approach to measurement uses wider definitions of childhood trauma and early life stress to create summative scores of exposure to adverse childhood experiences (ACEs) (Felitti et al., 1998) or risky families (Taylor et al., 2004) to create summative scores of traumatic childhood experiences. The ACEs self-administered questionnaire measures exposure to one or more of the following adverse childhood experiences: emotional abuse, physical abuse, sexual abuse, exposure to substance abuse, exposure to mental illness, violence in the home and exposure to criminal activity. Responses are binary and exposure to four or more ACEs is typically observed as marking high ACE exposure (Anda et al., 2009; Dong et al, 2004; Dube et al., 2004). The ACEs questionnaire has been adapted and used in various studies with reported Cronbach's alpha between 0.88 and 0.91. A second commonly used measure of early life trauma is the Risky Families questionnaire. The Risky Families questionnaire (Cronbach's alpha=0.77-0.85) was adapted from the ACEs questionnaire (Taylor et al., 2004). It includes the above ACE items as well as other aspects of the family environment including whether individuals lived in well-organized/well-managed households and whether family members knew what children were up to at any given time. Each item is measured on a 4-point scale ranging from 1 (rarely or none of the time) to 4 (most or all of the time) and higher values represent riskier families.

These differences in the operational definition of maltreatment can make it difficult to make comparisons across studies. Additionally, studies do not always identify the specific sub-type of maltreatment experienced by individuals (Fletcher, 1999). Most operationalize different

types of trauma as one phenomenon (Cuelho et al., 2013) by using general scores or by dichotomizing responses to the presence or absence of maltreatment. This is due in part to unreliable self-reports, limited information from Child Protective Services and the co-occurrence of different sub-types of maltreatment. There is conceptual support for using overall measures of adverse childhood events as is done with the Risky Families and ACEs questionnaires. Children who have experienced maltreatment, for example, may grow up in dysfunctional families characterized by conflict, parental substance use and strained parent-child relationships. Some studies report substantial proportions of victims of maltreatment, particularly those in protective services are victims of two or more sub-types of maltreatment (Finkelhor, 2013). Thus, exposure to any and all of these risk factors may result in negative health outcomes. It is also true, however, that the nature and health consequences of trauma can differ depending on the specific trauma under study (Manly et al., 1994) and that analyses of specific types of maltreatment offer a window into the nature of experiences and consequences of maltreatment (Widom, 1994). However, because few studies differentiate among the various sub-types of maltreatment, their relative impact on physical health outcomes is largely unknown.

2.2.2.1 Emotional Abuse

Emotional abuse in particular is understudied. A major deterrent in the study of emotional abuse is the difficulty in operationally defining and thus recognizing and measuring the construct. Examples of emotional abuse include acts of rejecting, exploiting, degrading, isolating, mis-socializing and denying emotional responsiveness (Claussen & Crittenden, 1991; Garbarino & Garbarino, 1986) The difficulty arises in part because most children occasionally experience one or more of these acts at low levels (Claussen & Crittenden, 1991). It is therefore unlikely single instances of emotional abuse are directly harmful in same way physical and

sexual abuse lead to injuries, leading to considerable debate about the levels of repeated occurrences of these acts constitute "abuse". The end result of which is delayed recognition of emotional abuse such that children are exposed to emotional abuse over a longer period of time (Glaser 2002). The matter is further complicated in those cases where there is no clear intent to harm the child although the interaction between the caregiver and child is harmful to the child. Lastly, more often than not emotional abuse does not cause bodily harm to the child. Despite the above stated difficulties in defining emotional abuse, empirical studies demonstrate professionals and lay persons alike are able to recognize emotionally abusive situations. For example, using 20 vignettes of interactions between adults and children, Burnett (1993) found that a group of 381 lay persons and 452 social work professions identified the same nine vignettes as emotional abuse. Nonetheless, among children involved in the child welfare system, emotional abuse is rarely cited as the primary reason for entry into the child welfare system (Hart & Glaser, 2011). Empirical studies show the presence of emotional abuse in the majority of referrals based on allegations of physical abuse and compared to physical abuse, emotional abuse more strongly predicted impairments in children's development (Claussen & Crittenden, 1991; Hart, Binggeli, & Brassard, 1998).

2.2.3 Adult Intimate Partner Violence

Adult intimate partner violence is defined by the Center for Disease Control and Prevention (CDC) as "physical or sexual violence, or threats of such violence between current spouses, former spouses or intimate partners" (Breiding, Chen & Black, 2015). The most current data are from the 2010 National Intimate Partner and Sexual Violence Survey (NISVS) which indicate that over a third of women (35.6%) and more than a quarter of men (28.5%) experience threats, physical or sexual assault by an intimate partner in their lifetime (Breiding, Chen &

Black, 2015). Further, an estimated six percent of women and five percent of men experience rape, physical violence, threats or psychological aggression by an intimate partner in the twelve months prior to data collection. Prior to the initiation of the NISVS, the 2005 Behavioral Risk Factor Surveillance System (BRFSS) indicated nearly twelve percent of women and eleven percent of men in the United States reported experiencing physical or sexual intimate partner violence during their lifetime with a twelve-month period prevalence of two and one percent for women and men respectively (Tjaden & Thoennes, 2000). The apparent increase in prevalence between 2005 and 2010 is due largely to the inclusion of threats as a dimension of intimate partner violence in 2010. Prevalence estimates are also conducted in medical settings, often in acute care settings however these estimates are generally higher than in population-based surveys due to the nature of the population seeking acute care. For example, the prevalence of intimate partner violence among women seeking treatment in primary or family care settings range from 35 percent to 50 percent (Bauer, Rodriguez & Perez-Stable, 2000; Coker et al., 2000; Kramer & Mueller, 2004).

By far the most widely used instruments to assess victimization by an intimate partner in community samples are the original Conflict Tactics Scale (CTS) (Strauss, 1979) and the revised Conflict Tactics Scale (CTS2) (Straus et al., 1996). The CTS2 has five subscales measuring negotiation: psychological aggression, physical assault, sexual coercion and injury. Respondents rate the number of times within the previous 12 months from a 0 (Never) to 5 (more than 20 times). In a review of studies using the CTS2 to assess intimate partner violence Cronbach's alpha ranged from 0.34 to 0.94 (Straus, 2005). Though the CTS is also used for clinical assessment (Aldarondo & Strauss, 1994), semi-structured interviews such as Slapped, Threatened and Throw (STaT) (Paranjape & Liebschutz, 2003) and staff-administered

questionnaires such as Hurt, Insult, Threaten and Scream (HITS) (Sherin wt al., 1993) are often used in clinic settings.

2.2.4 Poverty and the Family Environment

Despite voluminous literature in the social sciences on the causes, correlates and consequences of poverty, there are various approaches used to study and understand poverty. Some base definitions on social, behavioral, and political foundations of human well-being while others conceptualize poverty as a function of income and consumption, such that poverty is defined as an individual's ability to obtain a basic level of consumption (Hagenaars, 1991). The latter is the most widely used definition of poverty in the literature is further refined as absolute, relative and subjective concepts of poverty. Absolute poverty, typically based on the minimum food calorie intake refers to a lack of basic means for survival, of basic needs for survival. Here, being "non-poor" means refers to the ability to avoid absolute deprivation. Subjective poverty typically refers to respondents self-assessments using various monetary and non-monetary indicators. Relative poverty refers to populations who earn less than a predetermined amount of income (typically less than 50% of the mean or median income) in a given society. The US Census Bureau formally adopted the use of relative poverty in the 1960s by to define and track US poverty levels specific to household size (USDHHS, 2015). Families with annual incomes above these thresholds are deemed "not poor" while families with income below these thresholds are deemed "poor". Poverty thresholds differ by family size and are adjusted annually for changes in cost of living in the United States.

According to the US Census Bureau, in 2014 (the most recent year for which data are available), approximately 47.6 million people in the United States (14.8%) were living below the poverty line. This figure has hovered between 11 and 15.5% since 1990 and given its pervasive

nature, has led to extensive research on health consequences of poverty. Living in poverty is known to negatively affect the health of children and adults alike. One difficulty is income poverty itself is correlated with various social conditions that themselves lead to negative health and behavioral consequences (Aber, et al., 1997).

Much of the research on risk factors for child maltreatment and intimate partner violence is based on the understanding that trauma within families occur within multiple levels of embedded systems ranging from individual characteristics to structural factors (Smith et al., 2009). For example, researchers have identified socioeconomic correlates of maltreatment. Although not invariably linked to poverty, child maltreatment—physical and emotional abuse in particular—is more prevalent in low-income neighborhoods (Freisthler, Merritt & LaScala, 2006) and dynamics within families are influenced by macro-level factors such as poverty (Repetti et al., 2002). Thus, parents from lower socioeconomic households are more likely to employ harsh parenting styles and use corporal punishment (Straus et al., 2010). Similarly, intimate partner violence is more frequent in low socioeconomic groups (Jewkes, 2002). Some argue that lower income families are overrepresented because they are more likely to come to the attention of authorities compared to their well-to-do counterparts (Garbarino & Kostelny, 1992). However, for population based surveys using self-reports of maltreatment, an alternative explanation is that compared to families in higher socioeconomic positions, families living in poverty are exposed to a greater number and/or intensity of stressors related to material need (Hamilton et al., 1990).

A narrative review of risk factors for intimate partner violence identified low socioeconomic status and unemployment as important risk factors for intimate partner violence (Riggs, Caulfield & Street, 2000). Women living in low socioeconomic areas face higher risk of

intimate partner violence with studies reporting up to sixty-eight percent of women living in low socioeconomic neighborhoods experience intimate partner violence or another traumatic event at least once their lifetime (Resnick et al., 1993; Schumm et al, 2005). Financial stress is generally related to instability (Conger et al., 2010) and to intimate partner violence (Neff et al., 1995; Smith et al., 2010) in community samples in married and cohabiting couples.

2.3 Child Maltreatment, Intimate Partner Violence, Poverty and Cardiovascular Disease

In the last decade, research has shifted focus from the mental health correlates of child maltreatment and intimate partner violence to include associated chronic health conditions (Black, 2011; Campbell, 2002; Goodwin & Stein, 2004). Victims of child maltreatment and intimate partner violence are at higher risk for cancer (Breiding, Black & Ryan, 2008; Felitti et al., 1998), lung disease (Felitti et al., 1998; Goodwin and Stein, 2004), diabetes (Coker et al.,, 2000; Goodwin & Weisberg, 2002), stroke (Breiding, Black & Ryan, 2008) asthma (Lanier et al., 2010), and chronic migraines (Goodwin, 2003; Black, 2011). One of the most consistent findings is that child maltreatment and adult intimate partner violence are risk factors for cardiovascular disease (Batten, 2004; Dube, 2009; Felitti, 1998; Pearson et al., 2004; Wegman, 2009).

In the seminal Adverse Childhood Experiences study (ACEs), 17,337 adults enrolled in Kaiser Permanente's HMO Health Appraisal Clinic in San Diego were interviewed extensively about childhood experiences and adult physical health during 1995-1997 (Felitti, 1998). Using the aforementioned ACEs questionnaire, Felitti and colleagues found a dose response relationship between the number of ACEs reported and self-reports of heart disease after controlling for smoking and physical inactivity. Additionally, compared to young adults with no history of adverse childhood experiences, respondents with four or more ACEs were twice as

likely to report subsequent heart disease. This initial study was the first to highlight traumatic childhood experiences as independent predictors of cardiovascular disease (Wegman and Stetler, 2009).

Dong and colleagues (2004) built on this initial finding by testing for the specific effect of maltreatment on cardiovascular disease. They found that sexual, physical and emotional abuse were each associated with nearly a two-fold increase in cardiovascular disease. They also found a 20% increase in cardiovascular disease for each additional ACE reported. Finally, there was a 2- to 3-fold increase in cardiovascular disease for participants reporting four or more types of ACEs. These results were independent of traditional risk factors such as smoking, hypertension, diabetes, physical inactivity and demographic characteristics. In a nationally representative sample of US women, a history of abuse or neglect in childhood was associated with a nearly 6-fold increase in cardiovascular disease (Batten, 2004). Goodwin and colleagues found sexual abuse is associated with an increased risk of cardiac disease (Goodwin, 2004). Similarly, Roman and colleagues (2002) found childhood abuse was associated with chronic heart problems in a community sample of women in New Zealand.

Though there are fewer published studies available, there is a similar link between intimate partner violence and cardiovascular diseases (Black, 2011). In an epidemiologic study of women ages 18 to 65 recruited from family practice clinics, Coker and colleagues found that women who report ever experiencing physical or psychological intimate partner violence were twice as likely to report cardiovascular problems (Coker, 2000). Five years later the authors conducted a follow-up study looking specifically at history of intimate partner violence and chronic health conditions. They found that the most commonly reported chronic condition among this same sample of women was related to heart or circulatory problems. Coker and

colleagues also found that both current intimate partner violence and past experiences with intimate partner violence were significantly associated with higher risk of cardiovascular diseases, though the association was stronger for current intimate partner violence (Coker, 2005). In one of the few studies examining an association between intimate partner violence for men and women, Brieding and colleagues (2005) conducted a large multistate study as part of the CDC's Behavioral Risk Factors Surveillance System (BRFSS) study, a random-digit-dialed telephone survey. They found a positive association between lifetime intimate partner violence victimization and a history of heart attack and heart disease for both women and men after adjusting for demographic and behavioral risk factors including smoking and Body Mass Index (Breiding, Black & Ryan, 2008).

There is a large literature base linking low childhood socioeconomic status to cardiovascular disease. In 2006, Galobardes and colleagues reviewed 24 prospective studies from Europe and United States linking childhood socioeconomic status to cardiovascular disease. They found significant inverse associations between socioeconomic status and cardiovascular disease with the excess risk attributable to low socioeconomic status ranging from 30 to 60 percent. When researchers adjusted for adult socioeconomic status these associations were attenuated but excess risk of cardiovascular disease still ranged from 20 to 40 percent (Galobardes et al., 2009). Past work linking economic disadvantage to and cardiovascular disease has not generally incorporated official definitions of poverty.

2.4 Inflammation and Cardiovascular Disease: Overview of the Literature

2.4.1 Inflammation

In the immune system, the first line of defense against foreign bodies is inflammation, a process that involves the migration of white blood cells from the bloodstream to the site of an

infection. These activities are coordinated by pro-inflammatory cytokines (e.g interleukin-6), which in turn induce rapid synthesis of C-reactive protein. Together pro-inflammatory cytokines and C-reactive protein 1) guide cells to the site of injury, 2) signal them to kill invading bodies and 3) signal them to repair damaged tissue (Sompayrac, 2008). The same inflammatory response that is triggered by physical infection is also triggered in response to psychological stressors and trauma (Sapolsky, 2000; Sompayrac, 2008). This connection between the immune and nervous systems is thought to be an adaptation from ancestral environments characterized by threats to survival and consequently reproductive fitness. From an evolutionary perspective, organisms that were able to anticipate physical threat by quickly mounting an inflammatory response prior to sustaining physical injury were more likely to heal wounds and clear infections. This ultimately meant a higher likelihood of survival and reproductive viability (Karatoreos et al., 2013). Inflammation is critical for survival because without such a response, minor infections and wounds would be lethal (Sompayrac, 2008). Persistently elevated levels of inflammation in the absence of infection (systemic inflammation), however, can contribute to the emergence of a variety of chronic conditions over the life course. Thus, in the past ten years, systemic inflammation has been investigated as a potential risk factor for cardiovascular disease (Pollitt et al., 2008; Ridker, 2004).

While there are a large variety of biological markers of inflammation, only a few, including C-reactive protein and IL-6, have standardized measurement and analytic methods and are currently used in both research and clinical settings (Pearson et al., 2003). The development of highly sensitive assays known as high sensitivity CRP (hsCRP), has allowed for the assessment systemic inflammation as a risk factor for cardiovascular disease (Nazmi & Victora, 2007). HsCRP is implicated in the initiation and progression of atherosclerosis, an inflammatory

disease that has been shown to predict cardiovascular disease in apparently healthy men and women (Blake and Ridker, 2001; Cesari et al 2003; Kuo et al., 2005).

Ridker and colleagues (1997) measured CRP in 1,086 healthy men participating in the Physicians Health Study, half of whom developed myocardial infarctions, stroke or venous thrombosis over a six year period of time. They found CRP levels at baseline predicted incidence of myocardial infarctions and stroke. Men in the highest quartile of CRP had 3 times the risk of myocardial infarction and two times the risk of stroke compared to men in the lowest quartile (Ridker et al., 1997). In 1998, Ridker and colleagues conducted a similar study, this time among women enrolled in the Women's Health study. They again found women who developed cardiovascular problems over the course of three years had higher CRP at baseline. Women in the highest quartile of CRP at baseline had a seven-fold increase in risk of myocardial infarction or stroke (Ridker et al., 1998). In both studies, CRP predicted cardiovascular disease independent of smoking, body mass index, hypertension and diabetes, lending support to CRP as an important risk factor for cardiovascular. Similar results were found among a population of healthy middle-aged men in Germany (Koenig et al., 1999), in a prospective cohort study men and women aged 70 to 79 in the United States (Cesari et al., 2003) and among middle-aged men and women in Iceland (Danesh et al., 2004). The study by Cesari and colleagues (2003) also assessed IL-6 and found baseline IL-6 levels significantly predicted incident coronary heart disease and stroke. Pai and colleagues (2004) also reported both CRP and IL-6 were significantly associated with an increased risk of coronary heart disease in both men and women (Pai et al., 2004).

Taken together, these studies suggest hsCRP levels in non-clinical samples may be indicative of increased risk for cardiovascular diseases. CRP in particular has also been shown to improve the predictive value of traditional risk factors including cholesterol (Ridker, 2003) and

blood pressure (Blake, 2003). These findings have prompted the CDC and the American Heart Association (AHA) to conclude that it is reasonable to measure hsCRP in addition to established risk factors in assessing risk of cardiovascular diseases (Pearson, 2004).

2.4.2 Child Maltreatment and Inflammation

There is evidence of elevated levels of inflammation in children exposed to traumatic stress. Slopen, Koenen and Kubzansky (2013) examined the effects of 5 types of adverse childhood events (spending time in foster care, physical abuse, sexual abuse and separation from parents) on CRP, one biological marker used to detect systemic inflammation has been shown to predict future cardiovascular disease independent of traditional risk factors (Pearson et al., 2003). They found that maltreatment occurring between the ages of six and eight was associated with higher levels of CRP at age ten and that maltreatment occurring in early childhood (one and a half years of age) predicted CRP at age fifteen. Danese and colleagues also found an association between maltreatment and inflammation but only for children diagnosed with depression (2010).

Ten studies have tested the effect of child maltreatment on inflammation as measured by CRP in adults. Of the ten, eight were cross-sectional (Bertone-Johnson et al., 2012; Carpenter et al., 2012; Gouin et al., 2012; Hepgul et al., 2012; Matthews et al., 2013; Rooks et al., 2012; Taylor et al., 2006; Tietjen et al., 2012;) and two were longitudinal (Danese et al., 2007; Danese et al., 2008). Using a prospective cohort of adults (age 32) in New Zealand, Danese and colleagues (2007) found that individuals with histories of maltreatment as reported by parents, observers and retrospective reports had higher inflammation levels compared to individuals without a history of maltreatment. In a second study using the same population, a conditional effect of child maltreatment was observed such that individuals with depression and a history of child maltreatment had higher inflammation levels (Danese et al., 2008). Bertone-Johnson and

colleagues (2012) found sexual abuse in adolescence was positively associated with inflammation in adulthood while sexual abuse in childhood did not predict inflammation in adulthood (Bertone-Johnson et al., 2012). Hepgul et al (2012) tested the association between maltreatment and inflammation in first episodic psychosis patients and healthy controls. They found that patients with a history of maltreatment had elevated levels of CRP compared to healthy controls. Patients without a history of maltreatment however, were not significantly different from controls. In a sample of women including migraine sufferers, Tietjen and colleagues (2012) found that adverse childhood experiences were associated with elevated CRP levels independent of migraine presence. Rooks and colleagues (2012) examined male twins and found that when analyzed as individuals, experience of trauma predicted elevated inflammation and from the risky families literature, Taylor et al., (2006) found low socioeconomic status and poor family environments predicted elevated CRP levels. Two studies did not find associations between childhood adversity and inflammation. Gouin et al., 2012 found among healthy individuals between the ages of 45 to 90, child maltreatment did not predict elevated inflammation levels as measured by CRP but that child maltreatment contributed to elevated levels of interleuikin-6 (IL-6) and tumor necrosis factor alpha (TNF-α), two other markers of systemic inflammation. Carpenter and colleagues (2012) on the other hand did not find associations between any form of maltreatment and CRP in a community sample of healthy adults.

2.4.3 Intimate Partner Violence and Inflammation

There is a burgeoning body of research linking intimate partner violence to inflammation. In the first study to examine the effect of intimate partner violence on inflammatory markers, Newton and colleagues (2011) studied a community sample of healthy postmenopausal women

who were either separated or divorced. They found that women who reported past exposure to intimate partner stalking had significantly higher CRP levels compared to women without a history of intimate partner violence. The effect of intimate partner violence remained even after adjusting for BMI and other known risk factors for CRP (Newton, 2011). Additionally, a minimum of ten years had elapsed since the women experienced intimate partner violence, lending support to the assertion that traumatic experiences continue to exert biological consequences long after the physical threat itself is removed. A few years later, Out and colleagues followed a convenience sample of women seeking assistance from domestic violence shelters for 2 years and found that 37-49% of the relatively healthy population had CRP levels above the clinically significant cutpoint of 3mg/L (Out et al., 2012). This is notable because in the general US population only approximately 25% of the population has CRP levels above 3 mg/L (Ridker, 2003).

2.4.4 Poverty and Inflammation

Low socioeconomic status in childhood, adulthood, or both is associated with elevated inflammation (Miller et al, 2009; Nazmi & Victora, 2007; Owen, 2003; Pollitt et al, 2008; Taylor et al., 2006). Miller and colleagues (2009) found adults raised in low-income households had greater IL-6 production compared to participants who were raised in higher income households. Additionally they found adults raised in low socioeconomic households showed reduced cortisol-mediated signaling, an indication of reduced sensitivity to the anti-inflammatory properties of cortisol (Miller et al., 2009). Taylor and colleagues also found adults raised in low socioeconomic status households show higher CRP and they explained the association was due in part to low psychosocial functioning (Taylor et al, 2006). Pollitt and colleagues (2008) examined the effects of cumulative life course and adult socioeconomic status on inflammatory

markers in adulthood. They found statistically significant associations between low education and social position and elevated inflammatory markers. Further, there was a documented gradient such that greater exposure to low socioeconomic status led to higher levels of inflammation (Pollit et al, 2008) and in a systematic review assessing socioeconomic influences on CRP, Nazmi and Victora (2007) found an overwhelming majority of a total of thirty-two studies found an inverse association between socioeconomic status and CRP (Nazmi & Victora, 2007).

If systemic inflammation is a potential mechanism that explains the epidemiologic link between experiences of traumatic events and risk of cardiovascular disease, what are the biological processes that result in elevated inflammation in survivors of traumatic event? The following section discusses the interactive pathways between the central nervous system, the endocrine system and the immune system to shed light on how exposure to traumatic stressors may lead to systemic inflammation.

2.5 Biological Mechanisms linking Traumatic Stressors to Inflammation

2.5.1 Physiologic Response to Stressors

Generally, organisms regulate and maintain various physiologic measures at a relatively stable state. Under this state of homeostasis, body temperature, blood pressure, ideal oxygen intake and so on are all maintained under optimal conditions (McEwen, 1998). Biologically, social and environmental stressors threaten homeostasis. Under stressful conditions the brain coordinates body wide changes in an effort to address perceived threats or challenges. This is the classic “fight or flight” response. Energy is quickly mobilized, cognition is sharpened, pain is blunted and heart rate and blood pressure increase in order to distribute energy to tissues (Segerstrom & 2004). Overall the stress response is critical for survival and is exceptionally

adaptive for dealing with acute or short-term threats and is critical for survival. Prolonged activation of the stress response system, however can be harmful, leaving individuals vulnerable to a variety of chronic diseases. The fact the stress response is triggered by the expectation of harm is particularly critical for victims of child maltreatment and adult intimate partner violence because violence within families is uncontrollable and victims often live in anticipation of further harm even when the physical threat is removed (Miller, Chen & Parker, 2011; van Voorhees & Scarpa, 2004). The stress response involves the nervous, endocrine and immune systems. All three work in concert to direct energy away from normal (homeostatic) activities into survival mode until the perceived threat passes. The following section describes each of the three biologic systems, the ways in which each adapts to threat and available empirical studies testing these pathways.

2.5.2 Sympathetic Nervous System (SNS)

The SNS orchestrates an immediate response to threat by eliciting the production of adrenaline. Increased levels of circulating adrenaline in turn increase heart rate, mobilize energy and dilate blood vessels to increase blood flow to target muscle tissue (McCrory, 2010). All of these physiologic changes occur within seconds and have a net effect of preparing the body with the metabolic energy as well as the muscular ability needed to flee from threat. When the threat of harm is chronic, the SNS remains activated (Phillips, 2015). While activation of the SNS is adaptive in the short term, chronic activation leads to dysregulation in normal heart rate and blood pressure in in individuals exposed to chronic traumatic stressors (Ridker, 2003). One review article found that traumatized children are often in a state of low-grade fear that is reflected physiologically as increased heart-rate, blood pressure and respiration (Mulvihill, 2005). Some studies report an association between child maltreatment and elevated heart rate in

adults (Taylor et al., 2004) while others find an effect on blood pressure, but not on heart rate (Larkin & Read, 2008; Luecken et al., 1998). In the long term, activation of the SNS is associated with the development of hypertension and the progression of cardiovascular disease (Georgiades et al., 1997; Lynch & Cicchetti, 1998).

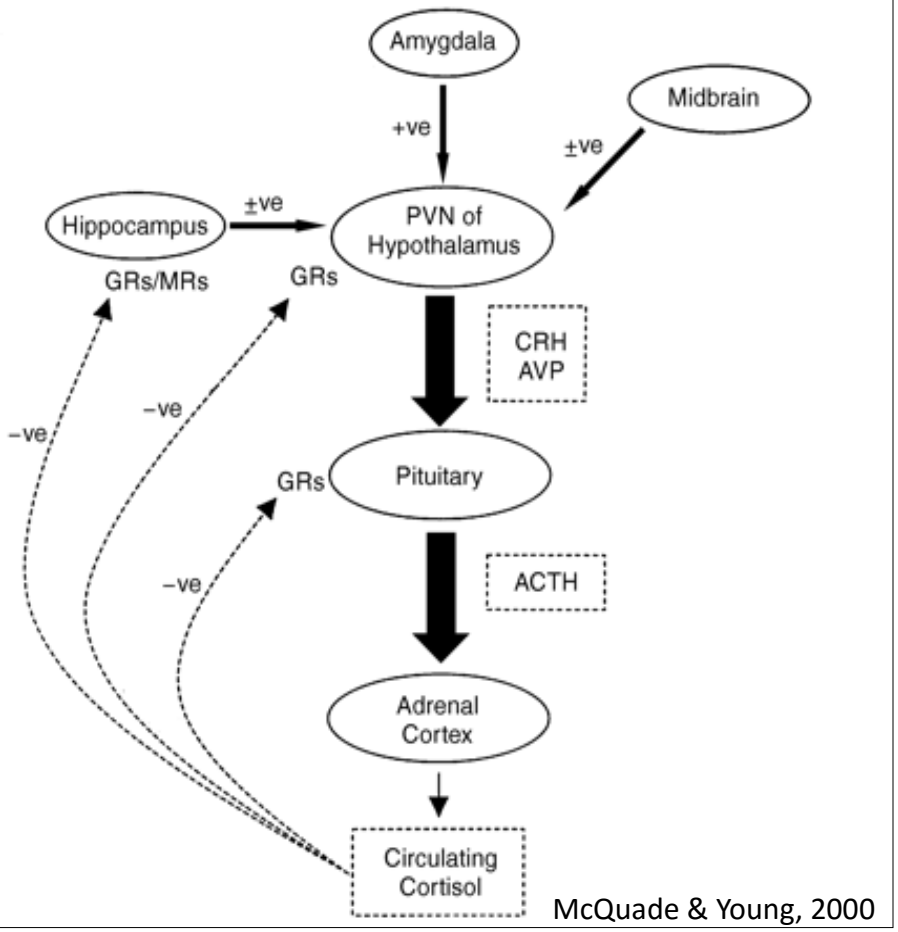
2.5.3 The Endocrine System (Hypothalamic-pituitary-adrenal [HPA] axis)

The second biologic system and perhaps the most widely studied system involved in stress response is the endocrine system. In the face of a stressor, signals from higher regions of the brain converge in the hypothalamus to signal the release of corticotropin-releasing hormones (CRH) (Figure 2.1). CRH travels from the hypothalamus to the pituitary and sets in motion the production of adrenocorticotropic hormone (ACTH). In turn, ACTH stimulates the adrenal gland to produce glucocorticoids (cortisol in humans and corticosterone in rodents), a hormone that plays an integral role in stress response. Glucocorticoids are the final product of the HPA axis and terminate stress response through a negative feedback loop at various levels of the system. When cortisol reaches peak levels it binds to receptor cells within the hypothalamus signaling the hypothalamus to stop producing CRH. This stops the pituitary gland from producing ACTH, which in turn stops the adrenal gland from producing more cortisol.

Cortisol, along with adrenaline released from the SNS, orchestrate the majority of changes that occur during the stress response. Under homeostatic conditions cortisol has a characteristic circadian rhythm whereby there is a gradual increase in the morning, followed by a gradual decrease over the course of the day (Gunnar & Donzella, 2002). Total cortisol production over the course of the day (basal cortisol) is typically low (Heim, & Nemeroff, 2001). Cortisol has two major roles during stress response. The first as described above, is to support and sustain the instantaneous changes orchestrated by the SNS and the second is to control the

length and magnitude of inflammation in the immune system (Sompayrac, 2008). During stress response, there is a sudden increase, as cortisol helps to increase oxygen intake, increase blood pressure and heart rate while diverting blood flow away from digestive and reproductive processes. This initial increase is followed by an abrupt decrease to baseline (Sapolsky, 2000; Gunnar & Quevedo, 2007). The initial release of cortisol increases energy and improves concentration, however continued exposure to cortisol has the opposite effect decreasing energy, decreasing the ability to concentrate, and increasing depressive mood (Scarpa, 2004). Though early research on the HPA-axis focused almost exclusively on increased reactivity of the HPA-axis, more recent work suggests dysregulation such that both under production of cortisol (hypocortisolism) and over production (hypercortisolism) are observed among individuals exposed to chronic traumatic stress (Cicchetti et al., 2001; Heim, Ulrike & Hellhammer 2000; Thaller et al., 1999; Yehuda, 2000). While the mechanisms leading to hypocortisolism are not yet clear it is hypothesized that the HPA-axis downregulates in response to prolonged exposure to stressors such that hypercortisolism is first observed. However, prolonged exposure leads to a downregulation at which point hypocortisolism is observed. (Miller, Chen & Zhou, 2007). Therefore observing an increase or decrease in production of cortisol may depend on the time between the onset of trauma and when cortisol is measured (Miller, Chen & Zhou, 2007).

Figure 2.1. The Hypothalamic-Pituitary-Axis (HPA)



Early evidence for trauma-induced disruption of the HPA-axis came from animal studies during which rat pups were removed from the mothers for brief periods of time during the first few days of life. When the pups were returned, the mothers engaged in extra care behavior (licking and grooming), however later tests revealed that these pups showed atypical corticosterone response to stress. Meaning, rather than showing the typical sudden rise in cortisol followed by a decrease to baseline levels, low overall basal levels, removed pups had blunted response to stressors and high basal cortisol levels compared to non-removed pups (Sapolspky, 1992). A series of studies involving children raised in orphanages in Romania and Russia found

overall high cortisol levels (Gunnar et al., 2001; Gunnar & Donzela, 2002). Sexually abused girls studied within six months of disclosure showed increased basal cortisol levels compared to non-abused controls that were matched on age, ethnicity, socioeconomic status and family composition (De Bellis & Putnam, 1994). A second group of studies on the other hand found evidence for chronically low levels of cortisol. Dozier and colleagues (2006) for example, found that more than half of a sample of children in foster care showed low levels of cortisol over the course of a day (Dozier, Manni et al., 2006). Chronically low or chronically higher cortisol levels leads to inefficient controlling of the magnitude and length of inflammation. In this way, the body attempts to re-establish homeostasis, chronic activation of stress response prevents a return to homeostasis. The physiological system then reorients itself its basal patterns of homeostasis. In other words, for individuals with a history of chronic traumatic stressors, dysregulation rather than homeostasis becomes the norm.

Among adults with a history of child maltreatment, Van der Vegt and colleagues (2009) found higher levels of cortisol in maltreated groups compared to participant with no such history (Van der Vegt, 2009). Heim and colleagues (2000) and Carpenter and colleagues (2007) both found chronically low levels of cortisol production among adults maltreated as children compared to adults with no history of maltreatment. Similar increases in cortisol have been found among victims of intimate partner violence (Black, 2011) with some noting specific patterns cortisol production such that compared to non-abused women, those exposed to intimate partner violence had higher evening and morning cortisol (Pico-Alfonso et al., 2004). Still others found higher cortisol only among victims or intimate partner violence who also suffered from PTSD (Inslicht et al., 2006).

2.5.4 The Immune System

As discussed above, cortisol plays an important role in regulating the magnitude and length of inflammatory response. To control inflammation, cortisol binds to glucocorticoid receptors on immune cells, thus the receptors' ability to "hear" the inhibitory action of cortisol is fundamental. Under stressful conditions during which basal cortisol levels are chronically high, the body down-regulates the number of cortisol receptors in an attempt to maintain homeostasis, effectively decreasing sensitivity to cortisol (Raison, 2003). The result is that while there is an abundance of cortisol, immune cells are unable to "hear" it (Miller, 2007). This process of glucocorticoid insensitivity reduces the ability of cortisol to suppress inflammatory response thus contributing to elevated levels of inflammation (Raison, 2003). Chronically low levels of cortisol can also contribute to high inflammation because in this case, immune cells cannot "hear" cortisol's anti-inflammatory signals, not because of desensitization but because the overall cortisol levels are low (Raison, 2003). Inflammatory response is therefore contained in the stress response system such that dysregulation in the form of either hypocortisolism or hypersortisolism leads to a failure to effectively control inflammation (Gunnar and Quevedo 2007).

2.6 Overview of the Influence of Gender on Child Maltreatment, Intimate Partner Violence and Inflammation

2.6.1 Gender Differences in Exposure to Child Maltreatment and Intimate Partner Violence

Research has documented gender differences in the risk of experiencing child maltreatment and intimate partner violence. For example, physical abuse is experienced more frequently and with greater severity among boys compared to girls (Thompson, Kingree & Desai, 2004). Young girls have about three times higher risk of experiencing sexual abuse than boys (Putnam, 2003) and sexual abuse among young girls is also more severe (Ullman &

Filipas., 2005). Leaders of the feminist movement were the first to call attention to intimate partner violence as a rampant problem and focused almost exclusively on intimate partner violence perpetrated by men. Newer evidence suggests men and women often perpetrate violence against each other and perpetration by both partners within a relationship, known as reciprocal intimate partner violence, is common (Whitaker et al., 2007). For example, in the National Survey of Families and Households, Brush found that in approximately half of intimate partner violence cases, violence was reciprocal (Brush, 1990). Similar results were found in a nationally representative dataset (Whitaker et al., 2007) and a meta-analysis found a woman's perpetration of violence was the strongest predictor of her becoming a victim of intimate partner violence months later (Golding, 1999). Reciprocity of violence however does not mean that severity of violence is equal between partners. Compared to men, women are seven to ten times more likely to report being beaten up or threatened with a weapon (Tjaden & Thoennes, 2000). Taking gender difference into consideration, Archer (2000) conducted a meta-analysis of eighty-two reports of aggression between heterosexual partners. He found although women used physical aggression against men nearly as often as men did against women, women reported injuries more frequently (62%) than men (39%) (Archer, 2000).

2.6.2 Gender Differences in Consequences of Child Maltreatment and Intimate Partner Violence

Prior research also suggests the effect of maltreatment and intimate partner violence mental health outcomes such as depression may also differ by gender. Gershon, Minor & Hayward (2008) outline various mechanisms that may lead conditional effects of child maltreatment and intimate partner violence on depression. First, gender differences may depend on the subtype of maltreatment under study and, second, young girls may internalize and blame themselves for abusive experiences more than boys so that in general child maltreatment leads

more often to depression in adult women compared to men (Thompson et al., 2004; Weiss et al., 1999). Romito and Grassi (2007) found evidence suggesting although the effects of physical abuse on depression were similar for college age men and women the effects of sexual violence on depression was larger for college age women.

In contrast, Fletcher (2009) noted gender differences such that physical abuse increased long term depression for women while sexual abuse increased long term risk of depression for men. Garnesfski & Arends (1998) found young boys who were sexually abused made significantly more suicide attempts compared to sexually abused girls (Garnefski & Arends, 1998). Although population level rates of sexual abuse are higher among women compared to men and that the female to male ratio among adults seeking medical attention is even higher (Heise, Elsberg & Gotmoeller, 2002), rates of reported sexual abuse, more so than emotional and physical abuse, are influenced by a gendered social context suggesting boys are less likely to disclose sexual abuse than girls (Ullman & Filipas, 2002). This possibility is supported by a study of young men and women with substantiated cases of sexual abuse that found similar levels of depressive symptoms (Ketring & Feinhauer, 1999).

With respect to intimate partner violence, the evidence is more mixed. One study found however that although women are more likely to report poor physical and mental health, however intimate partner violence is associated with physical and mental health for both men and women (Coker et al., 2000). Brieding (2005) found women and men who reported intimate partner violence were more likely to report a range of physical health problems including asthma and joint disease however only intimate partner violence among women was associated with heart disease and stroke. In a household probability sample, Caetano and Cunradi (2003) found female to male victimization was the strongest predictor of depression and reasoned this may be

due to individual reactions to men being victimized by women.

2.6.3 Gender Differences in Inflammation

Prior research shows gender differences in inflammation and immune functioning are related to biological differences between men and women (Pergola et al, 2008). Compared to men, women generally show greater changes in immune functioning in response to psychosocial stressors. This difference is hypothesized to be advantageous for battling infection however could be detrimental when sustained at elevated levels. Numerous studies consistently show women have higher CRP levels compared to men (Herd et al, 2012; Pearson, 2003; Rietzschel et al, 2012).

2.7 Other Risk Factors for Inflammation

2.7.1 Smoking and Body Mass Index (BMI)

Behaviors may also help explain the pathway between child maltreatment, intimate partner violence and inflammation. This is because exposure to child maltreatment and adult intimate partner violence may lead individuals to adopt unhealthy behaviors including smoking, physical inactivity and/or sedentary lifestyles, all of which are known risk factors for inflammation (Ridker et al., 2003; Pearson et al., 2003). Individuals reporting maltreatment in childhood are significantly more likely to ever smoke, to be current smokers as well as heavy smokers (Anda et al., 2009; Gilbert et al., 2009) and victims of intimate partner violence are also more likely to be current or former smokers (Bonomi et al., 2006). Some speculate that victims of trauma may initiate and continue to smoke due to the psychoactive benefits in regulating mood (Anda, 2006). Victims of child maltreatment and adult intimate partner violence are also more likely to have high levels have high (BMI) or be obese (Felitti et al., 1998; Gilbert et al., 2009; Noll, Zeller, Trickett, & Putnam, 2007). Among women ages eighteen to fifty, having one

or more adverse childhood events is associated with increased BMI (Tietjen et al., 2012) and victimization by an intimate partner has been found to be related to weight gain and higher BMI (Health et al, 2013). It is also worth noting prior research consistently shows socioeconomic status is inversely associated with smoking (Anda, 1999; Herd et al., 2012), poor dietary habits and inactive lifestyle (Cunradi, 2000; Matthews et al., 2014; Yudkin et al., 1999).

It has been shown that an inflammatory process is present in visceral fat commonly associated with higher BMI and numerous studies establish BMI as a standard covariate of CRP (Black, 2006; Everett et al., 2014; Carpenter et al., 2012) with some reporting a greater magnitude of the association in women compared to men (Choi, Joseph & Pilote, 2012). Current smoking is also an established risk factor for inflammation (Ridker et al., 2003)

2.7.2 Race/Ethnicity

Studies have consistently demonstrated patterning of CRP along racial lines (Gruenwald et al, 2009; Herd et al, 2012). A study of participants enrolled in the Women's Health Study found African American women had significantly higher median CRP concentrations compared to White, Hispanic and Asian participants (Albert et al., 2003). In the Dallas Heart Study, African American women had the highest mean concentrations of CRP (Kiera et al, 2005). Danner and colleagues also found African Americans were higher risk for elevated CRP compared to Whites although a Ford and colleagues found no association between race/ethnicity and CRP (Ford et al, 2003). It has also been shown in large population studies that Asians have lower CRP compared non-Asians (Kao et al., 2006).

2.7.3 Depression

There is mounting evidence to support a link between both maltreatment in childhood, adult intimate partner violence and poor mental health outcomes (Black, 2011; Felitti et al.,

1998; Miller Chen & Parker, 2011; Putnam, 2003; Wright et al., 2009). Depression, a measure of psychological distress, is by far the most prevalent mental health consequence of child maltreatment and intimate partner violence across all age groups (Campbell, 2002; Coker et al., 2000; Golding, 1999; Newman et al., 2000; Sansone, Wiederman & Sansone, 1997).

An association between physical and sexual abuse and depression has been documented using self-administered questionnaires (Batten et al., 2004; Dunn et al., 2013; Harris, 2009; Mueller, 2009; Newman et al., 2000) and among samples of substantiated cases of abuse (Care, 2001; Halfon, 1992; Wells & Guo, 1999; Woods, Farineau & Mowey, 2013). Using data from a birth-cohort in New Zealand, Fergusson and colleagues found that contextual factors largely explained the association between physical abuse and depression among young adults but that the association between sexual abuse and depression remained, accounting for approximately thirteen percent of mental health problems in the sample (Fergusson et al., 2008). In a nationally representative sample of adolescents from the United States, Fletcher (2009) also found that victims of physical and sexual abuse during childhood were more likely to be depressed in adolescence than non-physically abused controls (Fletcher, 2009). The Fletcher study is particularly salient because he found that although family, school and neighborhood level confounders explained a large proportion of the association between child maltreatment (physical and sexual abuse) and depression for adolescents, the strength of association among adults remained robust. This finding suggests that consequences of maltreatment on depression may become larger over time.

Emotional abuse has received less attention in the literature, however, existing work suggests emotional abuse predicts depressive symptoms (Hart & Glaser 2011; Wright, Crawford & Del Castillo, 2009). In a prospective study of college students, emotional abuse prospectively

predicted shorter time to the development of new depressive episodes (Liu, Alloy et al. 2009), controlling for other sub-types of maltreatment. Others have found that emotional abuse may have a stronger relationship to psychological functioning than physical and sexual maltreatment (Higgins & McCabe, 2003; Mullen et al., 1996; McGee et al., 1997). Spatz et al., (2007) for example found that emotionally abused children were at increased risk for current depression compared to those who had suffered from other sub-types of maltreatment (Spatz et al., 2007).

A similar link between intimate partner violence and depression has also been documented. A telephone survey of women enrolled in a large health plan, found that compared to women with no history of intimate partner violence, women reporting recent physical and/or sexual violence in intimate relationships were four times as likely to report severe depressive symptoms and nearly three times as likely to report minor depressive symptoms (Bonomi et al, 2012). Since most of these studies are cross sectional, the temporality of these associations is not always clear. For instance, some survivors of intimate partner violence may have had depression that was exacerbated by the additional stress of violence in an intimate partner relationship, however a handful of longitudinal studies show first episodes of depression can be triggered by experiences of violence in intimate relationships. Although not yet formally tested, some argue that gender differences in incidence of depression may be attributable to differences in experiences of child maltreatment and intimate partner violence.

Many of the biological responses to traumatic stressors such as dysregulation of the HPA-axis and its subsequent link to inflammation is also observed in individuals with depressive symptoms as well as those diagnosed with major depressive disorder (Kaufman & Charney, 2001). Miller and colleagues (2002) matched a community sample of depressed individuals to healthy controls and found, individuals with depression had significantly higher levels of CRP

and IL-6 compared to controls. Similar findings of higher inflammation among depressed groups have been reported in non-psychiatric community samples (Dentino et al., 1999; Kop et el., 2003; Suarez et al., 2003). In a nationally representative sample of US adults age eighteen to thirty-nine, Ford and Erlinger (2004) found a history of major depression was associated with higher CRP and that the association was stronger for women compared to men (Ford & Erlinger, 2004).

2.7.4 Age

As previously discussed, inflammation is a fundamental and necessary response to internal and external threats. Generally, inflammation is positive and protects against infections. Activation over the life course, leads to an increase in systemic inflammation. This process of "inflammaging" is thought to be partially responsible for the higher prevalence of chronic conditions at older ages (Franceschi, 2007). Across literature examining correlates of inflammation, a general trend is established such that older populations have higher mean CRP levels compared to younger ones.

2.8 Gaps in the Literature

Among studies examining inflammatory consequences of child maltreatment in healthy adults, three were convenience samples (Carpenter et al., 2012; Gouin et al., 2012; Rooks et al., 2012) and the majority of studies were among older adult populations. The one study among healthy young adults also used a convenience sample (Hepgul et al., 2012), making it difficult to generalize findings to the US population. Only one study examined the deferential impact of specific sub-types of maltreatment however it was among a group of older women (Bertone-Johnson et al., 2012), therefore there is a need to improve our understanding of the effects of specific subtypes of maltreatment on inflammation among younger populations and among men

and women. The literature examining the effect of intimate partner violence on inflammation in particular is in its infancy, therefore there is a need to extend the literature, especially among young adults. Moreover, none of the studies tested whether the effect of trauma on inflammation is contingent on gender. Given gender differences in the incidence and consequences of maltreatment and intimate partner violence it is important to understand a potential interaction between trauma and gender. Also missing in the literature are studies using more nuanced operational definitions of poverty. There is a need to better understand the ramifications of timing and chronicity of poverty as well as differences in inflammatory risk relating to upward or downward income. The present dissertation seeks to address these limitations in the literature guided by four major theoretical frameworks from the biological, psychological and sociological traditions. Chapter 3 presents an overview of the theories used to guide the dissertation followed by a synthesis of all four in a conceptual model for this study

CHAPTER 3

THEORETICAL FRAMEWORK AND CONCEPTUAL MODEL

3.1 Introduction

This dissertation is guided by four theories. The first two relate psychosocial experiences to biological processes. The biological embedding of childhood adversity model (Miller, Chen & Parker, 2011) is in the tradition of latent influences (Hertzman & Boyd, 2010) in that it posits traumatic experiences during sensitive developmental periods may have enduring effects on the immune system in a way that influences health years later net of intervening experiences. The psychobiology of stress model (Kemeny, 2003) describes biological consequences of exposure to trauma in adulthood. With the recognition that exposure to stress is not randomly distributed, the present dissertation is also informed by the fundamental causes theory and the stress process model, both of which describe how social class shapes exposure to and experiences of trauma. Taken together these four theoretical perspectives provide a framework for first understanding how experiences of victimization within violent, tumultuous families or intimate relationships is shaped by structural forces and second how such experiences "get under the skin" to confer vulnerability to disease.

3.2 The Biological Embedding of Childhood Adversity Model

The Biological Embedding of Childhood Adversity model (biological embedding) describes how traumatic experiences that occur during critical periods in childhood become biologically embedded (Miller, Chen & Parker, 2011). The model is a synthesis of ideas from various disciplines including life-course epidemiology (Lynch & Smith, 2005), stress physiology (McEwen, 1998), socio-emotional development (Repetti, Taylor & Seeman, 2002) and behavioral immunology (Raison & Miller, 2003). According to the biological embedding model,

sensitive periods in the development of the brain and other biologic systems begin in utero but continue to develop through childhood and adolescence. The development of these systems is mediated through caregivers and the home environment. As described above, traumatic experiences can lead to chronic activation of the stress response system in a way to chronically elevated inflammation levels. Further, chronic activation in childhood in particular disrupts developing biological systems such that elevated inflammation (pro-inflammatory phenotype) is observed well into adulthood. In this way, trauma becomes programmed or embedded in immune cells.

Miller and colleagues explain that trauma becomes embedded in immune cells through predictive adaptive response (PAR), or the ability to adapt to unexpected environmental circumstances and ultimately increase survival (Gluckman & Hanson, 2006). PARs are biological adaptations made in response to environmental stressors during sensitive periods of development and typically become embedded in physiology. A pro-inflammatory PAR for example allows an organism to heal wounds quickly, thus aiding in survival in a physically threatening environment.

Physiological stress response is often moderated by psychological factors and children also develop the emotional and social mechanisms needed to regulate stress in early life. As articulated in the risky families literature (Taylor et al., 2006), children are born with heightened abilities to sense danger so that any sudden change is perceived as dangerous and therefore triggers stress. Over time, however children, aided by caregivers learn to moderate these signals of stress on their own so that for example sudden noises no longer elicit physiologic stress response. In general, the ability to manage stressors, that is emotion regulation, is essential for managing whether challenges are perceived as are stressful. Children living in families

characterized by high levels of conflict or aggression protect themselves by remaining hypervigilant, as a result the development of emotion regulation skills is disrupted (Chen & Miller, 2012). Consequently, children may be deficient in expressing and controlling emotions, becoming aggressive or withdrawn when confronted with challenges (Taylor et al., 2004). Consistent with this articulation, Repetti and colleagues (2002) found that children from risky families have difficulty recognizing their own emotions and the emotions of others. Recall that when threats are perceived the hypothalamus signals the stress response by producing CRH. The end result is that the stress systems are routinely activated in response to everyday challenges, further exacerbating proinflammatory tendencies.

As children grow up, pro-inflammatory responses become exacerbated through behavior changes. Adults may become mistrustful and hypervigilant for threat, which shapes the way they relate to others. They may interpret routine experiences as stressful which in turn continues to trigger an already dysregulated physiologic system (Heim, 2000; Hertzman, 1999). They may find themselves in conflict-ridden situations. Behaviorally, lack of self-control may lead individuals to overcompensation with negative health behaviors such as smoking and poor diet. These psychological vulnerabilities and negative health behaviors work to exacerbate pro-inflammatory tendencies already programmed into cells by childhood trauma. Though this model draws on processes described in biologic stress response as described above, the central premise is the programming of pro-inflammatory phenotypes as a result of childhood traumatic experiences. This pro-inflammatory phenotype lasts well into adulthood even in the absence of cumulative stress.

3.3 The Psychobiology of Stress Model

The psychobiology of stress model also builds on the general understanding of biologic response to psychosocial stress by explaining that not all stressors elicit the same set of physiologic changes and that rather than one uniform stress response, there are specific patterns of biologic response depending on the way a particular stressor is appraised (Kemeny, 2003). Stressors such as intimate partner violence that are appraised as threats, that is, the perception that demands by a particular stressor outweigh resources or that a stressor is uncontrollable may lead for example to psychobiologic responses (e.g. withdrawal) that support the uncontrollable nature of the event and ensuing affective state (e.g. depression) which in turn lead to physiologic changes. Stressors that are appraised as challenges, that is resources meet or outweigh demands of the stressor, may lead to physiologic responses that support active coping processes. According to this model appraisal of intimate partner violence (a threat) leads to specific patterns of biologic reactivity that are distinct from events appraised as challenges. The psychobiology of stress model also focuses on the relationship between a specific biological marker and its influence in the etiology of a particular disease or condition. The rationale is that in order to understand the effect of psychosocial factors on specific disease outcomes it is first critical to establish physiological mediators that lead to the outcome of interest. In this case, adult intimate partner violence (a threat), leads inflammation (a biological mediator) that leads to risk of cardiovascular disease (the disease outcome).

3.4 Persistent Advantage/Disadvantage over the Life Course: Fundamental Causes and the Stress Process Model

The biological embedding and psychobiology of stress models present the ways in which traumatic experiences and psychosocial processes lead to dysregulation in inflammatory processes, the former during sensitive periods in childhood and the latter during adulthood.

These processes, however, occur within the broader social environment. It has long been established that social conditions play an important role in quality of life. There is a long scientific tradition establishing gradients in socioeconomic status and health such that as socioeconomic status decline so too does an individual's health (Adler, 1994). Across diverse health outcomes, individuals with lower income and lower levels of educational attainment are at greater risk for poor health compared to higher income and highly educated counterparts, and suffer from the excess morbidity and mortality associated with it (Feinstein, 1993; Pappas et al., 1993; Robert & House, 1994). The associations extend from relatively minor illnesses to life-threatening conditions and from the implications of childhood socioeconomic status (Galobardes et al., 2004) to socioeconomic status in adulthood (Preston & Taubman, 1994).

The theory of fundamental causes articulates ways in which the environments within which individuals live and work shape decisions people make to directly impact availability of health promoting options that persistently influence health gradients over time (Link & Phelan, 1995; Link & Phelan, 1996; Lutfey & Freese, 2005; Phelan et al., 2004). According to the theory, a fundamental cause of health outcomes has four components. First it affects multiple disease outcomes and second, it does so through multiple risk factors. Third, fundamental causes influence access to resources that may buffer risk and lastly the association between fundamental causes and health persists over time (Phelan, Link & Tehranifar, 2010). Those who are able to protect themselves from any given health condition, are individuals with the ability to draw on traditional indicators of social position such as income, education and knowledge, power and prestige and also on more intangible resources often derived from social connections (Link & Phelan, 1995). For example, families may share information as part of parent support groups or at the neighborhood level, monitor each other's children. Link and Phelan (1995) theorize at

socioeconomic status is a fundamental cause of health because individuals who are able to avoid health compromising risks are precisely those who have access to flexible resources.

In the context of child maltreatment, experiences of chronic abuse in childhood may lead to withdrawal that might then affect school attendance and ultimately educational attainment. A lack of educational attainment may eventually lead to low paying jobs, an inability to access health care services and residence in neighborhoods characterized by crime. In this way, fundamental causes are linked at the individual, family and neighborhood levels, translating one risk into another. Phelan, Link & Tehranifar (2010) term this effect the "risk of risks" or "causes of causes". Based on prior research showing that socioeconomic status tends to remain stable over time (Hertzman, 1999), Hayward and Gorman (2004) further conceptualize and differentiate the ways they further argue that socioeconomic in which socioeconomic status influence health over the lifecourse. They theorize that socioeconomic status in childhood strongly influences adult socioeconomic status in that resources within the family of origin dictate the ability to invest in educational opportunities and other status promoting resources (Hayward & Gorman, 2004). This assertion is further supported by studies showing families with greater economic resources are better able to make significant investments in the development of children while economically disadvantaged families tend to divert material resources into more immediate family needs (Bradley & Corwyn, 2002; Duncan & Magnuson, 2003). The duration of time spent in socioeconomically disadvantaged or advantaged households may confer even greater risk.

Within this broader conceptualization of social conditions as fundamental causes of health, the stress process model offers more specificity in that it also focuses on exposure to stressors and resources that may mediate or moderate consequences of stressors (Pearlin,1981).

According to this model, exposure to stressors is not random but rather differentially distributed along racial, socioeconomic and gendered lines (Pearlin, 1981; Pearlin, 2013; Turner, 2009). Further, available resources may serve to mediate or moderate stressors (Aneshensel & Mitchell, 2014). It is the articulation of unequal distribution of exposure to stressors that is most pertinent to the present dissertation. The degree of exposure to stressors is influenced by the socioeconomic context within which individuals live (Taylor & Repetti, 1997). Paradoxically, these same individuals may also lack the education, social connections as well as the material resources needed to cope with risks compared to higher income groups (Drake & Pandey, 1996; Link & Phelan, 1995). For both maltreatment and intimate partner violence, this may mean an inability to access a broad range of resources—basic health care services, knowledge of and access to various coping resources—that could minimize the consequences of violence within the family. Moreover, the few resources available may be further depleted by enduring or repeated exposure to stressors, potentially increasing the deleterious effects of trauma within the family (Pollitt et al., 2008).

In the literature examining the effect of violence within the home on health, socioeconomic status is typically conceptualized as a chronic stressor. Low socioeconomic status however may also compound the effects of trauma on health. Conflict related to finances has been shown to increase the risk of adverse health consequences associated with intimate partner violence (Jewkes, 2002; Coker, Weston, Creson, Justice, & Blakeney, 2005). In a cross-sectional study, Schreier et al., (2014) tested whether family chaos influenced adolescents' inflammatory profiles and whether adolescents from low socioeconomic environments were at higher risk for experiencing elevated levels of inflammatory (Schreier et al., 2014). They found

that the impact of inflammation was greater for individuals in lower socioeconomic groups compared to their well-off counterparts.

3.5 Conceptual Model

Informed by the previously discussed empirical literature (see Chapter 2) and theoretical frameworks (see Chapter 3), the conceptual model depicting the constructs and relationships assessed in the present dissertation is shown in Figure 3.1. The conceptual model shows the potential pathways for understanding the relationship between emotional abuse in childhood, intimate partner violence in adulthood and experiencing poverty over the life course. The arrows depicted in black are specifically tested in this dissertation while arrows shown in gray are depicted for completeness. From left to right, demographic characteristics (age and race/ethnicity) and family background characteristics (parents' income and education) are conceptualized as temporally preceding and influencing emotional abuse in childhood, intimate partner violence in adulthood as well as poverty status over the lifecourse. Demographic and family background characteristics also have a direct influence on CRP as well as exerting indirect influence through young adults' own socioeconomic characteristics, through lifestyle factors such as smoking and BMI as well as through depressive symptoms. Lastly the model includes the use of anti-inflammatory drugs, a known risk factor for elevated CRP levels.

Aim 1

In the growing literature examining biological consequences of child maltreatment few studies explore the direct contribution of emotional abuse over and above the effects of physical and sexual abuse. With the understanding that men and women differ in their experiences of as well as consequences of emotional abuse (Thompson et al., 2004; Putnam, 2003; Ullman et al., 2005), Aim 1 tests the biological embedding model in that the enduring effect of emotional abuse

in childhood on CRP is tested. Aim 1 first tests for an association between emotional abuse in childhood and CRP and second tests whether the effect of emotional abuse on CRP is contingent on gender. Childhood socioeconomic status, demographic and familial characteristics are taken into account to rule out alternative theoretical explanations. Smoking, depressive symptoms and BMI are known risk consequences of emotional abuse as risk factors for high CRP. Each of the above variables are expected to attenuate, but not eliminate, the effect of childhood emotional abuse on CRP.

Aim 1 hypotheses: Levels of CRP are expected to be higher among young adults with a history of emotional abuse. The association between emotional abuse and inflammation is expected to be contingent on gender such that emotionally abused women have higher CRP levels compared to emotionally abused men. Because different types of maltreatment tend to co-occur, physical and sexual abuse are expected to attenuate but not eliminate the association between emotional abuse and inflammation. Childhood socioeconomic status, age, race/ethnicity, young adult socioeconomic status, health behaviors and health status are all expected to attenuate the effect of emotional abuse on inflammation.

Aim 2

Aim 2 tests the psychobiology of stress model in that the effect of experiencing intimate partner violence in adulthood on inflammation is tested (Kemeny, 2003). Similar to Aim 1, gender is conceptualized as a moderator of the effect of intimate partner violence on systemic inflammation. Aim 2 first tests for an association between adult intimate partner violence and CRP. Experiences of emotional, physical and sexual abuse, childhood sociodemographic characteristics and young adult sociodemographic characteristics are included as alternate explanations of the association between intimate partner violence and inflammation. Depressive

symptoms, smoking and BMI are known consequences of child maltreatment and since all three are associated with increases in systemic inflammation, these variables are included as risk factors for systemic inflammation along with the use of anti-inflammatory drugs (Higgins, 2003; Miller, Chen & Zhou, 2007; Pearson et al., 2003).

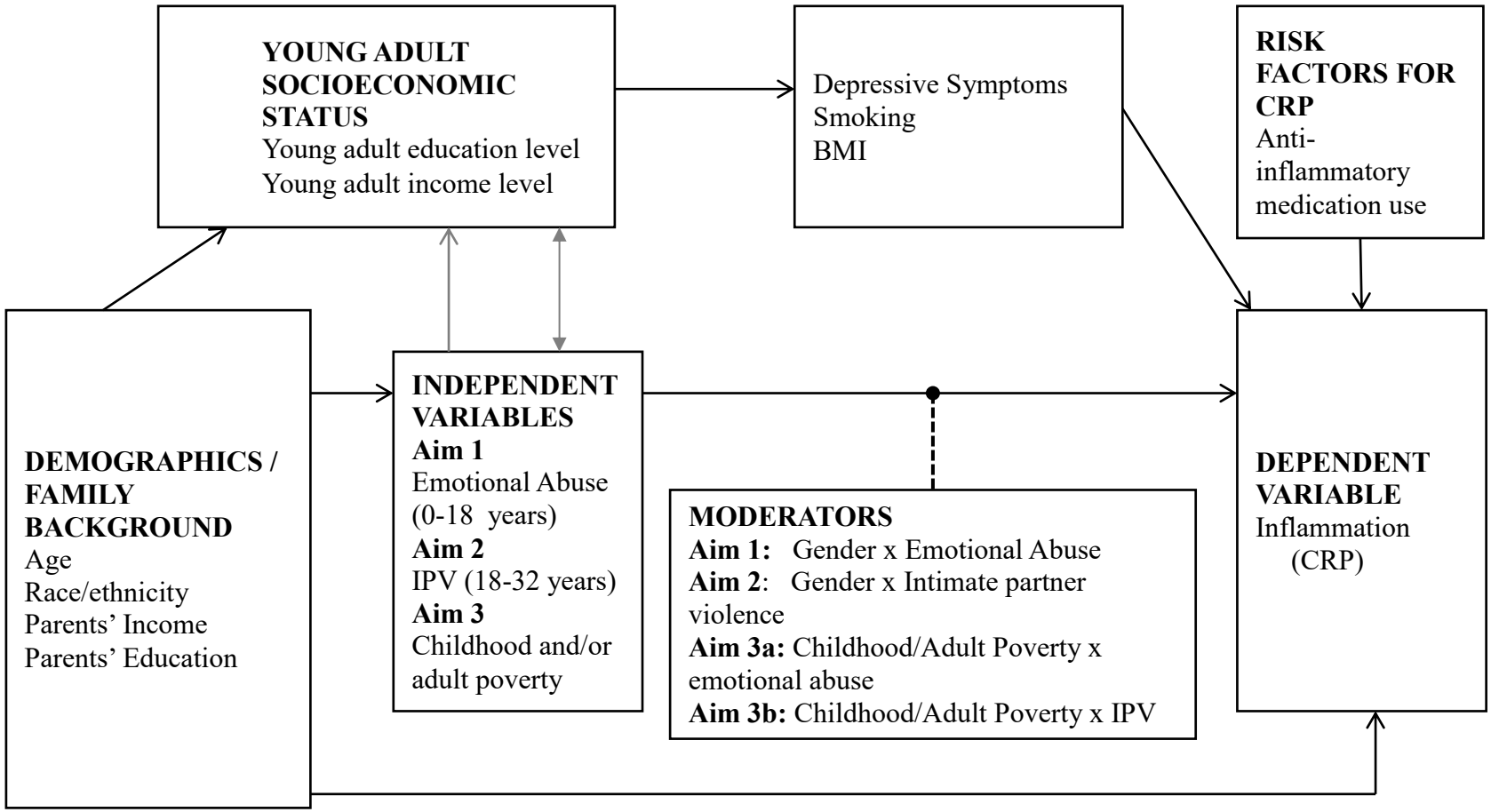
Aim 2 hypotheses: Levels of inflammation are expected to be higher among young adults with a history of intimate partner violence. The association between intimate partner violence and inflammation is expected to be contingent on gender such that women with a history of intimate partner violence have higher inflammation levels and compared to men with a history of intimate partner violence. Experiences of emotional, physical and sexual abuse, childhood sociodemographic characteristics, young adult socioecomonic status, health behaviors and health status known to be risk factors for inflammation are all taken into account and are expected to attenuate, but not eliminate, the effect of intimate partner violence on inflammation.

Informed by the fundamental causes and stress process theories, Aim 3 examines the experience of poverty as a social condition in relation to inflammation. Using a dynamic measure of poverty status over time, this Aims seeks to address 1) whether childhood is a sensitive period during which exposure to poverty can have lasting physiological effects and 2) whether the chronicity of poverty (i.e. persistent poverty) is more consequential for inflammation 3) the ways in which poverty status may compound the effects of emotional abuse and adult intimate partner violence on inflammation.

Aim 3 hypotheses: Because poverty status is associated with differential access to "flexible resources" over time, Young adults who experienced poverty in childhood and adulthood (persistent poverty) are expected to have the highest inflammation levels while those who never experienced poverty will have the lowest. The association between persistent poverty

and inflammation is expected to be contingent on emotional abuse such that the impact of childhood poverty on inflammation is greater for young adults who also experienced emotional abuse. Lastly, the association between persistent poverty and inflammation is expected to be contingent on intimate partner violence such that the impact of persistent poverty on inflammation is greater for young adults who also experienced intimate partner violence.

Figure 3.1 Conceptual Model of Relationship between Child Maltreatment, Intimate Partner Violence, Childhood/Adult Poverty Status and Inflammation



Notes: Childhood and/or adult poverty was calculated using the following variables: parents' income (adjusted for inflation), childhood household size, young adult income and young adult household size.

CHAPTER 4

RESEARCH DESIGN AND METHODS

4.1. Overview

This chapter presents the research methods used in this study. Secondary data analysis of the National Longitudinal Study of Adolescent to Adult Health (Add Health) was conducted to examine the influence of childhood maltreatment and young adult intimate partner violence on systemic inflammation. This on-going national survey of health-related behaviors from adolescence through young adulthood is well suited for this study because it allows for the examination of childhood as well young adult experiences that ultimately influence health. This chapter describes the Add Health study, outlines the derivation of the analytic sample, operationalizes key constructs, and describes analytic plans for each aim of this dissertation.

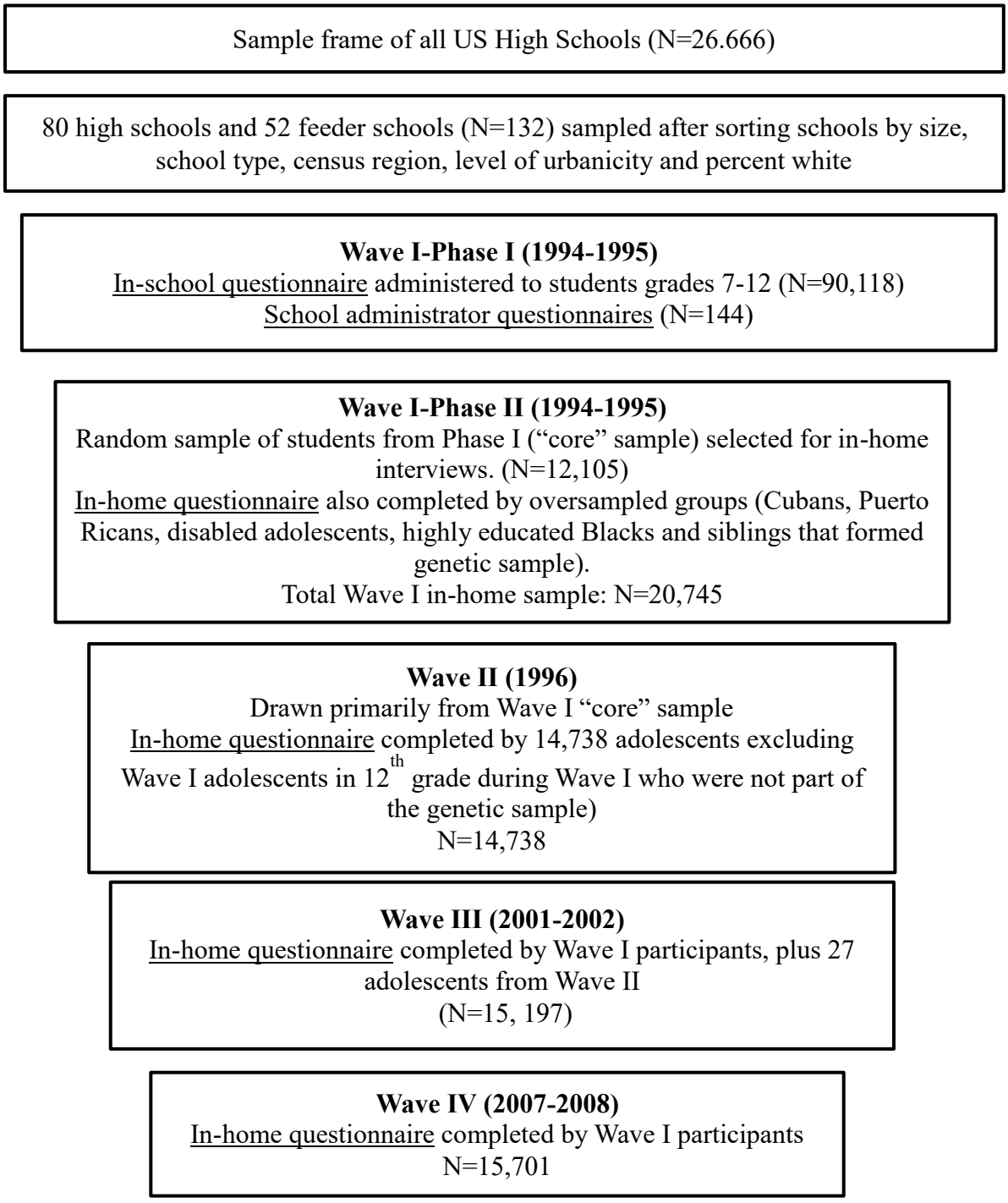
4.2. National Longitudinal Study of Adolescent to Adult Health (Add Health)

Add Health is a longitudinal study of a nationally representative sample of adolescents enrolled in grades 7-12 in the 1994-95 academic year (Chantala, 2006). The study was developed in response to a mandate from the U.S Congress to study the causes of adolescent health with special emphasis on the effects of family, peer and neighborhood contexts (Harris, 2009). The study was funded by the National Institute of Child Health and Development (NICHD) and conducted by the Carolina Population Center at the University of North Carolina at Chapel Hill (UNC). Since its inception 20 years ago, data have been collected at 4 different time points, with individuals spanning ages 11 through 32. Today, Add Health is a rich dataset containing information on early life experiences with parents/caregivers, health behaviors, quality of relationships outside the home, economic transitions as well as biological markers of cardiovascular, metabolic and immune function in young adulthood.

4.2.1. Study Design and Sampling Procedures

The Add Health study design and sampling procedures are shown in Figure 4.1 below. Data were collected using a multi-stage stratified cluster sample design with high schools as the primary sampling unit. This study design ensured that the Add Health sample is representative of US schools with respect to region of country, metropolitan area, school type, race/ethnicity and school size (Tourangeau, 1999). The initial sampling frame of 26,666 US high schools was derived from the Quality education Database (QED), a comprehensive database of schools in the United States. Eligible high schools included 11th grade and had more than 30 enrolled students. Schools were stratified according to size, school type, census region, percent White and level of urbanicity. A stratified sample of 80 high schools was then selected with probability of selection proportional to school size (Chantala, 2014). For each high school selected, a feeder school (middle or junior high school) was also selected resulting in a school pair (1 high school and 1 feeder school) for each of the 80 different communities. Since some high schools included middle or junior high schoolers, a total of 52 feeder schools were selected for a total of 132 schools. Overall, 79% of schools contacted agreed to participate in the original study (Bernat et al., 2012). Details on each of the four waves of data are described below.

Figure 4.1. Add Health Study Design, Waves I through IV



At Wave I, a 45-minute paper and pencil in-school questionnaire was administered to 90,118 students in attendance the day of the interview in the 1994-1995 academic year. The in-school questionnaire included topics related to social and demographic characteristics, risk behaviors, household structure, health status and expectations for the future. School administrators (N=144) completed interviews relating to school policies and procedures as well as teacher and student body characteristics (Harris, 2009).

In phase II of Wave I interviews, a "core sample" (N=12,105) of students who completed the in-school survey, was selected to complete in-home youth questionnaires. In addition to the "core sample", Add Health researchers oversampled certain groups including Black adolescents from well-educated families (parents with college degrees), Chinese adolescents, adolescents with physical disabilities involving the use of limbs and specific Hispanic subgroups including Cuban and Puerto Rican adolescents. Add Health also oversampled pairs of siblings living in the same households, fraternal and identical twins as well as half-related siblings and non-related siblings (step-siblings, foster and adopted children) to form a genetic subsample. In all, 20,745 adolescents in grades 7-12 completed Wave I in-home questionnaires (Chantala, 2006). The in-home questionnaire topics included developmental status (height and weight), family dynamics, and educational aspirations and expectations. Also at in phase II, a resident parent or guardian of the in-home sample was asked to complete in-home interviews (N=17,670). The parent interview provided additional information about family composition, household characteristics, educational attainment of adults as well as household income. Over 80% of residents' parents (typically mothers) provided information relating to household characteristics including the number of adults and children living in the home, educational attainment of adults. In sum, Wave I data consist of an in-school survey (N=90,118), a "core sample" that completed an in-home survey in

addition to the in-school survey (N=20,745), a parent survey drawn from the "core" sample (N=17,670) and a school administrator survey (N=144).

In 1996, all adolescents in grades 7 through 11 at Wave I who completed the in-home component were followed up one year later for Wave II in-home interviews (N=14,738) with a response rate of 88.6%. Adolescents who were in 12th grade at Wave I were largely excluded from the Wave II sample because they exceeded the grade eligibility requirement however 12th graders who were part of the genetic sample were re-interviewed. The Wave I disabled sample was not re-interviewed at Wave II. School administrators were re-interviewed (N=128) however no parent interviews were conducted for the second wave. Wave II also includes a small number (N=27) of adolescents who did not participate in Wave I but were added as part of the genetic sample. Waves I and II in-home interviews represent the adolescent period with respondents completing identical questionnaires one year apart (Harris, 2009). Wave II also includes the collection of anthropometric measures of height and weight.

Wave III data represent the transition to adulthood. The purpose of the third wave of data was to understand how experiences occurring in adolescence are linked to behaviors and experiences in the transition to adulthood (Harris, 2009). The Wave III questionnaire builds on previous waves by including the collection of relationship and marital experiences, childbearing, education, and labor force histories. In addition to anthropometric measures, Wave III features the collection of saliva and urine specimens that were later assayed for HIV and curable STIs. Data were collected in 2001-02 when respondents were between the ages of 18 and 26 (N=15,197). It includes 15,170 of the original Wave I respondents plus the 27 Wave II special genetic respondents. Interviews were conducted nationwide, including Alaska and Hawaii. Respondents who were overseas at the time of data collection were not eligible to be re-

interviewed. At Wave III respondents were asked to retrospectively recall experiences of intimate partner violence in sexual or romantic relationships that respondents themselves listed as important. The overall response rate for Wave III was 77.4%.

Wave IV data were collected in 2007-08 when respondents were between the ages of 24 and 32 and settling into young adulthood. This latest wave of data followed and re-interviewed 15,701 of the original "core" sample. The overall response rate of 80.3% is an improvement over Wave III and is comparable to other national longitudinal studies including the annual NLSY97 (84%) and MIDUS II (75%) (Harris et al., 2009). Of the 15, 701 young adults interviewed at Wave IV 13,148 also completed Waves I and III interviews. Because the purpose of Wave IV was to study trajectories across the life course as respondents transition into young adulthood, the questionnaire follows up on prior questions while adding developmentally appropriate questions for young adults. Survey questions were expanded on educational transitions, economic status and financial resources and strains as well as the nature of romantic/cohabiting/marriage relationships. There are two major features of Wave IV data that are pertinent to this dissertation. First, respondents were asked to retrospectively recall the frequency of abuse events (emotional, physical and sexual) prior to age 18. Second, Wave IV features the collection of biological markers (Harris, 2009). Wave IV biological markers include anthropometric (height, weight, and waist circumference), cardiovascular (blood pressure, pulse),) metabolic (lipids, glucose and glycosylated hemoglobin) and, specific to this study, measures of inflammatory processes (C-reactive protein and Ebstein Bar Virus).

4.2.2 Data Collection: In-Home Component

Waves I, II and III in-home surveys were administered using computer CAPI (computer assisted personal interview). Trained interviewers asked questions and respondents used CAPI to

record their answers. Sensitive information was obtained using audio-computer assisted self-interviewing devices (ACASI) such that respondents entered answers based on headphone administered audio questions (Harris, 2009). The parents' questionnaire administered at Wave I however, was conducted using paper and pencil methods.

Wave IV interviews were administered on laptop computers using Blasé Survey Software; a computer assisted interviewing software that allows for conditional routing, validity and range checks, hierarchical questionnaire models, and linkage to external files. The advanced technology allowed researchers to collect complete tables of data such as household rosters and relationship data and then display the tables back to respondents to review and make corrections as needed. As with prior waves, sensitive information including child maltreatment and intimate partner violence was obtained using ACASI (Harris et al., 2009).

4.2.3. Biomarker Collection and Protocol

Table 4.1 below shows anthropometric and biological measures available at each wave of Add Health. Pertinent to this dissertation is the collection of anthropometric measures (height and weight) and blood samples.

Table 4.1. Add Health Biological Data Across Waves

Adolescence Wave 1-11	Transition to Adulthood Wave III	Young Adulthood Wave IV
Height, Weight Physical Development	Height, Weight STI tests (urine) HIV test (saliva) DNA (buccal cell)	Height, Weight, Waist Metabolic (lipids, HBA1c, glucose) Immune function (EBV) Inflammation (CRP) Cardiovascular (BP, P) DNA (buccal cell) Medications

Note: HbA1c = Glycosylated hemoglobin; EBV = Epstein Barr Virus; CRP = C-reactive protein; BP = blood pressure; P = Pulse rate

Anthropometric measures: At all four waves, respondents were first asked to provide self-reports of their weight and height. Field interviewers then took exact height and weight measurements according to established guidelines in order to enable the computation of body mass index. Respondents were free to decline any or all measurements while still participating in other components of the study (Entzel, 2009). Height was measured to the nearest quarter inch. Respondents were instructed to take off their shoes and stand up against a wall. Facing respondents, field interviewers used a level to measure the exact height of the respondents by marking the height with self-sticking papers on the wall. Field interviewers then used measuring tape against the wall and measured from the floor to the top edge of the paper. The exact height (in feet and inches) was transferred to appropriate fields on the computer screen. Weight was measured for all respondents who consented using a calibrated digital bathroom scale that measure weight to the nearest 0.5 pound. Respondents were again asked to remove their shoes and stand on the scale. Field interviewers were instructed to wait for the scale to settle and read the scale while the respondent was still standing on it. At Wave IV waist circumference was added to the collection of anthropometric measures. Waist circumference was measured to the nearest 0.5 centimeters using a metric –increment circumference tape measure.

Blood samples: At Wave IV, interviewers obtained dried blood spots (DBS) via finger prick, which were then frozen and shipped to study laboratories for assay of lipids panel (cholesterol), glucose; glycosylated hemoglobin (HBa1c), Epstein-Barr virus (EBV) and pertinent to this dissertation, high-sensitivity C-reactive protein (hsCRP). To collect dried blood spots, field researchers were trained to clean respondents' fingers with an alcohol swab and then prick it with a lancet. Seven droplets of blood were collected on specially treated filter paper. The blood spots are allowed to dry 10-15 minutes before being frozen and mailed to the

University of Washington Medical Center, Immunology Lab for analysis. Ninety-five percent of participants consented to dried blood spot collection (Harris, 2013). Of those who consented to dried blood spot and DNA collection, 80% agreed to archive their blood spots and 78% agreed to archive their DNA for future analysis (Harris, 2013). Finally, all prescription and select over-the-counter medications were inventoried at the time of the survey to help identify cardiovascular diseases and determine any temporal changes or consequences.

As shown in Table 4.1 additional biomarkers in the Add Health dataset include saliva, buccal swabs for DNA extraction as well as cardiovascular measures including blood pressure (BP) and pulse rate (P).

4.2.4 Design Effects and Sample Weights

The clustered sample design of Add Health (and unequal probability of selection) means that estimates from analyses can be biased if a factor used for selection as a participant also influences the outcome of interest. As discussed above for example, Black adolescents whose parents earned college degrees of more were one of many oversampled groups. Parents' education influences selection of Blacks into the study and is also an important factor for family income. Unless adjustments are made to account for this oversampling, and any other factor that is oversampled, estimates will be biased. Special care must also be taken to account for differences in variance for respondents within the same clusters compared to those between clusters. To account for sampling design Add Health sample weights (described in detail below) were applied and survey estimation procedures (command: svy) in Stata 13 were used to conduct all analyses. Survey estimation procedures run estimation commands while accounting for the design characteristics in point estimates and variance estimation methods. The –svy- command in Stata declares survey design features and informs Stata of the design variables to include for

all analyses. By default –svy- computes standard errors by using the linearized variance estimator or first-order Taylor series linear approximation (Wolter, 2007).

Overall, Add Health sample weights were designed to turn the sample of adolescents interviewed into the population under study. To do this, sample weights were calculated to accomplish four major goals. First, weights compensate for differences in the selection probabilities across different members of the sample. Second, sample weights were calculated to compensate for difference in response rates across different subgroups and over time. The third and fourth purposes of Add Health sample weights are to adjust for chance fluctuations of the composition of the sample from the composition of the population as a whole and lastly to allow the sample totals to serve as estimates of population totals (Tourangeau, 1999). When the appropriate sample weights are applied, the Add Health sample is representative of US adolescents enrolled in 7-12th grade during the 1994-95 academic year. By using the appropriate sampling weights and adjusting for study design, unbiased estimates of population parameters and standard errors can be obtained. When combining multiple waves of data, Add Health technical staff recommend using sampling weights from the most recent panel data. Since sampling weights from Wave IV are calculated using weights from Wave I, respondents without valid weights at both waves (e.g. respondents added in the field, or not enrolled in the original 80 primary sampling units) should be dropped. I apply Wave IV grand sample weights for all participants.

4.2.5. *Data Permissions and Human Subjects Approval*

All original Add Health protocols received approval from the institutional review board. For Wave I and II data collection, passive parental consent was employed such that it was assumed a parent granted permission unless the child submitted a form with a signature

indicating otherwise. By Wave III, all participants were at least 18 years old and therefore parental consent was not needed however, participants gave written consent for the collection of all biological markers (Harris, 2013). This dissertation received Institutional Review Board approval from the UCLA Office of Human Research Protection Program (OHRPP) in June 2013 (IRB#13-001007).

4.3 Operationalization of Study Variables

4.3.1 Outcome Variable: C-reactive protein

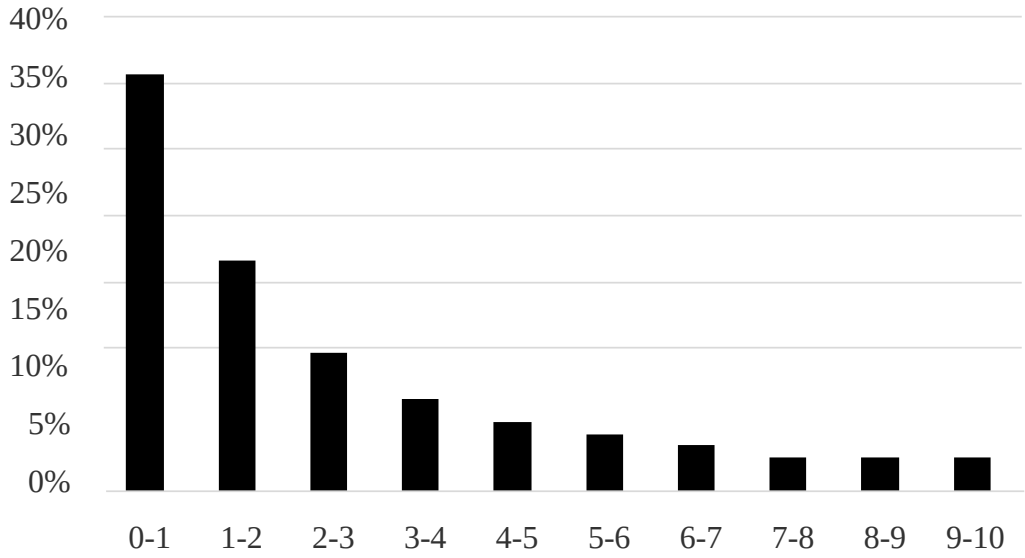
The outcome variable of interest is high sensitivity C-reactive protein (hsCRP), a marker of systemic inflammation. Dried blood spots (DBS) were collected as described above and shipped to the University of Washington Medical Center Immunology lab for analysis. The samples were assayed for hsCRP using sandwich enzyme-linked immunosorbent assay (sandwich ELISA) methods. The lower detection limit of DBS assays for hsCRP was 0.035mg/L compared to 0.082mg/L in plasma assays indicating that DBS has a lower detection limit. DBS are a minimally invasive procedure compared to plasma sampling; however, care must be taken to ensure that DBS produce similar results as plasma samples. For quality control, Add Health researchers also collected plasma samples from 87 respondents. Additionally, DBS and plasma samples were strongly and linearly correlated with a Pearson R of 0.98 indicating both methods yielded similar results (Whitsel et al., 2012).

CRP is operationalized as a continuous variable with values ranging from 0.28mg/L to 10mg/L. Both the Centers for Disease Control and Prevention and the American Heart Association classify CRP concentrations as follows: the expected concentration of CRP in healthy individuals is less than or equal to 3 mg/L. Levels between 3 to 10mg/L is considered "high risk" for cardiovascular diseases and values greater than 10 mg/L are often indicative of

active infections (e.g. a cold or flu) or ongoing chronic health conditions (Crawford, 2006). Figure 4.3 below shows the distribution of CRP in the analytic sample.

Because CRP is positively skewed (Figure 4.2) such that 84% of the sample had values less than 3 mg/L, CRP was log transformed (natural log) to obtain a distribution approximating normality. Raw CRP values were used for descriptive purposes.

Figure 4.2. Sample Distribution of CRP in 1 mg/L Intervals



4.3.2. Independent Variable (Aim 1): Emotional Abuse

The first explanatory variable of interest is emotional abuse. At Wave IV participants were asked to report retrospectively on maltreatment by adult caregivers prior to age 18 using modified questions from the Parent-Child Conflict Tactics Scale (Straus, 1990), and previous surveys (Finkelhor & Dziuba-Leatherman, 1994; Moore, 1995). Emotional abuse was measured with one item: "*How often did a parent or other adult caregiver say things that really hurt your feelings or made you feel like you were not wanted or loved*?" Add Health researchers grouped responses to these three items into six frequency categories: 0-Never happened to me, 1-One

time, 2-Two times, 3-Three to five times, 4-Six to ten times, and 5-More than ten times. For the purposes of this dissertation, response categories were recoded as 0-"This never happened to me", 1-"Once or Twice", 2-"Three to Five times" and 3-"Six or more times".

4.3.3. Independent Variable (Aim 2) (Aim 2): Adult Intimate Partner Violence

At Waves III and IV participants reported intimate partner violence occurring in romantic or sexual relationships specific to the 12 months before data collection. Questions related to intimate partner violence were derived from the Revised Conflicts and Tactics Scales (CTS2) (Straus, Hamby, Boney-McCoy, & Sugarman, 1996). During Wave III interviews, respondents listed all romantic and sexual partners since the summer of 1995 in a relationship roster. Recent sexual relationships and relationships that respondents deemed as important were selected for a more detailed relationship history including questions from the CTS2. To manage the complexity of Wave III IPV data, one current relationship per participant was selected by choosing the first current relationship listed on the roster. At Wave IV, rather than asking participants to report experiencing IPV in multiple relationships, they were asked instead to report IPV for one *current* relationship. If multiple current relationships were listed, priority was given based on the relationship type as follows: marriage, cohabiting, pregnancy and then dating. If two or more partners fell in the same relationship type, the longest relationship was selected. If two or more current relationships were of the same type and of the same duration, respondents was asked to select the partner they cared about the most. The intimate partner violence items at both Waves were as follows:

Threats: *How often (has/did) {initials} (threatened/threaten) you with violence (pushed/push) or (shoved/shove) you, or (thrown/throw) something at you that could hurt?*

Physical violence: *How often (has/did) {initials} (slapped/slap), hit or (kicked/kick) you?*

Forced sex: *How often (has/did) {initials} (insisted/insist) on or (made/make) you have sexual relations with (him/her) when you didn't want to?* Responses were grouped into 7 frequency categories: 0 -This has never happened to me, 1 -This has not happened in the past year, but it did happen before that; 2-Once in the last year of the relationship; 3-Twice once in the last year of the relationship, 4-Three to five times in the last year of the relationship, 5-Six to ten times in the last year of the relationship and 6-Eleven to twenty times in the last year of the relationship. History of adult intimate partner violence was operationalized using responses from Waves III and IV. Responses for each type of intimate partner violence were first dichotomized (never/hasn't happened in the past year versus happened one or more times in past year). A four category nominal variable was created indicating 0-Never, 1-Current exposure (intimate partner violence at Wave IV only), 2-Past exposure (intimate partner violence occurring at Wave III only) or 3-Chronic exposure (intimate partner violence occurring at both Waves III and IV). Respondents who were not in relationships at either Wave III or Wave IV (N=168) were coded as never experiencing adult intimate partner violence.

4.3.4. Independent Variable (Aim 3): Childhood and/or adult poverty

Childhood to adult poverty status was operationalized based on the official Federal poverty guidelines for 2008, the year Wave IV data were collected. The Federal poverty measure was created in the 1960s and consists of a series dollar amounts (guidelines) representing minimum standards of economic resources for families. Poverty guidelines were originally defined using figures for minimally adequate diet as developed by the US Department of Agriculture and figure were obtained based on the assumption that food expenditures constitute a third of household expenditures and remaining funds would be sufficient to cover other basic necessities. Poverty guidelines differ by family size and are adjusted annually for changes in cost

of living in the United States. By 1996, over two dozen federal programs based their eligibility standards on official poverty guidelines. Today Federal programs including Medicaid, Head Start and Women Infants, and Children (WIC) all the federal poverty line to determine eligibility. Families at 100% of the Federal poverty line represent absolute poverty in the United States, however many families are "near poor", meaning they have income between 100 and 185% of the Federal income line (Aber, Conley & Li, 1997). To capture the "near poor" many Federal programs set eligibility criteria above 100% of the Federal poverty line.

Childhood to adult poverty status was operationalized using two of the more frequently used eligibility criteria for Medicaid eligibility: 133% and 150% of Federal poverty line (FPL). At Wave 1 (1994) parents were asked to report total household income. Adolescents were also asked to describe each member in their household as part of a household roster, household size ranged from 1 to 15. I first used the Consumer Price Index to adjust for inflation between 1994 and 2008 (retrieved from http://www.stata.com/statalist/archive/2013-12/msg00134.html). Next using the 2008 cutoffs for federal poverty thresholds per household size, I calculated poverty status at both 133% and 150% FPL. The same process—except inflation adjustment—was carried out using Wave IV reports of household income and total number of households members. Next nominal variables were created as follows for 133% and 150% FPL respectively: 1-Never lived in poverty, 2-Poverty in Childhood only, 3-Poverty in Adulthood only, 4-Persistent Poverty. Cross tabulations confirmed the nominal variable at 150% of the Federal poverty line is a more liberal consideration in that the proportion of young adults who never experienced poverty under 133% FPL decreased from 68.1% to 65% under 150% FPL. Similarly, the proportion of young adults experiencing persistent poverty increased from 6.4% under 133% FPL to nearly 10% at 150% FPL. Nearly 6% of young adults in the Never category

under 133% FPL were captured as experiencing childhood poverty under 150% FPL, another 5% were captured as experiencing poverty in adulthood while less than 1% (0.4%) fell in the persistent poverty category. Of the number of respondents categorized as persistently poor at 150% FPL, approximately 18% were categorized as experiencing childhood poverty at 133%, nearly 13% were categorized at 133% as experiencing adult poverty only and just over 3% were previously categorized at 133% as never experiencing poverty.

4.3.5. Risk Factors for C-reactive Protein

Smoking is associated with maltreatment in childhood, intimate partner violence in adulthood and systemic inflammation (Pearson, 2011). Informed by CDC recommendations and prior research (Chen, 2012; Slopen, 2013), cigarette use is assessed based on two items. The first item asks *"Have you ever smoked an entire cigarette?*" The second asked *"During the past 30 days on how many days did you smoke cigarettes?"* Respondents who answered "no" on item one and 0 on item 2 were categorized as "Never" smokers. Those who answered "Yes" on item 1 and 0 on item 2 were categorized as former smokers. Respondents who smoked 20 cigarettes or less in the past 30 days were categorized as "intermittent" smokers and youth who smoked more than 20 cigarettes in a month were categorized as "current" smokers. Less than 1% of sample (N=71) had missing values on smoking. Missing values were redistributed proportionally across all four categories and non-smokers were selected as the reference category.

BMI: Adults with a history of child maltreatment and intimate partner violence are more likely to have high BMI (Williamson, 2002; Rohde, 2008; Felitti et al., 1998). BMI in turn, is strongly associated with inflammation (Yudkin, 1999; Hak et al., 2001). At Wave IV, researchers collected weight (pounds) and height (feet and inches) of respondents. BMI is operationalized as a continuous variable (weight in kilograms over height in meters squared $(kg)/(m)^2$) using an

existing measure created by Add Health. For approximately 2% (N=169) of the sample, BMI could not be calculated due to inconsistencies between height and weight, or refusal to participate. Regression imputation was used to retain these respondents.

Depressive Symptoms: Depressive symptoms refer to the emotions and behaviors associated with an affective state of extreme sadness and is a measure of psychological distress. At Wave IV, five of the original 20 items of the Center for Epidemiologic Studies Depression Scale (CES-D) were used (Goodman, 1999) to measure depressive symptoms. Respondents were asked to indicate how often over the past seven days they 1) were bothered by things that usually don't bother them; 2) could not shake off the blues; 3) felt depressed, 4) had trouble keeping their mind on what they were doing and 5) felt sad. Response choices were coded 0-Never or Rarely, 1-A lot of the time and 3-Most of the time or all of the time, with higher scores indicating more depressive symptoms. The items were summed to create a depressive symptoms score ranging from 0-15. Prior studies report good internal consistency (α=0.78) for the adapted version (Mueller, 2009). The reliability estimate for this sample (0.79) is similar to reliability estimates in a study by Mueller, et al. (2012) (α = .74).

4.3.6. Individual Socio-demographic Characteristics

The effect of maltreatment and intimate partner violence on systemic inflammation may differ by other childhood adversities such as childhood socioeconomic status. Therefore, I also examine childhood socioeconomic differences in inflammation. Childhood socioeconomic status is operationalized using parents' education and family income. Parents' education was calculated using Wave 1 reports from parents detailing the highest level of education completed in years. Biological mother's education level was used for respondents living in single mother households and the biological father's education level was used for those living in single father households.

For respondents living in two parent households, I use the parent with the higher education level. Missing values were imputed according to adolescents' report of parents' education level (Upchurch et al., 2002). When adolescents lived with a parent but neither adolescents nor parents reported parents' education level, weighted means based on racial categories were used to impute values. A subset of the sample (5.3%) reported not living with either parent. Rather than exclude this group from the analysis a dummy variable was created to indicate respondents did not have information on either parent. Parents' education was then categorized into four groups: less than high school, higher school diploma or GED earned, some college and college graduate or higher. The highest education level was selected as the reference category. Childhood family income was obtained from the parent's interview at Wave 1. Consistent with prior research (Upchurch et al., 2002), family income was determined by parents' total family income according to the family composition, with missing values imputed from a number of family background characteristics as reported by adolescents using OLS regression.[1] It was then coded into four categories: < $25,000, $25,000-$49,999, $50,000-$74,999 and ≥$75,000. The highest income group was set as the reference category.

Young adult individual characteristics—age and race/ethnicity are additional factors associated with CRP and were obtained from the Wave 1 adolescent interview (Danese, 2008; Slopen et al., 2013) The purpose of including age in this study is because of its positive association with CRP. Age is measured as a continuous variable and is calculated using the respondent's date of birth and the date of their participation in the Wave IV interview. Gender is coded 1-Female and 0-Male. Race/ethnicity was determined according to adolescent reports from

[1] Family background characteristics used in the regression imputation model included: family structure, mother's and father's working hours per week, parental public assistance, parental presence in the household and the education level of the mother and father

Wave I. Race/ethnicity is coded using into 4 categories giving priority to Hispanic ethnicity: non-Hispanic White, African American, Hispanic of any race and Asian/Pacific Islander. For multiracial respondents (4%), there was a follow up item asking to select a race/ethnicity with which they identify. If respondents selected a self-identified race/ethnicity, they were included in that racial/ethnic category. Non-Hispanic Whites were chosen as the reference category. Young adult socioeconomic status: Young adult education level and household income were obtained from young adult self-reports at Wave IV. There were four missing observations for education that were redistributed to the modal category. Young adult education level was coded into four categories: Less than High School, High School Graduate, Some College and College or more. The most educated group was chosen as the reference category. Missing values on Young adult household income were imputed using regression imputation[2] and coded into four categories: < $25,000, $25,000-$49,999, $50,000-$74,999 and ≥ $75,000. The highest earners were chosen as the reference group.

4.3.7 Other Covariates

Physical and sexual abuse: Young adults may have experienced physical or sexual abuse in childhood. Because both physical and sexual abuse have been shown influence inflammation levels (Bertone-Johnson et al, 2012), all analyses control for exposure to physical and sexual abuse. Both were dichotomized where 0 denotes no experience and 1 denotes at least one experience.

Use of anti-inflammatory medications: The use of anti-inflammation medication may confound estimates of CRP. At Wave IV, respondents were asked to report if they took any of the following medications in the four weeks prior to the date of the interview: Salicylate

[2] The variables used in the regression imputation model were age, race, gender, region, primary sampling unit and Wave IV sample weights.

medication, Cox-2 inhibitors, inhaled corticosteroids, corticotropin/glucocorticoids, antirheumatics, antipsoriatics or immunosuppressive medications. The variable is coded 1-Use of one or more of the above medications and 0-No use of anti-inflammatory medications.

4.4 Analytic Sample Derivation

The analytic sample was restricted to young adults who participated in the in-home interviews at Waves I, III and IV. Because Wave II data was collected just one year after Wave I, with identical questionnaires as Wave I and both represent the adolescent phase of Add Health, Wave II data were excluded from the analytic sample. In addition, respondents were excluded if they 1) did not have valid weights at Wave IV, 2) were pregnant at Wave IV, 3) identified as Native American or "Other" and 4) had CRP values greater than 10 mg/L (Figure 4.3). Of the 20,745 participants first interviewed at Wave I, 41 respondents were missing birth dates and were subsequently dropped for imputation purposes. At Wave III 5,575 respondents were lost to follow-up and an additional 1,981 were lost to follow-up at Wave IV. Of the 13,148 young adults interviewed at Waves I, II and IV, 901 were missing Wave IV sample weights because they were either added in the field (i.e. not part of the original Wave I school interview) or were not enrolled in the original 80 high schools. These 901 respondents were re-interviewed at Wave IV but were not assigned Wave IV sample weights. Additionally women who were pregnant at Wave IV (N=421) were excluded from the analytic sample because of the impact of pregnancy on hormone production and regulation. Due to the small size of young adults who identified as "Other" race/ethnicity or "Native American" (N=201), these respondents were excluded from the sample. Another 1,165 respondents were missing valid CRP values. Since individuals with CRP values greater than 10 mg/L (N=1,192) may be characterized by viral or bacterial infection I

follow established guidelines by excluding them from the sample (Ridker, 2003). The final analytic sample consists of 9,268 young adults (Figure 4.3).

4.5 Attrition Analysis

Respondents were asked to report experiences of child maltreatment at Waves III and IV. The present dissertation uses Wave IV maltreatment variables and an attrition analysis was conducted to determine whether reports of maltreatment at Wave III predict attrition at Wave IV after adjusting for demographic characteristics, parents' income and education level as well as young adult education and income at Wave III. This analysis is unweighted because it compares those used to derive the analytic sample to young adults who dropped out at Wave IV and therefore lack valid sample weights at Wave IV. Table 4.2 below provides results of these analyses and shows young adults reporting general neglect at Wave III were significantly less likely (approximately 10% less likely) to be lost to follow-up at Wave IV. Compared to men, young women were significantly less likely to be lost to follow-up and compared to Whites, racial minorities were more likely to be lost to follow-up at Wave IV. Lastly, the odds of non-response at Wave increases with decreasing education level. The above analysis provides confidence young adults reporting abuse at Wave III were not lost to follow-up at higher rates at Wave IV. Lastly, sample weights in the present study will decrease potential bias introduced due to lost to follow-up because sample weights were designed to adjust for attrition.

Table 4.2. Weighted Logistic Regression of Attrition at Wave IV.
Add Health Waves 1 and III (N=15,129)

	Adjusted Odds Ratio	95% CI
Report of Maltreatment at Wave III		
Physical Abuse (Ref = No)		
Yes	0.923	0.828, 1.012
Sexual Abuse (Ref = No)		
Yes	0.845	0.696, 1.078
Supervisory Neglect (Ref = No)		
Yes	0.897*	0.818, 0.984
General Neglect (Ref = No)		
Yes	1.133	1.023, 1.356
Young Adult Socio-demographics		
Age (range: 18 – 28 years)	0.982	0.955, 1.001
Gender (Ref=Male)		
Female	0.728***	0.650, 0.766
Race/Ethnicity (Ref=White)		
Black	1.387***	1.180, 1.549
Hispanic	1.328***	1.214, 1.549
Asian	1.818***	1.500, 2.035
Family SES		
Parents' Income (ref. = $75,000+)		
Less than $25,000	1.057	0.897, 1.244
$25,000 - $49,999	1.014	0.872, 1.178
$50,000 - $74,999	0.992	0.848, 1.159
Parent's Education (ref. = College or More)		
Less than High School	0.971	0.830, 1.135
High School Graduate	0.896	0.790, 1.017
Some College	1.051	0.934, 1.184
Young Adult SES		
Young Adult Income (ref. = $75,000+)		
Less than 25,000	0.725	0.412, 1.275
$25,000 - $49,999	0.753	0.424, 1.338
$50,000 - $74,999	1.173	0.608, 2.265
Young Adult Education Level (ref. = College or More)		
Less than High School	2.092***	1.418, 3.087
High School Graduate	1.780***	1.221, 2.597
Some College	1.494*	1.025, 2.175

Note: Wave IV sample weights used; CI = Confidence intervals; *p<.05; **p<.01; ***p<.001

4.6 Selection Analysis

Of the 12,247 respondents with valid sample weights interviewed at Waves I, III and IV, a total of 2,979 were excluded from the analytic sample. To understand differences between the excluded and analytic sample, a weighted logistic regression model predicting exclusion from the analytic sample as a function of young adult socio-demographic characteristics was conducted. The analysis was weighted using Wave IV sample weights to account for the complex study design using survey estimation commands. Table 4.3 presents results of this selection analysis. Women were more likely to be excluded because of the exclusion of pregnant women. Compared to Whites, Blacks and Hispanics were significantly more likely to be excluded from the analytic sample. Asians on the other hand were significantly less likely to be excluded compared to those whose parents had four years of college education or more. Respondents whose parents had less than a high school degree were significantly more likely to be excluded. In sum, non-whites, women and less educated respondents were more likely to be excluded from the analytic sample.

Table 4.3. Weighted Logistic Regression for Exclusion from Analytic Sample as a Function of Sociodemographic Characteristics, Add Health Waves 1, III, IV (N=9,268)

	Adjusted Odds Ratio	95% CI
Young Adult Sociodemographics		
Age (range: 24-32 years)	0.997	0.956, 1.041
Gender (Ref=Male)		
Female	5.133***	4.252, 6.200
Race/Ethnicity (Ref=White)		
Black	1.241*	1.009, 1.528
Hispanic	1.189*	1.009, 1.528
Asian	0.543**	0.330, 0.893
Young Adult Education Level (ref. = College or More)		
Less than High School	1.536*	1.008, 2.334
High School Graduate	1.189	0.922, 1.533
Some College	1.140	0.942, 1.378
Young Adult Household Income (ref. = $75,000+)		
Less than 25,000	0.973	0.755, 1.254
$25,000 - $49,999	1.042	0.863, 1.258
$50,000 - $74,999	1.067	0.894, 1.272
Family Background		
Parent's Education (ref. = College or More)		
Less than High School	0.876	0.642, 1.193
High School Graduate	1.050	0.833, 1.333
Some College	0.949	0.734, 1.225
Parent's Income (ref. = $75,000+)		
Less than $25,000	1.093	0.801, 1.491
$25,000 - $49,999	1.156	0.869, 1.540
$50,000 - $74,999	0.838	0.638, 1.010

Note: Wave IV sample weights used; CI = Confidence intervals; *p<.05; **p<.01; ***p<.001

4.7. Data Analysis Procedures

The primary objectives of this dissertation are 1) test for an association between emotional abuse and CRP and the extent to which the effect of emotional abuse on CRP are conditional on gender, 2) to test for an association between intimate partner violence and CRP the extent to which gender moderates the association between intimate partner violence and CRP and 3) test for an association between childhood and/or adult poverty and CRP and whether emotional abuse and intimate partner violence moderate its impact on CRP. The outcome variable, CRP, is continuous and because of the complex sample design and unequal probability of selection, weighted ordinary least squares (weighted OLS) regression models (EQ1) were used such that the contribution of each observation to the residual sum of squares is proportional to its population weight (Heeringa, et al., 2010). However because the outcome was highly skewed (see Figure 4.2) CRP was log transformed to improve distribution. The standard OLS regression model estimates the best linear relationship between a continuous dependent variable and a set of independent variables. In OLS, the best estimation is one that best minimizes the sum of squared residuals, the difference between an observed value and the fitted value provided by the regression model. In equation 1 (EQ1) below, $\hat{\mathbf{y}}$ is the predicted value of the dependent variable, **a** is the constant or intercept of the regression equation when all independent variables equal 0 and $\mathbf{b_1}$ and $\mathbf{b_2}$ are parameter estimates of the two independent variables $\mathbf{X_1}$ and $\mathbf{X_2}$. The first parameter estimate ($\mathbf{b_1}$) is the average expected change in the dependent variable for a 1-unit change in the first independent variable ($\mathbf{X_1}$) holding all other independent variables constant. Lastly, $\mathbf{b_2}$ is the average expected change in the dependent variable for a 1-unit change in the second independent variable ($\mathbf{X_1}$) holding all other independent variables constant.

$$\hat{\mathbf{Y}} = \mathbf{a} + \mathbf{b_1 X_1} + \mathbf{b_2 X_2} \quad \text{(EQ1)}$$

Although this same equation is used for analyzing complex study designs such as Add Health, the application of weights affects multiple linear regression by changing the method of estimation. Here, in a weighted least squares estimation, the contribution of each observation to the residual sum of squares is proportional to its population weight (Aneshensel, 2012). The estimates and corresponding standard errors from EQ1 are used to draw inferences about the true population values as shown below (EQ 2) where Y is the dependent variable, X_1 and X_2 are independent variables, β_1 and β_2 are population parameters and $\boldsymbol{\varepsilon}$ is an error term reflecting the difference between the actual value of the dependent variable, Y, and the predicted value, ŷ.

$$\mathbf{Y = \beta_0 + \beta_1(X_1) + \beta_2(X_2) + \varepsilon_i} \quad \text{(EQ 2)}$$

To test the hypothesis that there is no linear relationship between the dependent and independent variables (i.e. $\boldsymbol{\beta_1}$=0; $\boldsymbol{\beta_1}$=0), an adjusted Wald statistic is first used to test whether any of the coefficients for the independent variables differ from 0. The adjusted Wald statistic is similar to the overall F test statistic used in OLS, but rather than calculating the degrees of freedom as **n-k-1**, it is calculated as **df-k+1/df**, where **df** is the design-based degree of freedom (fixed at the number of clusters minus number of strata) and k is the number of parameters being tested (Heeringa, West, Berglund, 2010). Second, a t-statistic is used where SE is the standard error of $\mathbf{b_1}$ (EQ 3):

$$\mathbf{t = \frac{b_1}{SE(b_1)}} \quad \text{(EQ 3)}$$

Multiple regression allows for the quantification of the character of the relationship between maltreatment, CRP as well as other antecedent and intervening variables in a systematic way (Treiman, 2002). This means that I begin with a bivariate model (the focal relationship) and

then add variables sequentially to determine their effect on the focal relationship. To test the hypothesis that the coefficient of added variables equals 0, a modified partial Wald statistic (F distribution) is used with degrees of freedom m and ddf, where m is the difference in the number of variables from one model to the other and ddf is the design degrees of freedom as described above (Aneshensel, 2012).

4.7.1 Data Analysis Plan: Aim 1

Aim 1a: To examine the extent to which levels of CRP differ by experiences of emotional abuse independent of the presence of other known risk factors.

Aim 1b: To examine the extent to which the effect of emotional abuse on CRP emotional abuse is contingent on gender.

The first aim of this dissertation is to determine the extent to which levels of inflammation differ by exposure to emotional abuse. It is hypothesized that respondents with a history of emotional abuse will have higher mean levels of inflammation and that the associations will be contingent on gender. It is also hypothesized that differences in inflammation by emotional abuse will persist after controlling for physical and sexual abuse, experiences of physical and sexual abuse, individual demographic characteristics, childhood socioeconomic characteristics, young adult socioeconomic status, health behavior (smoking status) and measures of health status (BMI and depressive symptoms). Model building was performed in a sequential fashion, such that the focal independent variable (emotional abuse) is added first (Model 1) and theoretically similar variables are entered in a step-by-step fashion. An incremental F-test (labeled: F (d.f.) Statistic (Wald Test ΔR2)) is performed to determine whether the difference between nested models is statistically significant; that is, it tests the hypothesis H_0: $ß_1 = ß_2 = 0$

1. Weighted univariate analyses are conducted to obtain descriptive statistics (means, standard deviations, proportions).
2. The total association between emotional abuse and inflammation is estimated such that

$$Y_{CRP} = a + b_1(\text{EA-Once}) + b_2(\text{EA-Two to Five}) + b_3(\text{EA-SixPlus}) + e \quad \text{(Model 1)}$$

3. The total association between gender and inflammation is obtained such that

$$Y_{CRP} = a + b_1(\text{gender}) + e \quad \text{(Model 2)}$$

A three variable model regressing inflammation on emotional abuse and gender is obtained such that

$$Y_{CRP} = a + b_1(\text{EA\_Once}) + b_2(\text{EA\_Two-Five}) + b_3(\text{EA\_SixPlus}) + b_4(\text{gender}) + e \quad \text{(Model 3)}$$

4. CRP is regressed on emotional abuse, gender and the interaction of emotional abuse and gender. The interaction is operationalized by three product interaction terms obtained by multiplying gender by three separate dummy variables: experiencing emotional abuse once, experiencing emotional abuse two to five times and experiencing emotional abuse six or more times. emotional abuse

$$Y_{CRP} = a + b_1(\text{EA\_Once}) + b_2(\text{EA\_Two-Five}) + b_3(\text{EA\_SixPlus}) + b_4(\text{gender}) + b_5(\text{EA\_Once*gender}) + b_6(\text{EA\_Two-Five*gender}) + b_7(\text{EA\_SixPlus*gender}) + e \quad \text{(Model 4)}$$

An incremental F test is used to contrast this model with the "unconstrained" model (the model without the interaction term). The incremental F-test tests the null hypothesis that all coefficients for the interactions equal zero. Statistical significance indicates the interaction term improves the fit of the model. As such, the model containing the interaction is used for subsequent analyses, otherwise the more parsimonious model without the interaction term is used in nested regression models.

5. Starting with the more parsimonious model, theoretically similar variables are introduced (Aneshensel, 2013). Model 5 adjusts for physical and sexual abuse, Model 6 introduces gender, age and race/ethnicity, Model 7 adds parents' income and education, Model 8 adjusts for young adult income and education and last, Model 9 adds health factors including smoking status, BMI and depressive symptoms. All models control for the use of anti-inflammatory medications. Parameter estimates of new models are compared to estimates of previous models. The interpretation of change in the size and/or significance of regression coefficients depend on theoretical conceptualizations of groups of variables. For example, childhood socioeconomic status is conceptualized as an alternative independent variable in Aim 1. A loss of significance after adding childhood socioeconomic status would mean that the effect of emotional abuse on inflammation is redundant. That is, the relationship between emotional abuse and inflammation is not distinct from the inflammatory effect of childhood socioeconomic status. A loss of significance after the addition of a control variable (e.g. anti-inflammatory medication use) would mean the association between emotional abuse and inflammation is a spurious one that is confounded by the control variable. Lastly, a loss or reduction in significant after the addition of a variable conceptualized as a mediator (e.g. BMI) would indicate BMI mediates the association between emotional abuse and CRP.

4.7.2. Analytical Plan: Aim 2

Aim 2a: To examine the extent to which levels of CRP differ by history of adult intimate partner violence, independent of the presence of other known risk factors.

Aim 2b: To examine the extent to which the effect of intimate partner violence on CRP is contingent on gender.

The second aim of this dissertation is to examine whether exposure to intimate partner violence is associated with higher levels of CRP. It is hypothesized that respondents with a history of intimate partner violence will have higher mean levels of inflammation and that these associations will be contingent on gender. It is also hypothesized that differences in inflammation will persist after controlling for measures of childhood socioeconomic status, individual demographic characteristics, young adult socioeconomic status and young adult health behavior and health status. Model building was performed as described above for aim 1.

1. Weighted univariate analyses are conducted to obtain descriptive statistics (means, standard deviations, proportions).
2. The total association between intimate partner violence and inflammation is estimated such that

$$Y_{CRP} = a + b_1(IPV\_current) + b_2(IPV\_past) + b_3(IPV\_chronic) + e \quad \text{(Model 1)}$$

3. The total association between gender and inflammation is obtained such that

$$Y_{CRP} = a + b_1(gender) + e \quad \text{(Model 2)}$$

4. A three variable model regressing inflammation on intimate partner violence and gender is obtained such that

$$Y_{CRP} = a + b_1(IPV\_current) + b_2(IPV\_past) + b_3(IPV\_chronic) + b_4(gender) + e \quad \text{(Model 3)}$$

5. After examining the individual effects of intimate partner violence and gender on inflammation, CRP is regressed on intimate partner violence, gender and the interaction of intimate partner violence and gender (Model 4). The interaction terms are obtained by multiplying gender by three separate dummy variables for 1) experiencing intimate partner violence in a current relationship, 2) experiencing intimate partner violence in a

past relationship and 3) experiencing intimate partner violence chronically in past and current relationship where never experiencing IPV is the omitted reference group such that

$$Y_{CRP} = a + b_1(IPV\_current) + b_2(IPV\_past) + b_3(IPV\_chronic) + b_4(gender) + b_5(IPV\_current*gender) + b_6(IPV\_past*gender) + b_7(IPV\_chronic*gender) + e \quad \text{(Model 4)}$$

As with aim 1, an incremental F test is used to contrast this model with the "unconstrained" model. An inability to reject the null hypothesis that all coefficients for the interactions equal zero indicates the interaction term doe not improve the fit of the model. As such, the more parsimonious "unconstrained" model without the interaction term is used for subsequent analyses. I start with the more parsimonious model and introduce theoretically similar variables (Aneshensel, 2013). As with emotional abuse, model building is performed in a sequential fashion as previously described. Parameter estimates of each new model are compared to estimates of previous models and the interpretation of change in the size and/or significance of the regression coefficient depends on the theoretical conceptualizations of groups of variables.

4.7.3. *Data Analysis Plan: Aim 3*

Aim 3a: To examine the extent to which levels of CRP differ by childhood and/or adult poverty, independent of childhood experiences of emotional abuse, adult intimate partner violence and other known risk factors.

Aim 3b: To test for a synergistic relationship between by childhood and/or adult poverty, Abuse in childhood and intimate partner violence.

The third aim of this dissertation is to examine whether living in poverty in childhood and/or adulthood is associated with higher CRP levels net of childhood experiences of emotional abuse and adult experiences of intimate partner violence. It is hypothesized respondents who lived in poverty in childhood and adulthood (persistent poverty) will have the higher mean CRP levels compared to young adults who never lived in poverty. It is also hypothesized the

association between persistent poverty and CRP will be contingent on emotional abuse such that the impact of childhood and/or adult poverty on CRP is greater for young adults who also experienced emotional abuse. Lastly, the association between persistent poverty and inflammation is expected to be contingent on intimate partner violence such that the impact of persistent poverty on CRP is greater for young adults who also experienced intimate partner violence. Model building was performed as described above for Aims 1 and 2

1. Weighted univariate analyses are conducted to obtain descriptive statistics (means, standard deviations, proportions).
2. The total association between poverty status and inflammation is estimated such that

$$Y_{CRP} = a + b_1\,(\text{child poverty}) + b_2(\text{adult poverty}) + b_3(\text{persistent poverty}) + e \quad \text{(Model 1)}$$

3. A three variable model regressing inflammation on poverty status and emotional abuse is obtained such that

$$Y_{CRP} = a + b_1\,(\text{child poverty}) + b_2(\text{adult poverty}) + b_3(\text{persistent poverty}) + b_4(\text{EA\_Once}) + b_5(\text{EA\_Two-Five}) + b_6(\text{EA\_SixPlus}) + e \quad \text{(Model 3)}$$

4. After examining the individual effects of poverty status and emotional abuse on inflammation, CRP is regressed on poverty status, emotional abuse and the interaction of poverty status and emotional abuse. The interaction terms are obtained by multiplying each level of poverty status by each level of emotional abuse leaving respondents who never lived in poverty nor experienced emotional abuse as the reference category.

$$Y_{CRP} = a + b_1\,(\text{child poverty}) + b_2(\text{adult poverty}) + b_3(\text{persistent poverty}) + b_4(\text{EA\_Once}) + b_5(\text{EA\_Two-Five}) + b_6(\text{EA\_SixPlus}) + b_7(\text{child poverty*\ EA\_Once}) + b_8(\text{child poverty*\ EA\_Two-Five}) + b_9(\text{child poverty*\ EA\_SixPlus}) \ldots + b_{15}(\text{persistent poverty*\ EA\_SixPlus}) + e \quad \text{(Model 4)}$$

Steps two through four are repeated for intimate partner violence. As above, an incremental F test is used to contrast this model with the "unconstrained" model. An inability to reject the null hypothesis that all coefficients for the interactions equal zero indicates the interaction term does not improve the fit of the model. As such, the more parsimonious "unconstrained" model without the interaction term is used for subsequent analyses. I start with the more parsimonious model and introduce theoretically similar variables (Aneshensel, 2013). As before, model building is performed in a sequential fashion as previously described. Parameter estimates of each new model are compared to estimates of previous models and the interpretation of change in the size and/or significance of the regression coefficient depends on the theoretical conceptualizations of groups of variables.

4.8. Sample Characteristics

Table 4.4 presents weighted descriptive statistics and unweighted Ns of young adult and childhood socio-demographic characteristics and health factors for the analytic sample of 9,268 individuals. The average age is 28 years with a range of 24 to 32 years. The majority of young adults were White (71%), followed by Black (14%), Hispanic (11%) and Asian (3.8%). Almost one-third of young adults achieved four or more years of college and another 43% had some college education. About one quarter of young adults reported incomes between $50,000 and $75,000 and another 28% reported incomes of $75,000 and over. With respect to their parents, one-third of parents had four or more years of college and 13% did not complete high school. Only 12% of their parents had the highest income and over one-quarter had the lowest. Over one-quarter of young adults were current smokers and another 12% intermittent smokers. The average BMI is 28 kg/m^2 (overweight) with a range of 14 to 80 kg/m^2 and on average respondents showed low levels of depressive symptoms (approximately 3). Lastly 17% reported

experiencing physical abuse before the age of 18, 5% reported experiencing sexual abuse before the age of 18 and nearly 30% reported using anti-inflammatory medications.

Table 4.4. Weighted Distribution of Sociodemographic Characteristics and Health Factors, Add Health Waves I, III, IV (N=9,268)

Variables	% or Mean (SE) Weighted	N Unweighted
Young Adult Demographics (Wave IV)		
Age (range: 24-32 years)	28.3 (0.12)	9,268
Gender		
Men	52.7	4,537
Women	47.3	4,731
Race Ethnicity		
White	71.0	5,358
Hispanic	11.0	1,415
Black	14.0	1,846
Asian	3.8	649
Young Adult Education Level		
Less than High School	8.1	675
High School Graduate	17.2	1,439
Some College	43.4	4,105
College or More	31.3	3,049
Young Adult Household Income		
Less than $25,000	15.8	1,344
$25,000 - $49,999	33.0	2,983
$50,000 - $74,999	23.0	2,173
$75,000 or more	28.2	2,768
Family Background (Wave I)		
Parent's Education		
Less than high school	13.0	1,219
High school graduate	25.0	2,183
Some College	29.0	2,640
College or More	33.0	3,226
Parent's Income		
Less than $25,000	29.7	2,727
$25,000 - $49,999	35.7	3,351
$50,000 - $74,999	22.4	2,021
$75,000+	12.2	1,169

Table 4.4 Continued. Weighted Distribution of Sociodemographic Characteristics and Health Factors. Add Health Waves I, III, IV (N=9,268)

Variables	% or Mean (SE) Weighted	N Unweighted
Health Factors (Wave IV)		
Smoking		
Never	31.4	3,276
Former Smoker	28.8	2,584
Intermittent Smoker	11.6	1,115
Current Smoker	28.2	2,293
BMI (range: 14.4-80.4 kg/m^2)	28.5 (0.14)	9,268
Depressive Symptoms (range: 0-15)	2.6 (0.04)	9,268
Other Covariates		
Physical Abuse		
Yes	17.0	1,664
No	83.0	7,604
Sexual Abuse		
Yes	5.0	433
No	95.0	8,835
Anti-inflammatory Medication Use		
Yes	29.4	2,726
No	70.6	6,542

Note: Wave IV sample weights used.

Table 4.5 presents weighted distribution and unweighted sample sizes for emotional abuse and adult intimate partner violence. Since aims 1 and 2 of the present dissertation examine gender differences on the influence of emotional abuse and intimate partner violence on inflammation, Table 4.5 is stratified by gender. Forty-six percent of young adults reported experiencing emotional abuse at least once. Nearly 20% experienced emotional abuse two to five times while just over 16% experienced emotional abuse six or more times. Among women, just over half (53%) experienced emotional abuse compared to approximately 40% of men.

Just over 30% of young adults reported experiencing intimate partner violence in adult relationships. Fourteen percent reported experiencing intimate partner violence in their current relationships, another 11% report intimate partner violence in the past while 4% of young adults

report intimate partner violence in their past and current relationships. In the gender stratified models, a higher proportion of men reported intimate partner violence (32%) compared to women (27%).

The effect of childhood and/or adult poverty status on inflammation is examined in Aim 3. Because Aim 3 also tests for a synergistic relationship between poverty status, emotional abuse and intimate partner violence, Table 4.6 is stratified by emotional abuse and Table 4.7 is stratified by intimate partner violence. At a 133% of Federal poverty line, nearly 68% of respondents never lived in poverty, nearly 15% lived in poverty as children, a tenth of respondents lived in poverty as young adults and 7% experienced persistent poverty. At 150% of Federal poverty line, just of 60% of young adults never lived in poverty, approximately 16% lived in poverty as children, 12% lived in poverty as adults and 10% experienced persistent poverty. Table 4.6 also shows at 133% of FPL, 44% of respondents who never lived in poverty reported emotional abuse at least once, 49% of those who experienced childhood poverty reported at least one incident of emotional abuse while 48% of young adults who live persistent poverty reported one or more experiences of emotional abuse.

Table 4.5. Weighted Distribution of Emotional Abuse and Adult Intimate Partner Violence by Gender. Add Health Waves I, III, IV (N=9.268)

	Total (N=9,268)		Women (N=4,731)		Men (N=4,537)	
	%	N	%	N	%	N
Child Maltreatment (Wave IV)						
Emotional Abuse						
Never	54.0	4,966	47.0	2,271	60.1	2,695
Once	10.3	951	10.6	483	10.1	468
Two to five times	19.5	1,861	23.2	1,080	16.0	781
Six or more times	16.2	1,490	19.2	897	13.6	593
Adult Intimate Partner Violence (Waves III and IV)						
History of Adult Intimate Partner Violence						
Never	70.1	6,494	72.7	3,392	67.5	3,102
Current IPV	14.2	1,319	10.8	535	17.8	784
Past IPV	11.4	1,058	12.8	614	10.0	444
Chronic IPV	4.3	397	3.7	190	4.7	207

Note: Wave IV sample weights used.

Table 4.6. Weighted Distribution of Poverty Status by Emotional Abuse. Add Health Waves I, III, IV (N=9.268)

	Total Sample		Emotional Abuse							
			Never		Once		Two-Five		Six or More Times	
	%	N	%	N	%	N	%	N	%	N
Childhood to Adult Poverty Status (Waves I and IV)										
133% of Federal Poverty Level										
Never lived in Poverty	67.9	6,320	37.7	3,489	6.4	600	13.0	1,253	10.8	978
Poverty in Childhood	14.8	1,446	7.4	727	1.8	170	3.2	303	2.4	246
Poverty in Adulthood	10.3	908	5.1	443	1.4	112	1.9	178	1.9	175
Persistent Poverty	7.0	594	3.7	307	0.7	69	1.5	127	1.1	91
150% of Federal Poverty Level										
Never lived in Poverty	60.8	5,624	34.0	3,123	5.7	525	11.7	1,124	9.4	852
Poverty in Childhood	16.2	1,622	8.1	824	1.8	184	3.4	324	2.9	290
Poverty in Adulthood	12.6	1,113	6.4	552	1.6	138	2.3	216	2.3	207
Persistent Poverty	10.4	909	5.4	467	1.2	104	2.2	197	1.6	141

Table 4.7. Weighted Distribution of Poverty Status by Intimate Partner Violence. Add Health Waves I, III, IV (N=9.268)

	Intimate Partner Violence							
	Never		Current		Past		Chronic	
	%	N	%	N	%	N	%	N
Childhood to Adult Poverty Status (Waves I and IV)								
133% of Federal Poverty Level								
Never lived in Poverty	50.0	4,605	9.0	819	73.0	673	22.0	223
Poverty in Childhood	9.6	960	2.6	247	1.9	177	0.7	62
Poverty in Adulthood	6.4	556	1.9	159	1.3	122	0.8	71
Persistent Poverty	4.5	373	1.3	94	0.9	86	0.5	41
150% of Federal Poverty Level								
Never lived in Poverty	45.0	4,121	7.8	718	6.5	603	1.7	182
Poverty in Childhood	10.7	1,101	2.7	260	2.0	193	0.9	68
Poverty in Adulthood	7.8	703	2.3	190	1.4	131	1.0	89
Persistent Poverty	6.6	569	1.7	151	1.4	131	0.7	58

4.8.1 Distribution of Inflammation Levels by Study Variables

Table 4.8 shows weighted mean CRP levels by child emotional abuse, adult intimate partner violence and the two measures of childhood to adult poverty status. First, the mean CRP value for the analytic sample is 2.6 mg/L with a range of 0.08 to 10 mg/L. This mean is below the clinically significant cut-off of 3 mg/L. Per established guidelines, CRP levels less than or equal to 1 mg/L are considered low risk, values greater than 1 mg/L or equal to 3mg/L are considered moderate risk while values above 3 mg/L are considered high risk. Differences in mean CRP levels differ significantly across exposures to emotional abuse, intimate partner violence and both poverty measures. Young adults who experienced emotional abuse six or more times and respondents who experienced chronic intimate partner violence had the highest CRP levels in their respective groups. Young adults living in persistent poverty at both 133% and 150% FPL had the highest CRP levels.

While the Wald tests presented in Table 4.6 indicate one or more categories of each of the four independent variables were significantly difference from each other, it does not specify which categories differ from each other. A subsequent post estimation command showed mean CRP levels for young adults experiencing emotional abuse six or more times was significantly different from those who never experienced emotional abuse ($P<0.01$). Mean CRP levels for young adults experiencing emotional abuse six or more times were also significantly different from young adults who experienced emotional abuse two to five times. For intimate partner violence, mean CRP was significantly different for the extreme categories (never vs. chronic) with p value less than 0.01. There were no statistically significant differences in mean CRP for young adults with no experiences of intimate partner violence compared to those experiencing intimate partner violence in current relationships nor were differences observed in mean CRP

values between those who never experienced intimate partner violence and young adults with past histories of intimate partner violence ($p>0.05$). Mean CRP values differed significantly, however for young adults experiencing intimate partner violence in current relationships compared to those with chronic experiences of intimate partner violence. When it comes to poverty status at 133% FPL, there were observed differences in mean CRP values for young adults who never experienced poverty and those who lived in persistent poverty ($F(1, 128) = 16.56$, $p<0.001$), those who never experienced poverty compared to those who experienced childhood poverty ($F (1, 128) = 4.49$, $p<0.05$), between those who experienced poverty as adults and those who lived in persistent poverty ($F (1, 128) = 7.98$, $p<0.01$)) and also between experiencing poverty in childhood compared to living in persistent poverty ($F (1, 128) =7.43$, $p<0.01$). Differences in mean values for young adults who never experienced poverty and those who experienced poverty in adulthood; as well as between those who experienced poverty in childhood compared to those who experienced poverty in adulthood were not statistically significant ($F (1, 128) = 2.06$, $P > 0.05$; $F (1, 128) = 0.11$, $P > 0.05$ respectively).

A similar pattern was observed for poverty status at 150% FPL however here, differences in mean CRP for young adults who never experienced poverty and those who experienced poverty in adulthood were also statistically significant ($F (1,128) = 5.86$, $P < 0.01$).

Table 4.8. Weighted Mean CRP (mg/L) by Independent Variables: Emotional Abuse, Adult Intimate Partner Violence and Childhood and/or Adult Poverty. Add Health Waves I, III, IV (N=9,268)

	Mean	SE	P-Value
Total Sample	2.58	0.04	
Child Maltreatment (Wave IV)			
Emotional Abuse			
Never	2.51	0.53	**
Once	2.60	0.11	
Two to five times	2.60	0.08	
Six or more times	2.83	0.94	
History of Intimate Partner Violence (Waves III and IV)			
Never	2.53	0.05	*
Current IPV	2.61	0.09	
Past IPV	2.73	0.10	
Chronic IPV	3.01	0.18	
Childhood to Adult Poverty Status (Waves 1 and IV)			
133% of Federal Poverty Level			***
Never lived in Poverty	2.47	0.04	
Poverty in Childhood	2.69	0.09	
Poverty in Adulthood	2.64	0.12	
Persistent Poverty	3.33	0.21	
150% of Federal Poverty Level			
Never lived in Poverty	2.44	0.04	***
Poverty in Childhood	2.61	0.08	
Poverty in Adulthood	2.75	0.12	
Persistent Poverty	3.15	0.14	

Note: Wave IV sample weights used; SE=standard error; *p<.05; **p<.01; ***p<.001 for F-test of differences by each categorical variable

Table 4.9 shows weighted mean CRP values by socio-demographic factors, health behaviors and health status. The adjusted Wald test is conducted to test the hypothesis that there is no association between each of the socio-demographic characteristics, health behaviors and health status variables and CRP. Relative to Whites, Blacks had the highest mean CRP, followed by Hispanics and Whites and Asians had the lowest levels. Mean CRP increased incrementally with decreases in parents' education, parents' income, young adult education and young adult income (all p-values < 0.001), indicating at least two of the values for race/ethnicity, parents'

education, parents' income, young adult education and young adult income differ from one another. Surprisingly, intermittent smokers had the lowest mean CRP relative to other smoking categories. CRP increased with increasing BMI such that obese young adults had the higher mean CRP compared to respondents at normal weight and young adults who reported using anti-inflammatory drugs had significantly higher mean CRP compared to those who did not use anti-inflammatory drugs. Lastly, though mean CRP increased with increasing depressive symptoms, the differences were not statistically significant ($p>0.05$).

Table 4.9. Weighted Mean CRP (mg/L) by Sociodemographic Characteristics, Health Factors and Other Control Variables. Add Health Waves I, III, IV (N=9,268)

	Mean	SE	P-Value
Young Adult Demographics (Wave IV)			
Age (range: 24-32 years)			
24 - 29 years	2.57	0.05	
30 - 32 years	2.62	0.60	
Gender			***
Men	2.25	0.05	
Women	2.96	0.05	
Race Ethnicity			***
White	2.57	0.05	
Hispanic	2.67	0.09	
Black	2.80	0.10	
Asian	1.74	0.12	
Young Adult Education Level			***
Less than High School	2.81	0.14	
High School Graduate	2.79	0.09	
Some College	2.72	0.05	
College or More	2.22	0.06	
Young Adult Household Income			***
Less than $25,000	2.86	0.13	
$25,000 - $49,999	2.73	0.07	
$50,000 - $74,999	2.57	0.07	
$75,000+	2.25	0.06	
Family Background (Wave I)			
Parent's Education			***
Less than high school	2.81	0.12	
High school graduate	2.85	0.07	
Some College	2.55	0.07	
College or More	2.32	0.06	

Table 4.9 Continued. Weighted Mean CRP (mg/L) by Sociodemographic Characteristics, Health Factors and Other Control Variables.
Add Health Waves I, III, IV (N=9,268)

	Mean	SE	P-Value
Parent's Income			***
Less than $25,000	2.76	0.07	
$25,000 - $49,999	2.70	0.06	
$50,000 - $74,999	2.40	0.08	
$75,000+	2.13	0.08	
Health Factors (Wave IV)			
Smoking			***
Never	2.60	0.07	
Former Smoker	2.58	0.06	
Intermittent Smoker	2.42	0.11	
Current Smoker	2.63	0.07	
BMI			***
Normal Weight (18.5 – 24.9kg/m^2)	1.63	0.04	
Overweight (25.0–29.9m^2)	2.87	0.04	
Obese (30.0 and above)	5.24	0.16	
Depressive Symptoms (in tertiles, range: 0 - 15)			NS
First	2.53	0.06	
Second	2.57	0.06	
Third	2.66	0.08	
Control Variables (Wave IV)			
Physical Abuse			NS
Yes	2.58	0.04	
No	2.60	0.09	
Sexual Abuse			*
Yes	2.57	0.04	
No	2.88	0.14	
Anti-inflammatory Medication Use			***
Yes	2.84	0.07	
No	2.47	0.05	

Note: Wave IV sample weights used; *p<0.05; **p<.01; ***p<.001, NS=Not Significant

4.8.2 Chapter Summary

These preliminary results set the stage for analyses to follow. These results support a relationship between emotional abuse and inflammation and there were significant differences in inflammation by experiences of intimate partner violence. These preliminary analyses also support an association between both measure of childhood to adult poverty status and inflammation. Taken together these initial examinations support further modeling to understand the ways in which emotional abuse, intimate partner violence, exposure to poverty influence inflammation in young adulthood. The following chapter (chapter 5) describes differences in inflammation by emotional abuse and chapter 6 describes differences in inflammation by history of adult intimate partner violence. Lastly, chapter 7 describes the extent to which exposure to poverty in childhood and/or adulthood help to explain differences in inflammation over and above the effects of childhood emotional abuse and adult intimate partner violence.

CHAPTER 5

AIM 1 RESULTS: THE EFFECT OF EMOTIONAL ABUSE ON C-REACTIVE PROTEIN

5.1 Overview

This chapter describes associations between emotional abuse and CRP. Using weighted nested regression models, I elaborate the relationship between CRP and emotional abuse and each of the key variables. I describe the relationship between each of the key variables and CRP and discuss the extent to which the focal relationship between emotional abuse and CRP changes.

5.2 The Effect of Emotional Abuse on CRP

Table 5.1 presents results of weighted OLS regressions of CRP on emotional abuse, gender and an emotional abuse by gender interaction. For each model, a control variable for use of anti-inflammatory medication is included. Model 1 shows young adults with a history of emotional abuse had higher CRP levels compared to those with no history of emotional abuse. Specifically, compared to young adults with no history of emotional abuse, young adults who experienced emotional abuse six of more times had significantly higher levels of CRP ($p<0.001$). Experiencing emotional abuse once or two to five times however, did not significantly differ from no exposure. Emotional abuse alone explained less than one percent of the variance in CRP ($R^2=0.009$). Because it was hypothesized that the effect of emotional abuse on inflammation may be contingent on gender, Model 2 shows a bivariate association between gender and CRP; women had significantly higher CRP levels compared to men. The effect of emotional abuse occurring six or more times was reduced but remained statistically significant when both emotional abuse and gender were included in one model (Model 3) and the incremental F-test shows the addition of gender to Model 1 significantly improved model fit ($\Delta R^2 =0.012$; 25.51 (5,

124), p<0.001). The interaction between emotional abuse and gender however, was not statistically significant, and a post-hoc omnibus test for interaction showed a p-value of 0.75, suggesting the effect of emotional abuse on CRP was not contingent on gender (Model 4). In other words the magnitude of effect of emotional abuse on CRP is not different the same for men and women as hypothesized.

For each of the models a control variable for the use of anti-inflammatory medications was included and as expected, on average young adults who used anti-inflammatory medications had higher mean CRP. Aim 1 also assessed the effects of other forms of maltreatment (physical and sexual abuse) on CRP, and their interaction with gender. Physical abuse was not significantly associated with CRP levels (Table 5.2, Model 1) and a test of the interaction between physical abuse and gender was not significant (Table 5.2, Model 4). Table 5.3 shows young adults with a history of sexual abuse had higher CRP levels compared to those with no history of sexual abuse (Model 1), however when gender was added in Model 3, the association between sexual abuse and CRP was no longer statistically significant. The incremental F-test for Model 3 shows, the addition of gender significantly improved the fit of the model (64.49 (1, 128); p<0.001). A test of the interaction between sexual abuse and gender was not significant indicating the effect of sexual abuse on inflammation is not contingent on gender. For the remainder of the Aim 1 analyses, I focus on the effects of emotional abuse on inflammation and only present main effects models.

Table 5.1. Weighted OLS Regression of Inflammation on Emotional Abuse and Gender (N=9,268)

	Model 1		Model 2		Model 3		Model 4	
	b	SE	b	SE	b	SE	b	SE
Independent Variable								
Emotional Abuse (ref. = Never)								
Once	0.047	0.054			0.030	0.054	-0.003	0.069
Two to five times	0.034	0.042			-0.003	0.041	0.007	0.058
Six or more times	0.143***	0.044			0.107*	0.044	0.140*	0.060
Female			0.255***	0.031	0.250***	0.300	0.256***	0.040
Emotional abuse-x-Female Interactions								
Once-x-Female							0.070	0.095
Two to five times-x-Female							-0.018	0.077
Six or more times-x-Female							-0.060	0.080
Anti-inflammatory Medication Use	0.200***	0.031	0.180***	0.031	0.180***	0.030	0.177***	0.030
Constant	0.328***	0.272	0.247	0.027	0.231***	0.032	0.229***	0.034
Model Statistics								
R^2	0.009		0.015		0.021		0.022	
F(d.f.) Statistic (Wald Test)	14.08 (4, 125)***		79.07 (1, 128)***		25.51 (1, 128)***		15.88 (8, 121)***	
ΔR^2	---		---		0.012		--	
F (d.f.) Statistic (Wald Test ΔR^2)	---		---		25.51 (5, 124)***		0.39 (3, 128)	

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

Table 5.2. Weighted OLS Regression of Inflammation on Physical Abuse and Gender, Add Health Wave I, III, IV (N=9,268)

	Model 1		Model 2		Model 3		Model 4	
	b	SE	b	SE	b	SE	b	SE
Independent Variable								
Physical Abuse (ref. = No)								
Yes	0.074	0.068			0.080	0.067	0.050	0.103
Female			0.255***	0.031	0.256***	0.030	0.256***	0.035
Physical Abuse-x-Female Interaction							0.062	0.143
Anti-inflammatory Medication Use	0.210***	0.031	0.181***	0.031	0.180***	0.031	0.180***	0.030
Constant	0.357***	0.024	0.247	0.027	0.244***	0.029	0.244	0.030
Model Statistics								
R^2	0.008		0.021		0.022		0.022	
F(d.f.) Statistic (Wald test)	13.91 (4,125)***		79.07 (1, 128)***		22.33 (5, 124)***		25.54 (4, 125)***	
ΔR^2	---		---		0.001		0.001	
F (d.f.) Statistic (Wald Test ΔR2)					72.29 (1, 128)***		0.01(1, 128)	

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

Table 5.3. Weighted OLS Regression of Inflammation on Sexual Abuse and Gender, Add Health Wave I, III, IV (N=9,268)

	Model 1		Model 2		Model 3		Model 4	
	b	SE	b	SE	b	SE	b	SE
Independent Variable								
Sexual Abuse (ref. = No)								
Yes	0.205**	0.688			0.131	0.072	0.226	0.170
Female			0.255***	0.030	0.248***	0.031	0.252***	0.031
Sexual Abuse-x-Female Interaction							-0.126	0.180
Anti-inflammatory Medication Use	0.204***	0.031	0.181***	0.031	0.180***	0.031	0.180***	0.031
Constant	0.351***	0.023	0.247	0.028	0.245***	0.028	0.243***	0.028
Model Statistics								
R^2	0.009		0.020		0.021		0.021	
F(d.f.) Statistic (Wald test)			51.73 (2, 127)***		36.49 (3, 126)***		26.19 (4, 125)***	
ΔR^2	---		--		0.012		--	
F (d.f.) Statistic (Wald Test ΔR^2)					64.49 (1, 128)***		0.49 (1, 128)	

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

Table 5.4 presents weighted nested OLS regression models assessing the association between emotional abuse and CRP. These models were used to test the hypotheses that the association between emotional abuse and inflammation persists after accounting for exposure to physical and sexual abuse (Model 2), age, gender and race/ethnicity (Model 3), parents' education and income (Model 4), measures of young adult socioeconomic status (Model 5) and individual health factors that directly influence inflammation (Model 6). In Table 5.4, Model 1, physical and sexual abuse were added to the bivariate model previously presented in Table 5.1, Model 1. Compared to young adults with no history of maltreatment, experiencing emotional abuse six or more times was significantly associated with higher CRP levels. Though physical abuse was not significantly associated with CRP, a history of sexual abuse was significantly associated with higher CRP.

Model 2 adds individual demographic factors to the model. The coefficient for emotional abuse occurring six or more times was reduced by 24% but remained statistically significant. Sexual abuse however, was no longer associated with higher CRP levels net of other variables in the model. Compared to men, women had significantly higher CRP levels and age was positively and significantly associated with CRP. Compared to Whites, Hispanics had higher CRP and Asians had lower CRP; there was no significant difference in CRP levels between Whites and Blacks. With the addition of parents' income and education in Model 3, the coefficient for emotional abuse occurring six or more times reduced by 4% but remained significantly associated with higher CRP levels. The association between age and gender remained while the Hispanic-White difference was no longer statistically significant. Compared to parents' income of $75,000 or more, young adults from lower income families had significantly higher CRP. The effects of parent's education on CRP were more modest in that only those whose highest

education level was high school had higher CRP compared to young adults whose parents had four years of college education or more.

When young adult education and income were added (Model 4) the results remained largely the same as in Model 3; importantly the effects of six or more experiences of emotional abuse was still significantly associated with higher CRP relative to no report of maltreatment. There was a significant association between young adult education level and CRP with all lower levels of education having higher CRP relative to a college education or more. When smoking status, depressive symptoms and BMI, were added (Model 5) the coefficient for six or more experiences of emotional abuse reduced by 22% but remained significantly associated with higher CRP levels. Compared to never smokers, current smokers had significantly higher CRP and CRP increased significantly with increasing BMI. Depressive symptoms however were not significantly associated with CRP. Across all models, use of anti-inflammatory medications was significantly associated with higher CRP. The fully adjusted model explained 23% of the variance in CRP.

5.3 Summary of Key Findings

Results from this chapter showed repeated experiences of emotional abuse (as measured by six or more experiences of emotional abuse) is associated with higher CRP levels compared to no exposure to emotional abuse, even after accounting for exposure to physical and sexual abuse, individual demographic characteristics, childhood socioeconomic characteristics, young adult socioeconomic characteristics, smoking, BMI and depressive symptoms. There was no observed association between physical abuse and CRP and although exposure to sexual abuse was initially significantly associated with higher CRP levels, the effect was not statistically significant when individual demographic characteristics were added to the model. These analyses

revealed significant findings for exposure to emotional abuse in childhood as well as an enduring effect of childhood socioeconomic characteristics on CRP. Parents' income but not education level had a sustained inverse association with inflammation in the individual models as well as in the final model containing all three sub-types of maltreatment. The opposite was observed for young adult socioeconomic status such that education but not income had a consistently inverse association with CRP. Lastly smoking status, depressive symptoms and BMI explained the most variance in CRP. When these three variables were added in Model 5 the percent variance explained increased by 76% (R^2=0.05 in Model 4; R^2=0.23 in Model 6).

Table 5.4. Nested Weighted OLS Regression Models of Inflammation on Emotional Abuse. Add Health Wave I, III, IV (N=9,268)

	Model 1	Model 2	Model 3	Model 4	Model 5
	b	b	b	b	b
Emotional Abuse (ref. = Never)					
Once	0.051	0.038	0.026	0.020	-0.004
Two to five times	0.039	0.007	0.002	0.006	0.001
Six or more times	0.150**	0.114*	0.109*	0.108*	0.084*
Physical Abuse (ref. = Never)					
Yes	-0.060	-0.038	-0.052	-0.057	-0.063
Sexual Abuse (ref. = Never)					
Yes	0.175*	0.099	0.081	0.068	0.044
Female		0.248***	0.249***	0.265***	0.331***
Age (range: 24-32 years)		0.028***	0.029***	0.029*	0.018*
Race/Ethnicity (ref. = White)					
Hispanic-All Races		0.099*	0.036	0.041	0.021
Black/African American		0.063	0.016	0.005	-0.036
Asian		-0.392***	-0.348***	-0.346***	-0.229
Parent's Income (ref. = $75,000+)					
Less than $25,000			0.238***	0.153*	0.100*
$25,000 - $49,999			0.236***	0.171***	0.102*
$50,000 - $74,999			0.111*	0.074	0.048
Parent's Education (ref. = College or More)					
Less than high school			0.122	0.052	0.002
High school graduate			0.152***	0.097*	0.049
Some College			0.051	0.014	-0.011

Table 5.4. Continued. Nested Weighted OLS Regression Models of Inflammation on Emotional Abuse, Add Health Wave I, III, IV (N=9,268)

	Model 1	Model 2	Model 3	Model 4	Model 5
	b	b	b	b	b
Young Adult Education (ref. = College or More)					
Less than High School				0.213**	0.126
High School Graduate				0.213***	0.116*
Some College				0.179***	0.049
Young Adult Income (ref. = $75,000+)					
Less than $25,000				0.071	0.050
$25,000 - $49,999				0.076	0.033
$50,000 - $74,999				0.081	0.048
Smoking (ref. = Never)					
Former Smoker					0.048
Intermittent Smoker					0.014
Current Smoker					0.091*
BMI (range: 14.4-80.4 kg/m^2)					0.070***
Depressive Symptoms (range: 0-15)					-0-005
Anti-inflammatory Medication Use	0.200***	0.170***	0.165***	0.157***	0.130***
Constant	-0.327*	-0.562*	-0.809**	-0.927***	-2.445***
Model Statistics					
R^2	0.011	0.030	0.042	0.050	0.23
F(d.f.) Statistic (Wald test)	11.30 (6, 123)***	15.93 (11, 118)***	12.50 (18, 111)***	11.91 (24, 105)***	49.13 (29, 100***
ΔR^2	0.002	0.019	0.012	0.008	0.18
F (d.f.) Statistic (Wald Test ΔR^2)	3.40 (2, 127)*	20.76 (5,124)***	8.75 (7, 122)***	6.85 (6, 123)***	206.47 (5, 124)***

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

CHAPTER 6

AIM 2 RESULTS: THE EFFECT OF ADULT INTIMATE PARTNER VIOLENCE ON C-REACTIVE PROTEIN

6.1 Overview

The previous chapter examined the ways in which childhood experiences of emotional abuse affect inflammation levels in young adulthood. The current chapter focuses on adult experiences of intimate partner violence and the potential impact on inflammation, as measured by CRP. Using weighted nested regression models, I elaborate the relationship between inflammation and a history of adult intimate partner violence. I describe the relationship between each of the key variables and inflammation and discuss the extent to which they help explain differences in inflammation.

6.2 Effect of adult intimate partner violence on inflammation

Table 6.1 presents results of weighted OLS regressions of CRP on intimate partner violence, gender and intimate partner violence by gender interaction after controlling for the use of anti-inflammatory medication. Model 1 shows that compared to never experiencing intimate partner violence, chronic intimate partner violence (as measured by experiences of intimate partner violence at Waves III and IV) was significantly associated with higher CRP levels. However, those who were currently exposed to intimate partner violence or had been exposed in the past did not differ in CRP levels from young adults with no exposure to intimate partner violence. When both history of intimate partner violence and gender were included in one model (Model 2), the coefficient for chronic intimate partner violence increased by 9% and, as before, women had significantly higher CRP levels compared to men. Model 3 tested the interaction between intimate partner violence and gender. The interaction however was not statistically

significant, therefore, the main effects model (Model 3) was used in nested regression models described below.

Table 6.1. Linear Regression Models of Inflammation on Adult History of Intimate Partner Violence and Gender Add Health Waves I, III, IV (N=9,268)

	Model 1		Model 2		Model 3		Model 4	
	b	SE	b	SE	b	SE	b	SE
Independent Variable								
Intimate Partner Violence (ref. = Never)								
Current IPV Only	0.035	0.050			0.072	0.050	0.069	0.070
Past IPV Only	0.100	0.050			0.089	0.053	0.145	0.090
Chronic IPV	0.205*	0.080			0.224**	0.080	0.277**	0.090
Female			0.255***	0.030	0.260***	0.030	0.275***	0.030
IPV x Gender Interaction								
Current IPV Only -x-Female							0.013	0.100
Past IPV Only-x-Female							-0.107	0.110
Chronic IPV-x-Female							-0.124	0.150
Anti-inflammatory Medication Use	0.205***	0.030	0.181***	0.030	0.179***	0.030	0.180***	0.030
Constant	0.336***	0.030	0.247***	0.030	0.216***	0.030	0.208***	0.030
Model Statistics								
R^2	0.009		0.020		0.023		0.023	
F(d.f.) Statistic (Wald test)	12.73 (4,125)***		51.73 (2,127)***		20.76(5,124)***		13.82 (8,121)***	
ΔR^2	---		0.011		0.014		---	
F (d.f.) Statistic (Wald Test ΔR^2)	---		---		72.34 (1, 128)***		0.60 (3, 126)	

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

Table 6.2 presents the results of sequential model building as described in Chapter 5. In Model 1 experiences of childhood maltreatment (emotional, physical and sexual abuse) are added to the bivariate model presented in Table 6.1, Model 1. The coefficient for chronic intimate partner violence decreased by 5% but remained significantly associated with higher CRP. Compared to young adults reporting no history of emotional abuse, those reporting emotional abuse occurring six or more times as well as those reporting sexual abuse had significantly higher CRP. The addition of maltreatment variables in Model 1 increased the variance in CRP explained slightly but the change was statistically significant as indicated by the F statistic for incremental change (ΔR^2=0.003; F (5, 124)= 3.91; p<0.001). With the addition of individual demographic characteristics in Model 2 the coefficient for chronic intimate partner violence decreased by 7% but remained statistically significant. The coefficient for six or more experiences of emotional abuse decreased by 26% but also remained statistically significant though sexual abuse was no longer significantly associated with CRP. As before women had significantly higher CRP than men and older individuals had higher CRP compared to younger respondents. Compared to Whites, Hispanics had significantly higher CRP levels and Asians had significantly lower CRP levels. When parents' income and education level were added in Model 3, the coefficient for chronic intimate partner violence reduced by 10% but remained significantly associated with CRP. The coefficient for experiencing emotional abuse six or more times reduced slightly (3.7%) but also remained significantly associated with CRP. Lower parents' socioeconomic status during childhood was associated with higher CRP; specifically, a combined parental income of less than $50,000 and having parents with only a high school degree were associated with higher CRP compared to parental incomes of $75,000 of higher or parent education of college or higher, respectively. In Model 4, the association between chronic

intimate partner violence and CRP was no longer statistically significant, although compared to young adults with no history of emotional abuse, six or more experiences of emotional abuse was significantly association with higher CRP. The effects of parents' education on CRP was altered in Model 4. The coefficient for parents whose highest education was a high school degree reduced by 35% but remained statistically significant ($p<0.05$). Young adult education but not income was associated with higher CRP such that compared to earning a college degree or higher, those with less education had higher CRP. These effects were altered substantially with the addition of health factors in Model 5 such that only young adults whose highest education level was a high school degree had significantly higher CRP. In this final model, chronic intimate partner violence was significantly associated with higher CRP levels, indicating suppression by one or more the three health behavior/health status variables. Upon further investigation, smoking and depressive symptoms revealed the significance of chronic intimate partner violence ($p<0.05$). When BMI was entered, the coefficient for chronic intimate partner violence reduced by 8.5% but remained statistically significant ($p<0.05$). Six or more experiences of emotional abuse however, was no longer significantly associated with higher CRP. Current smokers had higher CRP levels compared to never-smokers and CRP increased with increasing BMI. This fully adjusted model explained 23% of the variance in CRP.

Summary of Key Findings

Analyses from this chapter revealed significant findings for the enduring effect of chronic intimate partner violence as well as family background measures of socioeconomic status on CRP. The effect of parents' income during childhood, in particular, persisted despite young adults own achieved socioeconomic status. Young adult education had a significant inverse

association with CRP while young adult income did not affect inflammation after all other explanatory and control variables were included in the model.

Table 6.2. Nested Linear Regression Models of Inflammation on Adult Intimate Partner Violence, Add Health Waves I, III, IV (N=9,268)

	Model 1	Model 2	Model 3	Model 4	Model 5
	b	b	b	b	b
Independent Variables					
Intimate Partner Violence (ref. = Never)					
Current IPV Only	0.025	0.061	0-045	0.021	0.035
Past IPV Only	0.097	0.076	0.058	0.045	0.044
Chronic IPV	0.193*	0.207*	0.186*	0.153	0.149*
Emotional Abuse					
Once	0.043	0.028	0.018	0.015	-0.009
Two to five times	0.033	-0.001	-0.005	0.001	-0.003
Six or more times	0.145***	0.107*	0.103*	0.103*	0.080
Physical Abuse					
Yes	-0.067	-0.044	-0.057	-0.060	-0.067
Sexual Abuse					
Yes	0.172*	0.093	0.077	0.067	0.043
Female		0.254***	0.254***	0.267***	0.334***
Age (range: 24-32 years)		0.028**	0.029***	0.029***	0.017*
Race/Ethnicity (ref. = Non-Hispanic White)					
Hispanic-All Races		0.092*	0.032	0.038	0.017
Non-Hispanic Black		0.053	0.009	0.001	-0.041
Asian		-0.392***	-0.386***	-0.348***	-0.232***
Parent's Income (ref. = $75,000+)					
Less than $25,000			0.234***	0.153*	0.099*
$25,000 - $49,999			0.232***	0.170***	0.100*
$50,000 - $74,999			0.108	0.073	0.047
Parent's Education (ref. = College or More)					
Less than high school			0.116	0.051	0.001
High school graduate			0.149***	0.097*	0.048
Some College			0.049	0.014	-0.012

Table 6.2 Continued. Nested Linear Regression Models of Inflammation on Adult Intimate Partner Violence, Add Health Waves I, III, IV (N=9,268)

	Model 1	Model 2	Model 3	Model 4	Model 5
	b	b	b	b	b
Young Adult Education (ref. = College or More)					
Less than High School				0.199**	0.115
High School Graduate				0.207***	0.111*
Some College				0.174***	0.045
Young Adult Income (ref. = $75,000+)					
Less than $25,000				0.067	0.048
$25,000 - $49,999				0.075	0.033
$50,000 - $74,999				0.080	0.047
Smoking (ref. = Never)					
Former Smoker					0.047
Intermittent Smoker					0.011
Current Smoker					0.086*
Health Status					
BMI (range: 14.4-80.4 kg/m^2)					0.070***
Depressive Symptoms (range: 0-15)					-0.006
Control Variable					
Anti-inflammatory Medication Use	0.199***		0.164***	0.156***	0.130***
Constant	0.309***	-0.580*	-0.820**	-0.928***	-2.446***
Model Statistics					
R^2	0.012	0.032	0.043	0.049	0.230
F(d.f.) Statistic (Wald test)	7.83 (9, 120)***	12.35 (14, 115) ***	10.61 (21, 108) ***	10.47 (27, 102) ***	45.8 (32, 97)***
ΔR^2	0.003	0.012	0.011	0.006	0.279
F (d.f.) Statistic (Wald Test ΔR^2)	3.91 (5, 124)***	20.67 (5, 124)***	8.93 (7, 122)***	6.28 (6, 123)***	208.51 (5, 124)***

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

CHAPTER 7

AIM 3 RESULTS: THE EFFECT OF CHILDHOOD AND/OR ADULT POVERTY ON C-REACTIVE PROTEIN

7.1 Overview

Chapters 5 and 6 focused on the extent to which elevated CRP levels are explained by traumatic experiences in childhood and adulthood respectively. In both chapters, an enduring effect of childhood and young adult socioeconomic status on inflammation was observed. The present chapter first explores experiences of childhood and/or adult poverty (i.e., poverty status) as alternative explanation for elevated inflammation levels. Second I examine whether childhood and/or adult poverty vary synergistically with emotional abuse and intimate partner violence respectively. This is achieved by testing the interactions between childhood and/or adult poverty and emotional abuse, and between childhood and/or adult poverty and intimate partner violence.

I used weighted nested regression models to test the relationship between poverty status and inflammation. Childhood and/or adult poverty was conceptualized to reflect both temporality and duration of poverty and was operationalized as 1) never living in poverty, 2) living in poverty in childhood only, 3) living in poverty in adulthood only or 4) living in poverty in both childhood and adulthood (persistent poverty). I examine two different definitions of poverty for comparison: first, poverty status at 133% of the Federal poverty line more liberal definition of 150% of the FPL. I discuss the extent to which each measure of poverty helps explain differences in inflammation over and above experiences of emotional abuse in childhood and intimate partner violence in adulthood.

First, I hypothesized that young adults who have always lived in poverty (persistent poverty) would have higher CRP levels relative to young adults who never experienced poverty.

Second, emotional abuse is hypothesized to moderate the association between poverty status and inflammation such that the effect of childhood and/or adult poverty on inflammation would be greater for young adults who also experienced emotional abuse. Third, intimate partner violence would moderate the association between childhood and/or adult poverty and inflammation such that the effect of childhood and/or adult poverty on inflammation would be greater for young adults who also experienced intimate partner violence.

7.2 Differences in Inflammation by Poverty at 133% of Federal Poverty Line

Table 7.1 presents results of weighted regressions of CRP on 1) childhood and/or adult poverty at 133% of Federal poverty line, 2) emotional abuse, 3) emotional abuse and childhood and/or adult poverty, and 4) an interaction between childhood and/or adult poverty and emotional abuse after controlling for the use of anti-inflammatory medication. Model 1 is the focal relationship, the effect of poverty status on inflammation. It shows that on average, young adults who lived in poverty during childhood only and young adults who have always lived in poverty have higher mean CRP compared to those who never lived in poverty. The coefficient for persistent poverty is noticeable larger than the coefficient for poverty in childhood. In other words, the difference in inflammation for those who lived in persistent poverty was more than two times the difference for those who lived in poverty in childhood. A post hoc test of these two subpopulations showed mean CRP levels for persistent poverty were significantly higher ($p < 0.05$) than those who lived in poverty as children. This lends support to the hypothesis that respondents who lived in persistent poverty would have the highest CRP levels followed by young adults experiencing poverty in childhood only. Model 2 is the familiar regression of CRP on emotional abuse explored in Chapter 5. When both childhood and/or adult poverty and emotional abuse were included in one model (Model 3), the coefficient for childhood poverty

decreased slightly (2.2%) but remained statistically significant. The coefficient for persistent poverty however, remained unchanged. Model 4 tested for an interaction between childhood and/or adult poverty and emotional abuse. The interaction was not significant (F (6, 133) =5.89; p=0.38).

Table 7.2 presents similar analyses for childhood and/or adult poverty and intimate partner violence. When both childhood and/or adult poverty and intimate partner violence are included in one model (Model 3), the coefficient for childhood poverty decreased by 4.3% but remained statistically significant. The coefficient for persistent poverty also decreased (2.9%) but remained statistically significant. Model 4 tests for an interaction between childhood and/or adult poverty and intimate partner violence. Here again the inclusion of interaction terms did not significantly improve model fit (F (9, 128) = 0.75, p = 0.78) therefore, main effect models are presented in subsequent nested regression models.

Table 7.1. Linear Regression Models of Inflammation on Childhood and/or Adult poverty (133% of Federal Poverty Level) and Emotional Abuse; Add Health Wave I, III, IV (N=9,268)

	Model 1		Model 2		Model 3		Model 4	
	b	SE	b	SE	b	SE	b	SE
Poverty Status – 133% (ref. = Never)								
Childhood Poverty Only	0.138***	0.045			0.135**	0.044	0.019*	0.067
Adult Poverty Only	0.055	0.050			0.050	0.050	0.133	0.070
Persistent Poverty	0.337***	0.077			0.337***	0.076	0.392***	0.112
Emotional Abuse (ref. = Never)								
Once			0.047	0.054	0.039	0.054	0.111	0.073
Two to five times			0.034	0.042	0.028	0.041	0.082	0.053
Six or more times			0.143***	0.044	0.141***	0.043	0.180***	0.053
Childhood Poverty-x-Once							-0.260	0.131
Childhood Poverty-x-Two to five times							-0.151	0.117
Childhood Poverty-x-Six or more times							0.030	0.118
Adult Poverty-x-Once							-0.160	0.183
Adult Poverty-x-Two to five times							-0.148	0.131
Adult Poverty-x-Six or more times							-0.178	0.137
Persistent Poverty-x-Once							-0.001	0.196
Persistent Poverty-x-Two to five times							-0.150	0.158
Persistent Poverty-x-Six or more times							-0.172	0.189
Anti-inflammatory Medication Use	0.207***	0.031			0.201***	0.031	0.202***	0.030
Constant	0.310***	0.024						
R^2	0.014		0.009		0.016		0.017	
F(d.f.) Statistic (Wald test)	15.48(5, 124)***		14.08(4,125)***		12.36(7, 122)***		5.89(6, 113)***	
ΔR^2	--		--		0.002		0.001	
F (d.f.) Statistic (Wald Test ΔR^2)	--		--		3.37(3, 126)*		1.08(9, 128)	

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

Table 7.2. Linear Regression Models of Inflammation on Childhood and/or Adult Poverty (133% of Federal Poverty Level) and Intimate Partner Violence; Add Health Wave I, III, IV (N=9,268)

	Model 1		Model 2		Model 3		Model 4	
	b	SE	b	SE	b	SE	b	SE
Poverty Status – 133% (ref. = Never)								
Childhood Poverty Only	0.138***	0.045			0.132**	0.045	0.157**	0.057
Adult Poverty Only	0.055	0.050			0.044	0.050	0.034	0.063
Persistent Poverty	0.337***	0.077			0.327***	0.077	0.041***	0.100
Intimate Partner Violence (ref. = Never)								
Current IPV			0.035	0.050	0.024	0.050	0.030	0.063
Past IPV			0.100	0.050	0.090	0.052	0.120	0.070
Chronic IPV			0.205*	0.080	0.175*	0.080	0.300**	0.094
Childhood Poverty-x-Current IPV							-0.040	0.144
Childhood Poverty-x-Past IPV							-0.062	0.147
Childhood Poverty-x-Chronic IPV							-0.451	0.240
Adult Poverty-x-Current IPV							-0.015	0.132
Adult Poverty-x-Past IPV							-0.004	0.155
Adult Poverty-x-Chronic IPV							0.046	0.209
Persistent Poverty-x-Current IPV							-0.128	0.201
Persistent Poverty-x-Past IPV							-0.276	0.215
Persistent Poverty-x-Chronic IPV							-0.447	0.224
Anti-inflammatory Medication Use	0.207***	0.031	0.205***	0.030	0.021***	0.031	0.207***	0.031
Constant	0.310***	0.024	0.336***	0.030	0.292***	0.027	0.283***	0.028
R^2	0.014		0.009		0.016		0.017	
F(d.f.) Statistic (Wald test)	15.48(5, 124)***		12.73 (4, 125)***		10.14(7,122)***		5.31(16, 113)***	
ΔR^2	--		--		0.002		0.001	
F(d.f.) Statistic (Wald test ΔR^2)	--		--		2.21(3,126)		0.75(9, 128)	

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

Table 7.3 presents nested regression models for the bivariate association between poverty status and CRP (Model 1), adjusting for childhood experiences of physical abuse, sexual abuse and adult intimate partner violence (Model 2), young adult demographic characteristics (Model 3), parents' educational background (Model 4), young adult educational background (Model 5) and individual health factors (smoking, BMI and depressive symptoms) that directly influence inflammation (Model 6). All models presented in the table control for the use of anti-inflammatory drugs.

Model 1 presents the bivariate association between childhood and/or adult poverty and CRP previously described. The addition of histories of emotional, physical and sexual in childhood and intimate partner violence in adulthood (Model 2), reduced the coefficients for the childhood only and persistent poverty variables by 5% and 4% respectively but did not change their statistical significance. Compared to young adults who never experienced maltreatment in childhood, having six or more experiences of emotional abuse was associated with significantly higher levels of CRP. Chronic intimate partner violence was also significantly associated with higher levels of CRP compared to young adults with no history of intimate partner violence. The addition of demographic characteristics in Model 3 led to a slight reduction in the coefficient for childhood poverty (1.5%) and in the coefficient for persistent poverty (3.5%), though both remained statistically significant. The coefficient for six or more experiences of emotional abuse remained statistically significant but was reduced substantially by 28.0%. The coefficient for chronic intimate partner violence however increased by 13% and remained statistically significant. As previously observed, age was significantly and positively associated with inflammation such that inflammation increased with age. Women had significantly higher CRP compared to men and Asian respondents had lower CRP levels compared to Whites.

Table 7.3. Nested Weighted OLS Regression Models of Inflammation on Childhood and/or Adult Poverty (133% of Federal Poverty Level); Add Health Wave I, III, IV (N=9,268)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	b	b	b	b	b	b
Poverty Status (ref. = Never)						
Childhood Poverty Only	0.138**	0.131**	0.133**	0.093	0.069	0.073*
Adult Poverty Only	0.055	0.038	0.007	-0.007	-0.044	-0.053
Persistent Poverty	0.337***	0.324***	0.289***	0.237**	0.189*	0.128
Emotional Abuse (ref. = Never)						
Once		0.039	0.026	0.021	0.018	-0.007
Two to five times		0.033	-0.001	-0.004	0.002	-0.004
Six or more times		0.154**	0.111*	0.113*	0.113*	0.086*
Physical Abuse (ref. = Never)						
Yes		-0.078	-0.053	-0.056	-0.059	-0.066
Sexual Abuse (ref. = Never)						
Yes		0.142	0.068	0.070	0.058	0.037
Intimate Partner Violence (ref. = Never)						
Current IPV Only		0.016	0.056	0.050	0.027	0.039
Past IPV Only		0.088	0.070	0.064	0.049	0.046
Chronic IPV		0.167*	0.192*	0.186*	0.156*	0.154*
Female			0.250***	0.248***	0.268***	0.337***
Age (range: 24-32 years)			0.029***	0.029***	0.028***	0.017*
Race/Ethnicity (ref. = White)						
Hispanic-All Races			0.048	0.033	0.033	0.009
Black/African American			-0.003	0.002	-0.003	-0.046
Asian			-0.400***	-0.384***	-0.352***	-0.237***

Table 7.3. Continued. Nested Weighted OLS Regression Models of Inflammation on Childhood and/or Adult Poverty (133% of Federal Poverty Level); Add Health Wave I, III, IV (N=9,268)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	b	b	b	b	b	b
Parent's Education (ref. = College or More)						
Less than high school				0.137*	0.057	
High school graduate				0.199***	0.128**	
Some College				0.095*	0.043	
Young Adult Education (ref. = College or More)						
Less than High School					0.223**	0.127
High School Graduate					0.240***	0.128**
Some College					0.203***	0.060
Smoking (ref. = Never)						
Former Smoker						0.044
Intermittent Smoker						0.008
Current Smoker						0.090*
BMI						0.070***
Depressive Symptoms						-0.006
Anti-inflammatory Medication Use	0.207***	0.201***		0.168***	0.166***	0.131***
Constant	0.310***	0.268***	-0.635*	0.710**	-0.787	-2.377***
Model Statistics						
R^2	0.014	0.019	0.037	0.042	0.048	0.230
F(d.f.) Statistic (Wald test)	17.18 (4, 125)***	8.08 (12,117)***	11.46 (17, 112)***	10.81 (21, 108)***	11.26 (24, 105)***	50.56 (29,100)***
ΔR^2	---	0.005	0.018	0.005	0.006	0.182
F(d.f.) Statistic (Wald test ΔR^2)	---	3.63 (5,124)**	19.88 (5, 124)***	5.91 (4, 125)***	12.17 (3, 126)***	200.95(5, 124)***

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

Adjusting for parents' educational background (Model 4) altered the effect of childhood poverty on inflammation such that it was no longer significant and while the coefficient for persistent poverty was reduced by 18%, it remained statistically significant. Emotional abuse occurring six or more times and chronic intimate partner violence also remained significantly associated with higher CRP levels. Previously observed associations between demographic characteristics and inflammation remain unaltered by the addition of parents' educational background. Compared to parents who earned college degrees or more, lower levels of education were significantly and positively associated with higher CRP levels, suggesting a protective effect of earning a college education or higher.

Adding young adult educational background in Model 5 altered the significance level of childhood poverty. Relative to never living in poverty, childhood poverty was no longer significantly associated with CRP. Although the coefficient for persistent poverty decreased by nearly 5% it remained statistically significantly associated with CRP as did six or more experiences of emotional abuse and chronic intimate partner violence. The effects of demographic variables remained unchanged from the previous model. The addition of young adult educational background rendered the effect of parents' education (less than high school and some college education) on inflammation not significant, however having parents who earned no more than a high school degree remained significantly associated with higher CRP levels. Young adults with less than a high school education, those who earned a high school degree as well as those with some college education all had significantly higher CRP levels compared to earning a college degree or more.

Interestingly, after adjusting for smoking, BMI and depressive symptoms in Model 6, the difference in inflammation between childhood poverty and never living in poverty became statistically significant while the difference between persistent poverty and never living in poverty became non-significant. To better understand which of the three variables was driving these changes, each variable was entered individually to Model 5. Adding smoking status alone did not significantly improve the fit of the model (ΔR^2=0.0006, F (3, 126), p>0.05); the coefficient for childhood poverty decreased slightly but the coefficient for persistent poverty remained the same. Adding depressive symptoms did not change the size or significance of the poverty coefficients, nor did the addition significantly improve model fit (ΔR^2=0.0005, F (1, 137), p>0.05). When BMI was added to Model 5, however, the variance in CRP explained increased by nearly 80% (ΔR^2=0.131, F (1, 128), p<0.001), and the effect of persistent poverty on inflammation was no longer statistically significant, suggesting BMI may mediate the association between persistent poverty and CRP but not the association between childhood poverty and CRP. Table 7.4 below shows mean BMI by childhood and/or adult poverty and lends further support to this assertion. Young adults who lived in persistent poverty had the highest BMI levels at 30.10 kg/m^2 (obese), followed by young adults who lived in poverty as children (28.96 kg/m^2); those who never lived in poverty had the lowest BMI levels at 28.18 kg/m^2.

With regard to the other variables in Model 5 of Table 7.3, the addition of smoking status, BMI and depressive symptoms increased the coefficient for women by 7.0%, and the difference in inflammation between having parents with college degrees or more and having parents with no more than a high school degree became non-significant. Similarly, the difference in inflammation between young adults with less than a high school degree compared to a college

degree and those with some college education compared to a college degree, also became non-significant. Current smokers had significantly higher CRP levels than never smokers, CRP increased with increasing BMI and the effect of depressive symptoms on CRP was not statistically significant.

Table 7.4. Weighted Mean BMI (kg/m^2) by Childhood and/or Adult Poverty (133% of Federal Poverty Level)Add Health Waves I, III, IV (N=9,268)

Variables	Mean[a]	SE	P-Value
Persistent Economic Status (Waves 1 and IV)			
133% of Federal Poverty Level			*
Never lived in Poverty	28.18	0.15	
Poverty in Childhood	28.96	0.25	
Poverty in Adulthood	28.67	0.37	
Persistent Poverty	30.10	0.45	

Note: Wave IV sample weights used; [a]Adjusted Wald test; *p<.05

7.3 Differences in Inflammation by Poverty at 150% Federal Poverty Line

Table 7.5 presents results of weighted regressions of CRP on 1) childhood and/or adult poverty at 150% of FPL, 2) emotional abuse, 3) emotional abuse and poverty status, and 4) an interaction between poverty status and emotional abuse after controlling for the use of anti-inflammatory medication. Similar to the 133% FPL model described above, Model 1 shows that on average, young adults who lived in poverty as children had significantly higher CRP levels compared to respondents who never lived in poverty. Persistent poverty was also significantly associated with higher levels of CRP. When both emotional abuse and childhood and/or adult poverty were included in one model, the coefficient for childhood poverty decreased by 4.4% while the effect of persistent poverty decreased by 1.1%. Both remained statistically significant.

Again, the interaction between emotional abuse and poverty status was not significant (F (9, 128)=0.96, p=0.478).

Table 7.6, shows the regression of CRP on poverty status (Model 1), intimate partner violence (Model 2), poverty status and intimate partner violence (Model 3) and a test of the an interaction between poverty status and intimate partner violence (Model 4). Model 1 shows childhood poverty is significantly associated with higher CRP levels and persistent poverty is also significantly associated with higher mean CRP levels. This first model explains 1.3% of the variance in CRP. Model 2 is the zero-order association between intimate partner violence and CRP previously presented in Table 6.1, Model 1. When both poverty status and intimate partner violence were included in one model, the coefficient for childhood poverty decreased by 6% but remained statistically significant. The coefficient for persistent poverty decreased by 3.7% but also remained statistically significant. Model 4 shows the test for the interaction between poverty status and intimate partner violence was not statistically significant (F (9, 128)=0.62; p=0.781), indicating the inflammatory consequences of intimate partner violence are not contingent on exposure to poverty. As with the 133% poverty measure, main effects models (without an interaction term) are presented in nested regressions presented below.

Table 7.7 presents results from nested regression models of poverty status and CRP (Model 1; bivariate model), adjusting for childhood experiences of physical abuse, sexual abuse and adult intimate partner violence (Model 2), young adult demographic characteristics (Model 3), parents' educational background (Model 4), young adult educational background (Model 5) and individual health factors (smoking, BMI and depressive symptoms) that directly influence inflammation (Model 6). As before, all models presented in the table controlled for the use of anti-inflammatory drugs. When physical abuse, sexual abuse and adult intimate partner violence

were included in the model (Model 2), the coefficient for both childhood poverty and persistent poverty decreased (9.6% and 5.2% respectively) but remained statistically significant. Childhood and/or adult poverty along with experiences of childhood maltreatment and adult intimate partner violence explained 1.3% of the variance in CRP. Both childhood poverty and persistent poverty remained significantly associated with higher mean CRP levels when demographic characteristics were added in Model 3. After adjusting for parents' education level (Model 4), childhood poverty was no longer statistically significantly associated with higher CRP levels. The coefficient for persistent poverty however decreased by 24% but remained associated with significantly higher mean CRP levels. The inclusion of young adult education level (Model 5) however altered the effect of persistent poverty on CRP such that the association was no longer statistically significant, and this model explained nearly 5% of the variance in CRP. Adjusting for smoking status, BMI and depressive symptoms in Model 6 did not alter the effect of childhood poverty nor persistent poverty on CRP. Both were non-significant. All other covariates were identical to previous models such that in the fully adjusted model (Model 6), emotional abuse occurring six or more times was significantly associated with CRP, as was chronic intimate partner violence. Women had higher CRP compared to men, CRP increased with increasing age and Asian respondents had significantly lower CRP compared to Whites. While parents' education level was not significantly associated with CRP, young adults who earned no more than a high school degree had significantly higher mean CRP levels. Current smokers had significantly higher CRP compared to never-smokers, CRP increased with increasing BMI and effect of depressive symptoms on CRP was not significant.

Summary of key findings

Analyses from this chapter revealed levels of inflammation differ by timing and duration of exposure to poverty at 133% of FPL but that these are independent effects rather than conditional. Young adults exposed to poverty in childhood, as well as those who lived in persistent poverty had significantly higher CRP levels compared to those who never lived in poverty. The differences between childhood poverty and never living in poverty and between persistent poverty and never living in poverty persisted after accounting for exposure to emotional, physical and sexual abuse, demographic as well as parents and young adult education levels. Differences between persistent poverty and never living in poverty were no longer significant in the fully adjusted model and seemed to be accounted for by differences in BMI, indicating a possible mediation by BMI. Differences between childhood poverty and never living in poverty however remained statistically significant indicating the effect of childhood poverty does not operate through BMI.

A different pattern emerged when childhood and/or adult poverty was operationalized using a more liberal measure of poverty at 150% of FPL. Though childhood poverty and persistent poverty were initially significantly associated with elevated CRP levels, differences in parents' education seem to account for the association between childhood poverty and inflammation. Adjusting for young adult education rendered the association between persistent poverty and inflammation non-significant.

These analyses also revealed experiencing emotional abuse six or more times as well as experiencing chronic intimate partner violence are consequential for inflammation even after adjusting for childhood and/or adult poverty. Additionally, the coefficients in the fully adjusted model at 133% of FPL are nearly identical to the coefficients in the fully adjusted model at 150%

of FPL. As previously noted, age inflammation levels increased with increasing age, women had higher inflammation compared to men and Asian respondents had significantly lower CRP levels compared to Whites.

Table 7.5. Linear Regression Models of Inflammation on Childhood-Adulthood Poverty Status (150% of Federal Poverty Level) and Emotional Abuse; Add Health Wave I, III, IV (N=9,268)

	Model 1		Model 2		Model 3		Model 4	
	b	SE	b	SE	b	SE	b	SE
Poverty Status – 150% (ref. = Never)								
Childhood Poverty Only	0.114**	0.039			0.109**	0.040	0.139*	0.055
Adult Poverty Only	0.098	0.051			0.093	0.051	0.096	0.068
Persistent Poverty	0.266***	0.058			0.263***	0.058	0.328***	0.085
Emotional Abuse (ref. = Never)								
Once			0.047	0.054	0.037	0.053	0.103	0.076
Two to five times			0.034	0.042	0.028	0.041	0.056	0.057
Six or more times			0.143***	0.044	0.137**	0.043	0.133*	0.054
Childhood Poverty x Once							-0.263*	0.123
Childhood Poverty x Two to five times							-0.055	0.107
Childhood Poverty x Six or more times							0.057	0.117
Adult Poverty x Once							-0.108	0.166
Adult Poverty x Two to five times							0.037	0.134
Adult Poverty x Six or more times							0.017	0.170
Persistent Poverty x Once							-0.030	0.143
Persistent Poverty x Two to five times							-0.216	0.153
Persistent Poverty x Six or more times							-0.097	
Anti-inflammatory Medication Use	0.202***	0.031			0.200***	0.053	0.198***	0.030
Constant	0.303	0.025			0.275***	0.030	0.263***	0.033
R^2	0.013		0.009		0.016		0.016	
F(d.f.) Statistic (Wald test)	15.89 (4, 125)***		14.08(4,125)***		5.64 (16, 113)***		5.64 (16, 113)***	
ΔR^2	--		--		0.007		--	
F(d.f.) Statistic (Wald test ΔR^2)	--		--		3.48 (3, 126)*		0.96 (9, 128)	

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

Table 7.6. Linear Regression Models of Inflammation on Childhood-Adulthood Poverty Status (150% of Federal Poverty Level) and Intimate Partner Violence; Add Health Wave I, III, IV (N=9,268)

	Model 1		Model 2		Model 3		Model 4	
	b	SE	b	SE	b	SE	b	SE
Poverty Status – 150% (ref. = Never)								
Childhood Poverty Only	0.114**	0.039			0.107**	0.040	0.146**	0.050
Adult Poverty Only	0.098	0.051			0.087	0.050	0.077	0.059
Persistent Poverty	0.266***	0.058			0.256***	0.058	0.310***	0.074
Intimate Partner Violence (ref. = Never)								
Current IPV Only			0.035	0.050	0.021	0.049	0.042	0.067
Past IPV Only			0.100	0.050	0.089	0.052	0.138*	0.070
Chronic IPV			0.205*	0.080	0.171*	0.078	0.238*	0.100
Childhood Poverty-x-Current IPV							-0.053	0.137
Childhood Poverty-x-Past IPV							-0.134	0.147
Childhood Poverty-x-Chronic IPV							-0.312	0.226
Adult Poverty-x-Current IPV							-0.021	0.135
Adult Poverty-x-Past IPV							-0.039	0.144
Adult Poverty-x-Chronic IPV							0.165	0.210
Persistent Poverty-x-Current IPV							-0.083	0.143
Persistent Poverty-x-Past IPV							-0.184	0.149
Persistent Poverty-x-Chronic IPV							-0.295	0.192
Anti-inflammatory Medication Use	0.202***	0.031	0.205***	0.030	0.202***	0.031	0.203***	0.031
Constant	0.303	0.025	0.336***	0.030	0.286***	0.028	0.276***	0.029
R^2	0.013		0.009		0.014		0.016	
F(d.f.) Statistic (Wald test)	15.89 (4, 125)***		12.73 (4, 125)***		9.45 (7, 122)***		4.96 (16, 113)***	
ΔR^2	--		--		0.005		--	
F(d.f.) Statistic (Wald test ΔR^2)	--		--		2.21 (3, 126)***		0.62 (9, 128)	

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

Table 7.7. Nested Weighted OLS Regression Models of Inflammation on Childhood-Adulthood Poverty Status (150% of Federal Poverty Level); Add Health Wave I, III, IV (N=9,268)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	b	b	b	b	b	b
Poverty Status (ref. = Never)						
Childhood Poverty Only	0.114**	0.103**	0.107*	0.061	0.032	0.039
Adult Poverty Only	0.098	0.082	0.056	0.041	-0.002	-0.034
Persistent Poverty	0.266***	0.252***	0.219***	0.166*	0.113	0.095
Emotional Abuse (ref. = Never)						
Once		0.037	0.024	0.019	0.016	-0.008
Two to five times		0.032	-0.001	-0.004	0.002	-0.004
Six or more times		0.150**	0.108*	0.111*	0.111*	0.084*
Physical Abuse (ref. = Never)						
Yes		-0.076	-0.052	-0.055	-0.058	-0.065
Sexual Abuse (ref. = Never)						
Yes		0.141	0.067	0.071	0.061	0.038
Intimate Partner Violence						
Current IPV Only		0.013	0.054	0.048	0.026	0.039
Past IPV Only		0.087	0.070	0.063	0.048	0.046
Chronic IPV		0.163*	0.188*	0.182*	0.154	0.154*
Female			0.249***	0.247***	0.267***	0.336***
Age (range: 24-32 years)			0.029***	0.029***	0.028**	0.017*
Race/Ethnicity (ref. = White)						
Hispanic-All Races			0.054	0.038	0.039	0.013
Black/African American			-0.000	0.008	0.005	-0.043
Asian			-0.397***	-0.380***	-0.348***	-0.235***
Parent's Education (ref. = College or More)						
Less than high school				0.150*	0.074	0.005
High school graduate				0.202***	0.134**	0.066
Some College				0.096*	0.047	0.005

Table 7.7. Continued. Nested Weighted OLS Regression Models of Inflammation on Childhood-Adulthood Poverty Status (150% of Federal Poverty Level); Add Health Wave I, III, IV (N=9,268)

	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
	b	b	b	b	b	b
Young Adult Education (ref. = College or More)						
Less than High School					0.225**	0.128
High School Graduate					0.240***	0.128**
Some College					0.203***	0.060
Smoking (ref. = Never)						
Former Smoker						0.043
Intermittent Smoker						0.007
Current Smoker						0.089*
BMI (range: 14.4-80.4 kg/m^2)						0.070***
Depressive Symptoms (range: 0-15)						-0.007
Anti-inflammatory Medication Use	0.202***	0.197***	0.164***	0.163***	0.157***	0.131***
Constant	0.303***	0.263***	-0.639*	-0.709**	-0.779**	-2.371***
Model Statistics						
R^2	0.013	0.017	0.035	0.040	0.047	0.230
F(d.f.) Statistic (Wald test)	15.89(4,125)***	7.30 (12, 117)***	11.50 (17,112)***	10.58(21,108)***	11.22(24, 105)***	49.94(29,100)***
ΔR^2	--	0.004	0.018	0.005	0.007	0.183
F(d.f.) Statistic (Wald test ΔR^2)	--	2.79 (8, 121)***	19.76 (5,124)***	6.13(4, 125)***	11.53 (3,126)***	210.14 (5, 124)***

Note: b = unstandardized regression coefficient; SE = standard error; *p<.05; **p<.01; ***p<.001

CHAPTER 8

DISCUSSION

8.1 Introduction

The purpose of this dissertation was to contribute to the growing research on the effects of negative childhood experiences and adult traumatic experiences on inflammation, an early risk marker for cardiovascular disease. Inflammation is associated with the incidence and progression of cardiovascular disease so that understanding the ways in which adversities in childhood and in adulthood contribute to inflammation independent of other known risk factors helps to explain the higher incidence of cardiovascular disease among adults with a history of maltreatment and also among adults exposed to intimate partner violence. First, I examined the effects of emotional abuse in childhood on CRP levels in young adulthood and the extent to which these effects are moderated by gender. Second, I tested the effect of adult intimate partner violence on CRP and the extent to which the effect of intimate partner violence on CRP was contingent on gender. Lastly, I examined the influence of exposure to poverty in childhood and/or adulthood on CRP and tested for a synergistic effect of 1) childhood and/or adult poverty and 2) childhood and/or adulthood and intimate partner violence on inflammation.

Data for the study come from Add Health, a nationally representative study of young adults in the United States. The analytic sample included 9,268 young adults ages 24 to 32 who were interviewed at Waves I, III and IV of the study. By design, approximately 71% of respondents were White, 14% were African American, just over 11% were Hispanic with Asians making up nearly 4% of the sample. This is highly educated sample with nearly three in four young adults reporting some college education or more. One in three grew up in households earning $50,000 or more and over 50% report earning over $50,000 in young adulthood. Percent

smoking (28%) is above the national percentage of 24.1% however BMI (28.5 kg/m^2) is consistent national estimates for US Adults (CDC, 2015 MMWR).

The most commonly reported sub-type of child maltreatment was emotional abuse with 46% of the sample reporting experiencing at least one incident. Approximately 18% of young adults reported experiencing physical abuse at least once. These estimates are slightly higher than community surveys reporting 4-16% of adults have a history of physical abuse (Gilbert et al., 2009). Consistent with prior estimates, approximately 5% of young adults reported experiencing sexual abuse at least once before age eighteen (Finkelhor, 2013; Finkelhor, 2009). Compared to physical and sexual abuse, fewer studies assess prevalence of emotional abuse with the few available estimates ranging from 8.7% to 36% (Spinazzola et al., 2014; Tietjen, 2016). Emotional abuse was more common among women (53%) compared to men (40%) and a larger proportion of women reported sexual abuse (7.5% compared to 2%). The proportion of men and women reporting physical abuse however was similar with just over 17% of each group reporting exposure to physical abuse.

Thirty percent of this young adult sample reported experiencing intimate partner violence in a past relationship, current relationship or both. Fourteen percent were in current relationships characterized by intimate partner violence, just over 11% experienced intimate partner violence in past relationships while approximately 4% were chronically exposed to intimate partner violence. The finding that 14% of young adults experienced intimate partner violence in the 12-months prior to data collection is higher than national estimates (Breiding, Chen, & Black, 2015; Tjaden & Thoennes, 2000). Compared to women, a higher percentage of men reported experiencing intimate partner violence in current relationships, although more women reported past experiences with intimate partner violence. Finally mean CRP for this sample (2.58 mg/L) is

below the clinically significant level of 3 mg/L and given the relatively young age of the sample, this is to be expected.

The major findings of the dissertation are:

1. Emotional abuse occurring six or more times is significantly associated with higher CRP levels, net of exposure to physical abuse, sexual abuse, demographic characteristics, parents socioeconomic status, young adult socioeconomic status as well as lifestyle characteristics including smoking, depressive symptoms.
2. Gender and emotional abuse have significant but independent effects on CRP, suggesting the magnitude of differences in inflammation by emotional abuse is similar for men and women.
3. A history of chronic intimate partner violence is associated with higher CRP levels net of demographic characteristics, childhood socioeconomic status, young adult socioeconomic status, young adult health factors and young adult histories of childhood emotional, physical and sexual abuse.
4. Gender and chronic intimate partner violence have significant but independent effects on CRP, suggesting the magnitude of differences in inflammation by emotional abuse is similar for men and women.
5. At 133% FPL, young adults who experienced poverty in childhood had significantly higher levels of CRP than those who never experienced poverty in fully adjusted models.
6. At 133% FPL, young adults who lived in persistent poverty had higher CRP levels compared to those who never lived in poverty, however this difference appeared to be fully accounted for by BMI.

7. While young adults who experienced childhood poverty at 150% FPL, had significantly higher levels of CRP than those who never experienced poverty, these differences appeared to be fully accounted for by parents' education level.
8. Although young adults who experienced persistent poverty at 150% FPL, had significantly higher levels of CRP compared to those who never experienced poverty, this difference appeared to be fully accounted for by young adult education level.

8.2 Aim 1: The Effect of Emotional Abuse on Inflammation

The finding that experiencing emotional abuse repeatedly is significantly associated with inflammation is consistent with a handful of studies linking emotional abuse to inflammation. In a study of twins middle aged twins, Rooks and colleagues (2013) examined the effect of physical, sexual and emotional abuse on CRP. They found that of the three different sub-types of abuse assessed, emotional abuse was more strongly related to clinically significant levels of CRP (3mg/L or more). Matthews and colleagues (2013) found women who reported a history of emotional abuse initially had significantly higher CRP however the association became non-significant after adjusting for BMI. They found however, emotional abuse was significantly related to percent change in CRP over a seven-year period. A third study study found a positive association between childhood emotional abuse and NFkB, a molecule responsible for the expression of pro-inflammatory genes including cytokines and chemokines (Pace et al, 2012). There was no association however between an index measure of child maltreatment (assessing physical abuse, sexual abuse and neglect) and NFkB. Although NFkB is not commonly used to assess inflammation, there is some empirical evidence supporting the role of NFkB activation in chronic conditions including atherosclerosis and cardiovascular disease (Urtasun et al, 2012). Additionally, in a recent systematic review of the literature, Cuelho and colleagues (2014)

concluded the role of NFkB in mediating inflammatory response to negative childhood experiences should be further explored. Although the independent effect of emotional abuse on inflammation is rarely assessed in relation to inflammation, emotional abuse has been shown to be associated with PTSD and chronic stress (Yehuda, 2000). PTSD in turn is known shown to disrupt the HPA-axis, resulting in lower cortisol levels that then lead to an inability to inhibit inflammatory response. This process mirrors the type of HPA-axis dysregulation commonly reported among victims of maltreatment (Miller, Chen & Parker, 2011). Further, there is growing evidence showing elevated inflammation levels in patients with PTSD (Boscarino, 2004; Gander et al., 2006; Otoole & Catts, 2008). Emotional abuse is also linked to depression, anxiety, externalizing behavior, negative self-esteem and self-punishing behaviors (Hart, Brassard, & Karlson, 1996; O'Dougherty, Crawford & Del Castillo, 2009).

There are two primary aspects of emotional abuse that may help shed light on findings from this study. Unlike physical abuse, emotional abuse does not result in physical evidence in the form of bruises or scars, nor does it carry strong social/moral taboo like sexual abuse. By its very nature then, emotional abuse is difficult to identify, meaning it often hides in plain sight (Hart & Glaser, 2011; Spinazzola et al., 2014). This could result in failure to intervene such that children fall victim to emotional abuse over a longer period of time, unable to cope. A second factor is, children may occasionally encounter mild or sporadic experiences of certain aspects of emotional abuse (e.g. denigration) so that it is unlikely single incidents constitute emotional abuse or are harmful in the same way physical and sexual abuse may result in injury (Claussen & Crittenden, 1991). Findings from this study support both lines of thought in that significant effects were found only for the group of respondents who experienced emotional abuse six or more times.

In this analytic sample, physical abuse was not significantly associated with CRP. Studies relating physical abuse to CRP report a positive association (Bertone-Johnson et al, 2012; Matthews et al., 2013) however a study using Add Health data to study sexual orientation and gender differences in inflammation found a significant negative association between physical abuse and inflammation (Everet et al., 2014) and Carpenter and colleagues (2012) did not find a significant association between physical abuse and inflammation in a community sample of adult women (Carpenter et al., 2012). Although the association between sexual abuse and CRP was significant at the bivariate level, the association was fully accounted for by gender. Bertone-Johnson et al., (2012) found a positive association between sexual abuse and CRP in a population of women reporting sexual abuse in adolescence. However a higher proportion (41%) reported experiencing unwanted touching or forced sex. Since only 5% of this sample reported experiencing sexual abuse this lack of finding may be due to lack of power to detect differences.

8.3. Aim 2: The Effect of Adult Intimate Partner Violence Inflammation

It was hypothesized that young adults experiencing intimate partner violence in past relationships as well as in the twelve months prior to data collection (current exposure) would have higher levels of inflammation respectively. This hypothesis was partially supported in that young adults experiencing chronic intimate partner violence had higher CRP. Respondents in past relationships characterized by intimate partner violence as well as those in current violent relationships did not have higher CRP levels. These findings are partially consistent with the two previous studies linking intimate partner violence to CRP. In a population of middle-aged women, Fernandez-Botran and colleagues (2011) found a history (ever exposed) of intimate partner violence significantly predicted higher CRP in a community sample of post-menopausal women. The second study, also among middle-aged women found exposure to intimate partner

violence, stalking in particular was significantly associated with higher CRP levels. Both studies assessed intimate partner violence using the widely validated Conflict Tactics Scale, however neither distinguished between past, current nor chronic intimate partner violence. It is surprising that neither current nor past exposure to intimate partner violence was significantly associated with CRP, especially given fourteen percent of the sample reported experiencing intimate partner violence in current relationships and just over eleven percent reported past exposure. This compared to just over four percent reporting chronic exposure. In their meta-analysis of HPA reactivity, Miller, Chen and Zhou (2007) note chronicity as one of the key factors that govern HPA reactivity and the resulting influence on CRP. Perhaps effects of intimate partner violence on CRP may not yet be observed among young adults exposed to intimate partner violence in the twelve months prior to data collection. Young adults with past histories of intimate partner violence may have accessed services to cope with the trauma, however because this mechanism was not tested there is a need to further explore the differential effects of past, current and chronic exposure to intimate partner violence.

8.4 Other Influences on Inflammation

Although the focus of the first two aims was on the influence of emotional abuse and intimate partner violence on CRP, a few covariates emerged as interesting. Notably, parents' income persisted in its negative association with inflammation regardless of young adult's own income. Interestingly, while parents' income was consistently associated with higher inflammation, parent's education was not. This suggests access to material resources may be more strongly related to inflammation. For young adults, education level rather than income was consistently associated with inflammation. This could be due to the young age of this sample in that adults between the ages of 24 to 32 may still be at the beginning of their careers so that

income is less meaningful for health. The enduring effects of parents income is line with numerous studies reporting an association between both childhood and adult socioeconomic status and CRP (Miller et al, 2009; Nazmi & Victora, 2007; Owen, 2003; Taylor et al., 2006). In a population of older adults enrolled in the Atherosclerosis Risk in Communities (ARIC) study, Pollitt and colleagues (2008) used retrospective childhood and prospective adult measures of socioeconomic status to assess cumulative burden of socioeconomic status. Similar to findings from the present dissertation, they found both childhood and adult measures of socioeconomic status were associated with CRP. Although ARIC is a study of older adults and Add Health studies young adults, these similar findings suggest these relationships may operate similarly over the life course.

For each of the two aims described above, smoking, BMI, depressive symptoms and anti-inflammatory drugs were assessed as known risk factors for high inflammation levels. BMI was positively associated CRP, and this association was virtually identical in all models. Circulating CRP is considered a marker of obesity related inflammation (Yudkin et al., 1999) and studies examining causal direction between BMI and CRP have shown increased BMI causes increased CRP levels (Timpson et al., 2005). The strong association between BMI and CRP in the present study is in line with previous work from population based surveys (Slopen et al., 2013; Danese et al., 2008) as well as community and clinic samples (Carpenter et al., 2012; Hepgul et al., 2012; Matthews et al., 2013). As previously reported (Slopen, 2012), BMI levels in the Add Health sample are high and in this analytic sample nearly sixty percent are overweight and approximately 7% are obese. Though this is a young sample, sixteen 60% had CRP levels above the clinically significant cut point of 3.0 mg/L. Since obesity has been previously shown as a cause of elevated CRP, the high proportion of overweight and obese in this young adult

population could mean some also have abnormally high CRP levels, leaving them vulnerable for chronic diseases with inflammatory origin. Consistent with prior studies current smoking and the use of anti-inflammatory medications were both strongly associated with inflammation (Danese, 2007; Slopen, 2010; Taylor et al., 2006).

In this analytic sample, mean number of depressive symptoms was relatively low (2.5; range 0-15) and did not predict CRP in bivariate models. The lack of association between depressive symptoms and CRP is consistent with some prior research (Steptoe et al., 2003; Pace & Heim, 2011) but could partially reflect the low symptom levels in the present sample as most report a positive association between depressive symptoms and CRP. (Danese, 2008; Gouin et al., 2012; Miller et al., 2003; Slopen et al., 2013; Taylor et al., 2006).

8.5 Differences in Inflammation by Poverty Status in Childhood/and or Adulthood

At 133% above the federal poverty line, young adults who experienced poverty in childhood had significantly higher levels of CRP compared to those who never lived in poverty net of a history of abuse in childhood, adult intimate partner violence, demographic characteristics, educational background, health behavior and health characteristics. Although living in persistent poverty was significantly associated with higher CRP levels, this difference was accounted for by BMI. Young adults in persistent poverty were significantly more likely to be obese (mean BMI =30.10), relative to those who never lived in poverty. Controlling for BMI however did not fully account for the effect of childhood poverty on CRP. While weighted OLS regression does not permit a formal test of interactions, it provides an indication of potential mediation through BMI. There are a variety of reasons why the persistently poor may have higher BMI. Food choices and decisions related to physical activity may depend on the social environment in which one lives such that healthy food choices including fruits and vegetables

may be unavailable or more expensive (Lee, Harris & Gordon-Larsen, 2009). In addition, poor neighborhoods may lack access to recreational facilities or have higher rates of crime which may limit physical activity (Hannon, 2005). While persistently poor young adults may have these barriers to contend with, young adults who grew up in poverty but no longer live in poverty may have greater access to healthy foods and well as safe environments to engage in physical activity. It is possible childhood poverty exerts influence on CRP through a variable that is unaccounted for in the present model.

These general findings are in line with prior studies showing socioeconomic variations in CRP such that CRP levels are higher among those with lower income (Alley et al. (2005; Jousilahti et al, 2003; Onat et al (2001). It is also consistent with prior studies that have linked childhood poverty to higher CRP (Danese et al., 2009; Pollitt et al, 2007; Schreier and Chen, 2010). Although these studies link low socioeconomic status in childhood to CRP, it is uncertain whether these effects are attributable to childhood exposure to low income or poverty. In other words, childhood exposure to low socioeconomic status may be acting as a proxies for other forms of adversity or perhaps cumulative disadvantage. That said, the present study included childhood experiences of abuse and adult experiences of intimate partner violence, so that the effect of childhood poverty on CRP is net of these traumatic experiences.

Of the four categories of childhood and/or adult poverty tested, the two involving childhood poverty were significantly associated with CRP. This finding lends support to the biological embedding theory in that childhood may be a sensitive period for exposure to poverty. Additionally, childhood poverty remained significantly associated with CRP in the fully adjusted model suggesting the inflammatory risk of childhood poverty persists despite upward mobility by young adults. This is consistent with prior studies that have found risk of myocardial

infarctions (Ljung & Hallquist, 2006) and stroke (Hart, Hole & Smith, 2000) remain for adults who grew up in low socioeconomic families despite upward social mobility.

Using the more liberal definition of poverty at 150% above the federal poverty line generated a different pattern of results. Though both childhood poverty and persistent poverty were significantly associated with CRP, parents' education accounted for the effect of childhood poverty while young adult education accounted for persistent poverty. For young adults who may not yet be financially established higher education may carry benefits similar to income at older ages. For example, attending college may mean having a place to live and access to health services that may offset the low income or lack of the young person's own resources.

The current study makes important contributions to the study of socioeconomic status and disease risk in two major ways. First, by using a more nuanced measure of poverty that incorporates both the timing of poverty as well as the chronicity of poverty. The timing of exposure to low-income has received some attention in the literature however, the length of exposure as well as the pattern of exposure over the life-course is relatively rare.

Second the present study includes other forms of disadvantage such as child maltreatment and intimate partner violence that tend to co-occur with poverty. The data in this study did not support interactions between poverty status and emotional abuse or intimate partner violence.

8.6 Limitations and Strengths

8.6.1 Limitations

There are several limitations worth noting. First this study relies on retrospective reports of maltreatment in childhood and intimate partner violence in adulthood. A variety of factors can affect the accuracy of recollection of childhood events in particular, including degradation of memory repression or even false positive reporting (Bernstein, 1997). Some have noted however

that memories of childhood events may be enhanced when events are unusual, unexpected or traumatic (Brewin, Andrews & Gotlib, 1993). Although recall bias is an important consideration for the maltreatment questions, the bias could be small for several reasons. First respondents were young adults when asked about childhood maltreatment. This has the advantage that 1) not too much time had elapsed and 2) young adults were mature enough to understand and report such events (Perkonig, Kessler, Sorz, & Wittchen, 2000). Finally, respondents answered sensitive questions using laptop computers rather than directly to interviewers which may reduce bias in the data, by limiting social desirability bias.

Second, C-reactive protein is one of several inflammatory markers and it is only measured at one point in time. CRP however, has been shown to be a clinically useful risk marker of early cardiovascular disease risk. Moreover, compared to other inflammatory markers, C-reactive protein has a longer half-life and is relatively stable over time.

8.6.2 Strengths

Despite the aforementioned limitations, a great strength of this study is the use of longitudinal, nationally representative data. Use of a longitudinal dataset meant the ability to examine the temporal order of exposure two independent variables: intimate partner violence and childhood and/or adult poverty. Additionally, it allowed for an assessment of the cumulative health impact of persistent economic status. Finally, findings are generalizable to the U.S. population of young adults. The present study is also strengthened by the use of not only theoretical models relating trauma to physiologic change but also the use of two theoretical models that articulate social placement and the differential ways it influences exposure and experiences of trauma.

The present study makes important contributions to the literature on physiologic consequences of trauma. This is one of the first studies to examine the independent effect of emotional abuse on CRP. Given the relatively young age of the analytic sample, positive findings point to risk of cardiovascular disease at a young age. Additionally, it provides evidence of the enduring effects of childhood socioeconomic characteristics on inflammation and makes important contributions to the literature by finding support for the additional health burden of persistent economic disadvantage.

8.7 Public Health Implications

In recent years, the effect of early life adversity on later health has received a lot of attention in academic settings as well as in popular media outlets. The idea the repeated exposure to stressors in childhood often manifests physiologically has led to the inclusion of early life stress questionnaires (e.g. ACES questionnaire) in hospital screenings (Corbin et al., 2013). Findings from this study support a similar focus on intimate partner violence. One review of 237 articles addressing clinic-based interventions with victims of intimate partner violence showed most women stated they want health care providers to ask about victimization (MacFarlane et al, 2006).

Given the high prevalence of emotional abuse, adult intimate partner violence, the ever widening gap between the rich and poor, its effect on inflammation has important implications for public health practitioners and clinician alike (Brieding, 2014). Intimate partner violence is preventable and a potentially treatable risk factor for cardiovascular disease. Primary prevention strategies include health education interventions that incorporate violence within the home as a public health issue with consequences for physical health. Secondary prevention strategies include screening for exposure to intimate partner violence in health care settings. A first step is

to test interventions such as that described by the March of Dimes Birth Defects Foundation (Parker et al., 1999). This protocol is not specific to abuse during pregnancy and can be used in any health care setting to screen for exposure to intimate partner violence and offer effective treatment and/or referral services. Healthcare workers often come into contact with victims of intimate partner violence and are in a good position to identify victims of intimate partner violence and refer them to needed services. Finally, any intervention should recognize and address social placement as a fundamental cause that shapes that confers physiologic risk, especially when experienced at an early age.

From a research perspective it would be valuable for researchers to include measures of inflammation into population surveys and intervention studies. Such information would allow further investigation into the correlates of inflammation as well as provide insight as to if and how it is possible to reverse or minimize systemic inflammation. Effective pharmacological therapies have been identified, the cautious adoption of which may lead to reductions in inflammation levels among victims of intimate partner violence and ultimately decrease risk of cardiovascular disease (Ridker et al., 2005).

8.8 Future Directions

Violence within families and intimate relationship is increasingly recognized as a public health issue with important health implications (Butchart, et al., 2006; Mercy et al., 2015). Research relating violence to inflammation provides valuable insight into physical health consequences and serves to identify populations at risk of developing associated chronic diseases including cardiovascular disease. Future research is needed to further explore changes in inflammation over the life course. First, very little is known about how CRP changes over time starting with a relatively young population. With Wave V of Add Health data collection under

way, future studies can begin to elucidate patterns in how CRP changes according to young adult history of trauma. Second, the present study did not specifically examine the role of psychosocial and behavioral factors that may differentially affect the relationship between trauma and inflammation. For example, alcohol and substance use as well as PTSD all influence inflammatory processes and may be ways through which traumatic experiences may lead to changes in inflammation. Future research is needed to better "unpack" pathways that carry the deleterious effects of trauma forward.

While traumatic events tend to disrupt and negatively affect physical health, it is also true that not all victims of maltreatment go on to develop physical health problems. That is, two children with the same history of maltreatment can have very different health outcomes. Data shows even among children with histories of severe maltreatment, approximately 20% went on to develop cardiovascular disease (Dong et al., 2004). This suggests a critical need for future research to incorporate protective factors that may buffer the negative health effects of traumatic experiences.

LITERATURE CITED

Adler, N. E., Boyce, T., Chesney, M. A., Cohen, S., Folkman, S., Kahn, R. L., & Syme, S. L. (1994). Socioeconomic status and health: the challenge of the gradient. *American psychologist*, *49*(1), 15.

American Psychiatric Association (2001) Diagnostic and Statistical Manual of Mental Disorders (4th edn, revised) (DSM-IV-TR). Washington, DC: APA.

Anda, R. F., Dong, M., Brown, D. W., Felitti, V. J., Giles, W. H., Perry, G. S., ... & Dube, S. R. (2009). The relationship of adverse childhood experiences to a history of premature death of family members. *BMC Public Health*, *9*(1), 106.

Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C. H., Perry, B. D., ... & Giles, W. H. (2006). The enduring effects of abuse and related adverse experiences in childhood. *European archives of psychiatry and clinical neuroscience*, *256*(3), 174-186.

Anda, R. F., Croft, J. B., Felitti, V. J., Nordenberg, D., Giles, W. H., Williamson, D. F., & Giovino, G. A. (1999). Adverse childhood experiences and smoking during adolescence and adulthood. *Jama*, *282*(17), 1652-1658.

Aneshensel, C. S., & Mitchell, U. A. (2014). The stress process: its origins, evolution, and future. In Sociology of Mental Health (pp. 53-74). Springer International Publishing.

Aneshensel, C. S. (2012). *Theory-based data analysis for the social sciences*. Sage.

Archer, J. (2000). Sex differences in aggression between heterosexual partners: a meta-analytic review. *Psychological bulletin*, *126*(5), 651.

Batten, S. V., Aslan, M., Maciejewski, P. K., & Mazure, C. M. (2004). Childhood maltreatment as a risk factor for adult cardiovascular disease and depression. *Journal of Clinical Psychiatry*, *65*(2), 249-254.

Bernstein, D. P., Ahluvalia, T., Pogge, D., & Handelsman, L. (1997). Validity of the Childhood Trauma Questionnaire in an adolescent psychiatric population. *Journal of the American Academy of Child & Adolescent Psychiatry*, *36*(3), 340-348.

Bernstein, D. P., & Fink, L. (1998). CTQ Childhood Trauma Questionnaire. *A retrospecitive self-report. Manual. San Antonio: The Psychological Corporation*.

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvalia, T., ... & Zule, W. (2003). Development and validation of a brief screening version of the Childhood Trauma Questionnaire. *Child abuse & neglect*, *27*(2), 169-190.

Bertone-Johnson, E. R., Whitcomb, B. W., Missmer, S. A., Karlson, E. W., & Rich-Edwards, J. W. (2012). Inflammation and early-life abuse in women.*American journal of preventive medicine*, *43*(6), 611-620.

Black, M. C. (2011). Intimate partner violence and adverse health consequences: implications for clinicians. American Journal of Lifestyle Medicine, 1559827611410265.

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

Blake, G. J., & Ridker, P. M. (2001). Novel clinical markers of vascular wall inflammation. Circulation research, 89(9), 763-771.

Blake, G. J., Rifai, N., Buring, J. E., & Ridker, P. M. (2003). Blood pressure, C-reactive protein, and risk of future cardiovascular events. *Circulation*,*108*(24), 2993-2999.

Bonomi, A. E., Thompson, R. S., Anderson, M., Reid, R. J., Carrell, D., Dimer, J. A., & Rivara, F. P. (2006). Intimate partner violence and women's physical, mental, and social functioning. *American journal of preventive medicine*, *30*(6), 458-466.

Breiding, M. J., Smith, S. G., Basile, K. C., Walters, M. L., Chen, J., & Merrick, M. T. (2015). Prevalence and Characteristics of Sexual Violence, Stalking, and Intimate Partner Violence Victimization—National Intimate Partner and Sexual Violence Survey, United States, 2011. *American Journal of Public Health*, *105*(4), E11.

Breiding, M. J., Black, M. C., & Ryan, G. W. (2008). Chronic disease and health risk behaviors associated with intimate partner violence—18 US states/territories, 2005. Annals of epidemiology, 18(7), 538-544.

Bremner, J. D., Vermetten, E., & Mazure, C. M. (2000). Development and preliminary psychometric properties of an instrument for the measurement of childhood trauma: the Early Trauma Inventory. *Depression and anxiety*,*12*(1), 1-12.

Brewin, C. R., Andrews, B., & Gotlib, I. H. (1993). Psychopathology and early experience: a reappraisal of retrospective reports. Psychological bulletin, 113(1), 82.

Brown, J., Cohen, P., Johnson, J. G., & Smailes, E. M. (1999). Childhood abuse and neglect: specificity of effects on adolescent and young adult depression and suicidality. *Journal of the American Academy of Child & Adolescent Psychiatry*, *38*(12), 1490-1496.

Brown, G. W., Craig, T. K., Harris, T. O., Handley, R. V., & Harvey, A. L. (2007). Validity of retrospective measures of early maltreatment and depressive episodes using the Childhood Experience of Care and Abuse (CECA) instrument—A life-course study of adult chronic depression—2.*Journal of affective disorders*, *103*(1), 217-224.

Brown, D. W., Anda, R. F., Tiemeier, H., Felitti, V. J., Edwards, V. J., Croft, J. B., & Giles, W. H. (2009). Adverse childhood experiences and the risk of premature mortality. *American journal of preventive medicine*, *37*(5), 389-396.

Boscarino, J. A. (2004). Posttraumatic stress disorder and physical illness: results from clinical and epidemiologic studies. Annals of the New York Academy of Sciences, 1032(1), 141-153.

Brownstein, N., Kalsbeek, W. D., Tabor, J., Entzel, P., Daza, E., & Harris, K. M. (2010). Non-Response in Wave IV of the National Longitudinal Study of Adolescent Health. *Chapel Hill, NC: Carolina Population Center*.

Brush, L. D. (1990). Violent acts and injurious outcomes in married couples: Methodological issues in the National Survey of Families and Households.*Gender and Society*, 56-67.

Butchart, A., Harvey, A. P., Mian, M., & Furniss, T. (2006). Preventing child maltreatment: a guide to taking action and generating evidence.

Caetano, R., & Cunradi, C. (2003). Intimate partner violence and depression among Whites, Blacks, and Hispanics. *Annals of epidemiology*, *13*(10), 661-665.

Campbell, J. C. (2002). Health consequences of intimate partner violence. *The Lancet*, *359*(9314), 1331-1336.

Carpenter, L. L., Carvalho, J. P., Tyrka, A. R., Wier, L. M., Mello, A. F., Mello, M. F., ... & Price, L. H. (2007). Decreased adrenocorticotropic hormone and cortisol responses to stress in healthy adults reporting significant childhood maltreatment. *Biological psychiatry*, *62*(10), 1080-1087.

Carpenter, L. L., Gawuga, C. E., Tyrka, A. R., & Price, L. H. (2012). C-reactive protein, early life stress, and wellbeing in healthy adults. *Acta Psychiatrica Scandinavica*, *126*(6), 402-410.

Centers for Disease Control and Prevention (CDC).. (2008). Adverse health conditions and health risk behaviors associated with intimate partner violence—United States, 2005. Morbidity and Mortality Weekly Report, 57, 113–117

Cesari, M., Penninx, B. W., Newman, A. B., Kritchevsky, S. B., Nicklas, B. J., Sutton-Tyrrell, K., ... & Pahor, M. (2003). Inflammatory markers and onset of cardiovascular events results from the Health ABC Study. Circulation,108(19), 2317-2322.

Chantala, K. (2006). Guidelines for analyzing Add Health data. *National Longitudinal Study of Adolescent Health: University of North Carolina at Chapel Hill*.

Chen, E., & Miller, G. E. (2012). "Shift-and-Persist" Strategies Why Low Socioeconomic Status Isn't Always Bad for Health. *Perspectives on Psychological Science*, *7*(2), 135-158.

Chen, E., Matthews, K. A., & Boyce, W. T. (2002). Socioeconomic differences in children's health: how and why do these relationships change with age?. *Psychological bulletin*, *128*(2), 295.

Cicchetti, D. and F. A. Rogosch (2001). "The impact of child maltreatment and psychopathology on neuroendocrine functioning." Development and Psychopathology **13**(04): 783-804.

Clark, C. J., Alonso, A., Everson-Rose, S. A., Spencer, R. A., Brady, S. S., Resnick, M. D., ... & Feng, S. L. (2016). Intimate partner violence in late adolescence and young adulthood and subsequent cardiovascular risk in adulthood. *Preventive medicine*, *87*, 132-137.

Cohen, S., Kessler, R. C., & Gordon, L. U. (1995). Strategies for measuring stress in studies of psychiatric and physical disorders. Measuring stress: A guide for health and social scientists, 3-26.

Coker, A. L., Smith, P. H., Bethea, L., King, M. R., & McKeown, R. E. (2000). Physical health consequences of physical and psychological intimate partner violence. *Archives of family medicine*, *9*(5), 451.

Corbin, T. J., Purtle, J., Rich, L. J., Rich, J. A., Adams, E. J., Yee, G., & Bloom, S. L. (2013). The prevalence of trauma and childhood adversity in an urban, hospital-based violence intervention program. *Journal of health care for the poor and underserved*, *24*(3), 1021-1030.

Cunradi, C. B., Caetano, R., Clark, C., & Schafer, J. (2000). Neighborhood poverty as a predictor of intimate partner violence among White, Black, and Hispanic couples in the United States: a multilevel analysis. *Annals of epidemiology*, *10*(5), 297-308.

DeNavas-Walt, Carmen and Bernadette D. Proctor, U.S. Census Bureau, Current Population Reports, P60-252, Income and Poverty in the United States: 2014, U.S. Government Printing Office, Washington, DC, 2015.

Danese, A., & McEwen, B. S. (2012). Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & behavior*,*106*(1), 29-39.

Danese, A., Caspi, A., Williams, B., Ambler, A., Sugden, K., Mika, J., ... & Arseneault, L. (2010). Biological embedding of stress through inflammation processes in childhood. *Brain, Behavior, and Immunity*, *24*, S8.

Danese, A., Moffitt, T. E., Pariante, C. M., Ambler, A., Poulton, R., & Caspi, A. (2008). Elevated inflammation levels in depressed adults with a history of childhood maltreatment. *Archives of general psychiatry*, *65*(4), 409-415.

Danese, A., Pariante, C. M., Caspi, A., Taylor, A., & Poulton, R. (2007). Childhood maltreatment predicts adult inflammation in a life-course study.*Proceedings of the National Academy of Sciences*, *104*(4), 1319-1324.

Dong, M., Giles, W. H., Felitti, V. J., Dube, S. R., Williams, J. E., Chapman, D. P., & Anda, R. F. (2004). Insights into causal pathways for ischemic heart disease adverse childhood experiences study. *Circulation*, *110*(13), 1761-1766.

Dozier, M., Manni, M., Gordon, M. K., Peloso, E., Gunnar, M. R., Stovall-McClough, K. C., ... & Levine, S. (2006). Foster children's diurnal production of cortisol: An exploratory study. *Child Maltreatment*, *11*(2), 189-197.

Dube, S. R., Fairweather, D., Pearson, W. S., Felitti, V. J., Anda, R. F., & Croft, J. B. (2009). Cumulative childhood stress and autoimmune diseases in adults. *Psychosomatic medicine*, *71*(2), 243-250.

Dunn, E. McLaughlin, K. Slopen, N. Rosand, J. Smoller, J. (2013). Developmental Timing of Child Maltreatment and Symptoms of Depression and Suicidal Ideation in Young Adulthood: Results from the National Longitudinal Study of Adolescent Health. *Depression and Anxiety, 00: 1-10*

Fallon, Trocmet t al. (2010). "Methodological challenges in measuring child maltreatment." Child Abuse & Neglect **34**(1): 70-79.

Feinstein, J. S. (1993). The relationship between socioeconomic status and health: a review of the literature. The Milbank Quarterly, 279-322.

Fernandez-Botran, R., Miller, J. J., Burns, V. E., & Newton, T. L. (2011). Correlations among inflammatory markers in plasma, saliva and oral mucosal transudate in post-menopausal women with past intimate partner violence.*Brain, behavior, and immunity*, *25*(2), 314-321.

Felitti, M. D., Vincent, J., Anda, M. D., Robert, F., Nordenberg, M. D., Williamson, M. S., ... & James, S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults: The Adverse Childhood Experiences (ACE) Study. *American journal of preventive medicine*, *14*(4), 245-258.

Fergusson, D. M., Horwood, L. J., & Lynskey, M. T. (1996). Childhood sexual abuse and psychiatric disorder in young adulthood: II. Psychiatric outcomes of childhood sexual abuse. *Journal of the American Academy of Child & Adolescent Psychiatry*, *35*(10), 1365-1374.

Fergusson, D. M., J. M. Boden, et al. (2008). "Exposure to childhood sexual and physical abuse and adjustment in early adulthood." Child Abuse & Neglect **32**(6): 607-619.

Finkelhor, D., Turner, H. A., Shattuck, A., & Hamby, S. L. (2013). Violence, crime, and abuse exposure in a national sample of children and youth: An update. *JAMA pediatrics*, *167*(7), 614-621.

Finkelhor, D., Turner, H., Ormrod, R., & Hamby, S. L. (2009). Violence, abuse, and crime exposure in a national sample of children and youth. Pediatrics, 124(5), 1411-1423.

Finkelhor, D., & Dziuba-Leatherman, J. (1994). Children as victims of violence: A national survey. Pediatrics, 94(4), 413-420.

Fletcher, J. M. (2009). Childhood mistreatment and adolescent and young adult depression. *Social Science & Medicine*, *68*(5), 799-806.

Freisthler, B., Merritt, D. H., & LaScala, E. A. (2006). Understanding the ecology of child maltreatment: A review of the literature and directions for future research. *Child Maltreatment*, *11*(3), 263-280.

Ford, D. E., & Erlinger, T. P. (2004). Depression and C-reactive protein in US adults: data from the Third National Health and Nutrition Examination Survey.*Archives of internal medicine*, *164*(9), 1010-1014.

Galobardes, B., Lynch, J. W., & Smith, G. D. (2004). Childhood socioeconomic circumstances and cause-specific mortality in adulthood: systematic review and interpretation. *Epidemiologic reviews*, *26*(1), 7-21.

Galobardes, B., Smith, G. D., & Lynch, J. W. (2006). Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. Annals of epidemiology, 16(2), 91-104.

Gander, M. L., & von Känel, R. (2006). Myocardial infarction and post-traumatic stress disorder: frequency, outcome, and atherosclerotic mechanisms. European Journal of Cardiovascular Prevention & Rehabilitation, 13(2), 165-172.

Gelles, R. J. (1992). Poverty and violence toward children. *American behavioral scientist*.

Gershon, A., Minor, K., & Hayward, C. (2008). Gender, victimization, and psychiatric outcomes. *Psychological medicine*, *38*(10), 1377-1391.

Gilbert, R., Widom, C. S., Browne, K., Fergusson, D., Webb, E., & Janson, S. (2009). Burden and consequences of child maltreatment in high-income countries. *The lancet*, *373*(9657), 68-81.

Gilles, E. E. (1999). Integrating a neurobiological systems approach into child neglect and abuse theory and practice. *Children's Health Care*, *28*(2), 167-187.

Golding, J. M. (1999). Intimate partner violence as a risk factor for mental disorders: A meta-analysis. *Journal of family violence*, *14*(2), 99-132.

Goodman, E. (1999). The role of socioeconomic status gradients in explaining differences in US adolescents' health. *American Journal of Public Health*, *89*(10), 1522-1528.

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

Goodwin, R. D., & Weisberg, S. P. (2002). Childhood abuse and diabetes in the community. *Diabetes care*, *25*(4), 801-802.

Gouin, J. P., Glaser, R., Malarkey, W. B., Beversdorf, D., & Kiecolt-Glaser, J. (2012). Chronic stress, daily stressors, and circulating inflammatory markers.*Health Psychology*, *31*(2), 264.

Gruenewald, T. L., Cohen, S., Matthews, K. A., Tracy, R., & Seeman, T. E. (2009). Association of socioeconomic status with inflammation markers in black and white men and women in the Coronary Artery Risk Development in Young Adults (CARDIA) study. Social science & medicine, 69(3), 451-459.

Gunnar, M. and K. Quevedo (2007). "The neurobiology of stress and development." Annu. Rev. Psychol. **58**: 145-173.

Gunnar, M. R., & Donzella, B. (2002). Social regulation of the cortisol levels in early human development. *Psychoneuroendocrinology*, *27*(1), 199-220.

Gunnar, M. R., Morison, S. J., Chisholm, K. I. M., & Schuder, M. (2001). Salivary cortisol levels in children adopted from Romanian orphanages.*Development and psychopathology*, *13*(03), 611-628.

Hagenaars, A. (1991). "The Definition and Measurement of Poverty", in L. Osberg (Ed.), Economic Inequality and Poverty: International Perspectives, Armonk, NY: M. E. Sharpe

Hak, A. E., Pols, H. A., Stehouwer, C. D., Meijer, J., Kiliaan, A. J., Hofman, A., ... & Witteman, J. C. (2001). Markers of inflammation and cellular adhesion molecules in relation to insulin resistance in nondiabetic elderly: the Rotterdam study. *Journal of Clinical Endocrinology & Metabolism*, *86*(9), 4398-4405.

Halfon, N., Berkowitz, G., & Klee, L. (1992). Mental health service utilization by children in foster care in California. *Pediatrics*, *89*(6), 1238-1244.

Hardt, J., & Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: review of the evidence. *Journal of Child Psychology and Psychiatry*, *45*(2), 260-273.

Hart, C. L., Hole, D. J., & Smith, G. D. (2000). Influence of socioeconomic circumstances in early and later life on stroke risk among men in a Scottish cohort study. Stroke, 31(9), 2093-2097.

Harris, K. M. (2013). The Add Health study: design and accomplishments.Carolina Population Center, University of North Carolina at Chapel Hill.

Harris, K. M. (2009). The National Longitudinal Study of Adolescent Health (Add Health), Waves I & II, 1994–1996; Wave III, 2001–2002; Wave IV, 2007–2009

Hayward, M. D., & Gorman, B. K. (2004). The long arm of childhood: The influence of early-life social conditions on men's mortality. *Demography*,*41*(1), 87-107.

Hayward, M. D., Miles, T. P., Crimmins, E. M., & Yang, Y. (2000). The significance of socioeconomic status in explaining the racial gap in chronic health conditions. *American sociological review*, 910-930.

Heeringa, S. G., West, B. T., & Berglund, P. A. (2010). *Applied survey data analysis*. CRC Press.

Heim, C., Ehlert, U., & Hellhammer, D. H. (2000). The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders.*Psychoneuroendocrinology*, *25*(1), 1-35.

Heim, C. and C. B. Nemeroff (2001). "The role of childhood trauma in the neurobiology of mood and anxiety disorders: preclinical and clinical studies." Biological psychiatry **49**(12): 1023-1039.

Heise, L., Ellsberg, M., & Gottmoeller, M. (2002). A global overview of gender-based violence. International Journal of Gynecology & Obstetrics, 78, S5-S14.

Hepgul, N., Pariante, C. M., Dipasquale, S., Diforti, M., Taylor, H., Marques, T. R., ... & Mondelli, V. (2012). Childhood maltreatment is associated with increased body mass index and increased C-reactive protein levels in first-episode psychosis patients. *Psychological medicine*, *42*(09), 1893-1901.

Hertzman, C. (1999). The biological embedding of early experience and its effects on health in adulthood. *Annals of the New York Academy of Sciences*,*896*(1), 85-95.

Higgins, D. J. and M. P. McCabe (2003). "Maltreatment and family dysfunction in childhood and the subsequent adjustment of children and adults." Journal of family violence **18**(2): 107-120.

Holmes, G. R., Offen, L., & Waller, G. (1997). See no evil, hear no evil, speak no evil: Why do relatively few male victims of childhood sexual abuse receive help for abuse related issues in adulthood?. *Clinical Psychology Review*,*17*(1), 69-88.

Inslicht, S. S., Marmar, C. R., Neylan, T. C., Metzler, T. J., Hart, S. L., Otte, C., ... & Baum, (2006). Increased cortisol in women with intimate partner violence-related posttraumatic stress disorder. *Psychoneuroendocrinology*,*31*(7), 825-838.

Kaplan, J. R., Adams, M. R., Clarkson, T. B., Manuck, S. B., Shively, C. A., & Williams, J. K. (1996). Psychosocial factors, sex differences, and atherosclerosis: lessons from animal models. *Psychosomatic Medicine*, *58*(6), 598-611.

Kaplan, S. J., Pelcovitz, D., & Labruna, V. (1999). Child and adolescent abuse and neglect research: A review of the past 10 years. Part I: Physical and emotional abuse and neglect. *Journal of the American Academy of Child & Adolescent Psychiatry*, *38*(10), 1214-1222.

Kaplow, J. B., & Widom, C. S. (2007). Age of onset of child maltreatment predicts long-term mental health outcomes. *Journal of abnormal Psychology*,*116*(1), 176.

Karatoreos, I. N., & McEwen, B. S. (2013). Annual research review: the neurobiology and physiology of resilience and adaptation across the life course. *Journal of Child Psychology and Psychiatry*, *54*(4), 337-347.

Kao, P. C., Shiesh, S. C., & Wu, T. J. (2006). Serum C-reactive protein as a marker for wellness assessment. Annals of Clinical & Laboratory Science,36(2), 163-169.

Kaufman, J., Plotsky, P. M., Nemeroff, C. B., & Charney, D. S. (2000). Effects of early adverse experiences on brain structure and function: clinical implications. *Biological psychiatry*, *48*(8), 778-790.

Ketring, S. A., & Feinauer, L. L. (1999). Perpetrator-victim relationship: Long-term effects of sexual abuse for men and women. *American Journal of Family Therapy*, *27*(2), 109-120.

Kiecolt-Glaser, J. K., Preacher, K. J., MacCallum, R. C., Atkinson, C., Malarkey, W. B., & Glaser, R. (2003). Chronic stress and age-related increases in the proinflammatory cytokine IL-6. *Proceedings of the National Academy of Sciences*, *100*(15), 9090-9095.

King, K. A., Vidourek, R. A., Davis, B., & McClellan, W. (2002). Increasing self-esteem and school connectedness through a multidimensional mentoring program. *Journal of School Health*, *72*(7), 294-299.

Koenen, K. C., Roberts, A. L., Stone, D. M., & Dunn, E. C. (2010). The epidemiology of early childhood trauma. *The impact of early life trauma on health and disease: The hidden epidemic*, *1*.

Koenig, W. (2001). C-Reactive protein and cardiovascular risk: has the time come for screening the general population?. *Clinical chemistry*, *47*(1), 9-10.

Koziol-McLain, J., Coates, C. J., & Lowenstein, S. R. (2001). Predictive validity of a screen for partner violence against women. *American journal of preventive medicine*, *21*(2), 93-100.

Kramer, A., Lorenzon, D., & Mueller, G. (2004). Prevalence of intimate partner violence and health implications for women using emergency departments and primary care clinics. *Women's Health Issues*, *14*(1), 19-29.

Kuo, H. K., Yen, C. J., Chang, C. H., Kuo, C. K., Chen, J. H., & Sorond, F. (2005). Relation of C-reactive protein to stroke, cognitive disorders, and depression in the general population: systematic review and meta-analysis.*The Lancet Neurology*, *4*(6), 371-380.

Lanier, P., Jonson-Reid, M., Stahlschmidt, M. J., Drake, B., & Constantino, J. (2010). Child maltreatment and pediatric health outcomes: A longitudinal study of low-income children. *Journal of pediatric psychology*, *35*(5), 511-522.

Lansford, J. E., K. A. Dodge, et al. (2002). "A 12-year prospective study of the long-term effects of early child physical maltreatment on psychological, behavioral, and academic problems in adolescence." Archives of Pediatrics and Adolescent Medicine **156**(8): 824.

Larkin, W., & Read, J. (2008). Childhood trauma and psychosis: evidence, pathways, and implications. *Journal of postgraduate medicine*, *54*(4).

Ledue, T. B., & Rifai, N. (2003). Preanalytic and analytic sources of variations in C-reactive protein measurement: implications for cardiovascular disease risk assessment. *Clinical Chemistry*, *49*(8), 1258-1271.

Leeb, R. T. (2008). *Child maltreatment surveillance: Uniform definitions for public health and recommended data elements*. Centers for Disease Control and Prevention, National Center for Injury Prevention and Control.

Liberatos, P., Link, B. G., & Kelsey, J. L. (1988). The measurement of social class in epidemiology. *Epidemiologic reviews*, *10*(1), 87-121.

Link, B. G., & Phelan, J. (1995). Social conditions as fundamental causes of disease. Journal of health and social behavior, 80-94.

Link, B. G., & Phelan, J. C. (1996). Understanding sociodemographic differences in health-the role of fundamental social causes. *American journal of public health*, *86*(4), 471 473.

Liu, R. T., Alloy, L. B., Abramson, L. Y., Iacoviello, B. M., & Whitehouse, W. G. (2009). Emotional maltreatment and depression: Prospective prediction of depressive episodes. *Depression and Anxiety*, *26*(2), 174-181.

Ljung, R., & Hallqvist, J. (2006). Accumulation of adverse socioeconomic position over the entire life course and the risk of myocardial infarction among men and women: results from the Stockholm Heart Epidemiology Program (SHEEP). Journal of epidemiology and community health, 60(12), 1080-1084.

Lu, S., Peng, H., Wang, L., Vasish, S., Zhang, Y., Gao, W., ... & Li, W. (2013). Elevated specific peripheral cytokines found in major depressive disorder patients with childhood trauma exposure: a cytokine antibody array analysis. *Comprehensive psychiatry*, *54*(7), 953-961.

Luecken, L. J. (1998). Childhood attachment and loss experiences affect adult cardiovascular and cortisol function. *Psychosomatic Medicine*, *60*(6), 765-772.

Lutfey, K., & Freese, J. (2005). Toward some fundamentals of fundamental causality: socioeconomic status and health in the routine clinic visit for diabetes1. American Journal of Sociology, 110(5), 1326-1372.

Lutgendorf, S. K., Anderson, B., Sorosky, J. I., Buller, R. E., & Lubaroff, D. M. (2000). Interleukin-6 and use of social support in gynecologic cancer patients. *International Journal of Behavioral Medicine*, *7*(2), 127-142.

Luthar, S. S., D. Cicchetti, et al. (2000). "The construct of resilience: A critical evaluation and guidelines for future work." Child development **71**(3): 543-562.

Luthar, S. S., J. A. Sawyer, et al. (2006). Conceptual issues in studies of resilience - Past, present, and future research. Resilience in Children. B. M. Lester, A. S. Masten and B. McEwen. Oxford, Blackwell Publishing. **1094:** 105-115.

Lynch, J. W., Kaplan, G. A., & Salonen, J. T. (1997). Why do poor people behave poorly? Variation in adult health behaviours and psychosocial characteristics by stages of the socioeconomic lifecourse. *Social science & medicine*, *44*(6), 809-819.

Lynch, M. and D. Cicchetti (1998). "An ecological-transactional analysis of children and contexts: The longitudinal interplay among child maltreatment, community violence, and children's symptomatology." Development and Psychopathology **10**(2): 235-257.

Lynch, J., & Smith, G. D. (2005). A life course approach to chronic disease epidemiology. *Annu. Rev. Public Health*, *26*, 1-35.

Manly, J. T., Cicchetti, D., & Barnett, D. (1994). The impact of subtype, frequency, chronicity, and severity of child maltreatment on social competence and behavior problems. *Development and psychopathology*,*6*(01), 121-143.

Manuck, S. B., Marsland, A. L., Kaplan, J. R., & Williams, J. K. (1995). The pathogenicity of behavior and its neuroendocrine mediation: an example from coronary artery disease. *Psychosomatic Medicine*, *57*(3), 275-283.

Matthews, K. A., Chang, Y. F., Thurston, R. C., & Bromberger, J. T. (2014). Child abuse is related to inflammation in mid-life women: role of obesity.*Brain, behavior, and immunity*, *36*, 29-34.

McBride, C. M., Curry, S. J., Cheadle, A., Anderman, C., Wagner, E. H., Diehr, P., & Psaty, B. (1995). School-Level Application of a Social Bonding Model to Adolescent Risk-Taking Behavior. *Journal of School Health*, *65*(2), 63-68.

McCrory, E., De Brito, S. A., & Viding, E. (2010). Research review: the neurobiology and genetics of maltreatment and adversity. *Journal of Child Psychology and Psychiatry*, *51*(10), 1079-1095.

McDade, T. W., Hawkley, L. C., & Cacioppo, J. T. (2006). Psychosocial and behavioral predictors of inflammation in middle-aged and older adults: the Chicago health, aging, and social relations study. *Psychosomatic Medicine*,*68*(3), 376-381.

McFarlane, J. Groff, J. O'Brien, J. Wilson, K (2006). Secondary Prevention of Intimate Partner Violence. *Nursing Research*, Vol 55, No. 1

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

McLaughlin, K. A., Conron, K. J., Koenen, K. C., & Gilman, S. E. (2010). Childhood adversity, adult stressful life events, and risk of past-year psychiatric disorder: a test of the stress sensitization hypothesis in a population-based sample of adults. *Psychological medicine*, *40*(10), 1647-1658.

Miller, G. E., Stetler, C. A., Carney, R. M., Freedland, K. E., & Banks, W. A. (2002). Clinical depression and inflammatory risk markers for coronary heart disease. *The American journal of cardiology*, *90*(12), 1279-1283.

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: moving toward a model of behavioral and biological mechanisms. *Psychological bulletin*,*137*(6), 959.

Miller, G. E., Chen, E., & Zhou, E. S. (2007). If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. *Psychological bulletin*, *133*(1), 25.

Miller, G., Chen, E., & Cole, S. W. (2009). Health psychology: Developing biologically plausible models linking the social world and physical health. *Annual review of psychology*, *60*, 501-524.

Mullen, P. E., Martin, J. L., Anderson, J. C., Romans, S. E., & Herbison, G. P. (1993). Childhood sexual abuse and mental health in adult life. *The British Journal of Psychiatry*, *163*(6), 721-732.

Muller, R. T., Gragtmans, K., & Baker, R. (2008). Childhood physical abuse, attachment, and adult social support: Test of a mediational model. *Canadian Journal of Behavioural Science/Revue canadienne des sciences du comportement*, *40*(2), 80.

Mulvihill, D. (2005). The health impact of childhood trauma: An interdisciplinary review, 1997-2003. *Issues in comprehensive pediatric nursing*, *28*(2), 115-136.

Nazmi, A., & Victora, C. G. (2007). Socioeconomic and racial/ethnic differentials of C-reactive protein levels: a systematic review of population-based studies. BMC public health, 7(1), 212.
Neff, J. A., Holamon, B., & Schluter, T. D. (1995). Spousal violence among Anglos, Blacks, and Mexican Americans: The role of demographic variables, psychosocial predictors, and alcohol consumption. *Journal of Family Violence*, *10*(1), 1-21.
Newman, M. G., Clayton, L., Zuellig, A., Cashman, L., Arnow, B., Dea, R., & Taylor, C. B. (2000). The relationship of childhood sexual abuse and depression with somatic symptoms and medical utilization. *Psychological medicine*, *30*(5), 1063-1077.
O'Donovan, A., Epel, E., Lin, J., Wolkowitz, O., Cohen, B., Maguen, S., ... & Neylan, T. C. (2011). Childhood trauma associated with short leukocyte telomere length in posttraumatic stress disorder. *Biological psychiatry*, *70*(5), 465-471.
O'Toole, B. I., & Catts, S. V. (2008). Trauma, PTSD, and physical health: an epidemiological study of Australian Vietnam veterans. Journal of psychosomatic research, 64(1), 33-40.
Out, D., Hall, R. J., Granger, D. A., Page, G. G., & Woods, S. J. (2012). Assessing salivary C-reactive protein: longitudinal associations with systemic inflammation and cardiovascular disease risk in women exposed to intimate partner violence. *Brain, behavior, and immunity*, *26*(4), 543-551.
Pace, T. W., Wingenfeld, K., Schmidt, I., Meinlschmidt, G., Hellhammer, D. H., & Heim, C. M. (2012). Increased peripheral NF-κB pathway activity in women with childhood abuse-related posttraumatic stress disorder. Brain, behavior, and immunity, 26(1), 13-17.
Pace, T. W., Mletzko, T. C., Alagbe, O., Musselman, D. L., Nemeroff, C. B., Miller, A. H., & Heim, C. M. (2006). Increased stress-induced inflammatory responses in male patients with major depression and increased early life stress. *American Journal of Psychiatry*.
Pappas, G., Queen, S., Hadden, W., & Fisher, G. (1993). The increasing disparity in mortality between socioeconomic groups in the United States, 1960 and 1986. New England journal of medicine, 329(2), 103-109.
Paranjape A, Liebschutz J. STaT: A three question screen for intimate partner violence. J Women's Health. 2003;1 2(3):233-240.

Parker, B., McFarlane, J., Soeken, K., Silva, C., & Reel, S. (1999). Testing an intervention to prevent further abuse to pregnant women. Research in nursing & health, 22(1), 59-66.
Pearlin, L. I., & Bierman, A. (2013). Current issues and future directions in research into the stress process. In Handbook of the sociology of mental health (pp. 325-340). Springer Netherlands.
Pearlin, L. I., Menaghan, E. G., Lieberman, M. A., & Mullan, J. T. (1981). The stress process. Journal of Health and Social behavior, 337-356.
Pearson, T. A., Mensah, G. A., Alexander, R. W., Anderson, J. L., Cannon, R. O., Criqui, M., ... & Vinicor, F. (2003). Markers of inflammation and cardiovascular disease application to clinical and public health practice: a statement for healthcare professionals from the centers for disease control and prevention and the American Heart Association. *Circulation*, *107*(3), 499-511.
Phelan, J. C., Link, B. G., Diez-Roux, A., Kawachi, I., & Levin, B. (2004). "Fundamental causes" of social inequalities in mortality: a test of the theory. Journal of health and social behavior, 45(3), 265-285.
Phillips, A. C. (2015). Stress and Cardiovascular Reactivity. Handbook of Psychocardiology.

Pico-Alfonso, M. A., Garcia-Linares, M. I., Celda-Navarro, N., Herbert, J., & Martinez, M. (2004). Changes in cortisol and dehydroepiandrosterone in women victims of physical and psychological intimate partner violence.*Biological psychiatry*, *56*(4), 233-240.

Pollak, S. D. (2008). Mechanisms Linking Early Experience and the Emergence of Emotions Illustrations From the Study of Maltreated Children. *Current Directions in Psychological Science*, *17*(6), 370-375.

Pollitt, R. A., Kaufman, J. S., Rose, K. M., Diez-Roux, A. V., Zeng, D., & Heiss, G. (2008). Cumulative life course and adult socioeconomic status and markers of inflammation in adulthood. *Journal of Epidemiology and Community Health*, *62*(6), 484-491.

Preston, S. H., & Taubman, P. (1994). Socioeconomic differences in adult mortality and health status. *Demography of aging*, *1*, 279-318.

Putnam, F. W. (2003). Ten-year research update review: Child sexual abuse. *Journal of the American Academy of Child & Adolescent Psychiatry*, *42*(3), 269-278.

Raison, C. L., & Miller, A. H. (2003). When not enough is too much: the role of insufficient glucocorticoid signaling in the pathophysiology of stress-related disorders. *American Journal of Psychiatry*, *160*(9), 1554-1565.

Repetti, R. L., S. E. Taylor, et al. (2002). "Risky families: Family social environments and the mental and physical health of offspring." Psychological bulletin **128**(2): 330.

Resnick HS, Kilpatrick DG, Dansky BS, Saunders BE, Best CL (1993). Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. J Consult Clin Psych ;61:984–91. [12]

Resnick, M. D., Bearman, P. S., Blum, R. W., Bauman, K. E., Harris, K. M., Jones, J., ... & Udry, J. R. (1997). Protecting adolescents from harm: findings from the National Longitudinal Study on Adolescent Health. *Jama*, *278*(10), 823-832.

Reynolds, A. J., Ou, S. R., & Topitzes, J. W. (2004). Paths of effects of early childhood intervention on educational attainment and delinquency: A confirmatory analysis of the Chicago Child-Parent Centers. *Child development*, *75*(5), 1299-1328.

Rich-Edwards, J. W., Mason, S., Rexrode, K., Spiegelman, D., Hibert, E., Kawachi, I., & Wright, R. J. (2012). Physical and sexual abuse in childhood as predictors of early onset cardiovascular events in women. *Circulation*, CIRCULATIONAHA-111.

Ridker, P. M., Cannon, C. P., Morrow, D. F., Rifai, N., Rose, L. M., McCabe, C. H., & Braunwald, E. (2005). Pravastatin or Atorvastatin Evaluation and Infection Therapy-Thrombolysis in Myocardial Infarction 22 (PROVE IT-TIMI 22) Investigators. C-reactive protein levels and outcomes after statin therapy.*N Engl j Med*, *352*(1), 20-28.

Ridker, P. M., Buring, J. E., Cook, N. R., & Rifai, N. (2003). C-reactive protein, the metabolic syndrome, and risk of incident cardiovascular events an 8-year follow-up of 14 719 initially healthy American women. *Circulation*,*107*(3), 391-397.

Ridker, P. M., Rifai, N., Rose, L., Buring, J. E., & Cook, N. R. (2002). Comparison of C-reactive protein and low-density lipoprotein cholesterol levels in the prediction of first cardiovascular events. *New England journal of medicine*, *347*(20), 1557-1565.

Ridker, P. M., Buring, J. E., Shih, J., Matias, M., & Hennekens, C. H. (1998). Prospective study of C-reactive protein and the risk of future cardiovascular events among apparently healthy women. Circulation, 98(8), 731-733.

Ridker, P. M., Cushman, M., Stampfer, M. J., Tracy, R. P., & Hennekens, C. H. (1997). Inflammation, aspirin, and the risk of cardiovascular disease in apparently healthy men. New England Journal of Medicine, 336(14), 973-979.

Rietzschel, E., & De Buyzere, M. (2012). High-sensitive C-reactive protein: universal prognostic and causative biomarker in heart disease?. *Biomarkers in medicine*, *6*(1), 19-34.

Robert, S. A., & House, J. S. (1994). Socioeconomic status and health over the life course. Aging and quality of life, 253-274.

Roberts, A. L., Gilman, S. E., Breslau, J., Breslau, N., & Koenen, K. C. (2011). Race/ethnic differences in exposure to traumatic events, development of post-traumatic stress disorder, and treatment-seeking for post-traumatic stress disorder in the United States. *Psychological medicine*,*41*(1), 71-83.

Romano, E., & De Luca, R. V. (2001). Male sexual abuse: A review of effects, abuse characteristics, and links with later psychological functioning.*Aggression and Violent Behavior*, *6*(1), 55-78.

Romans, S., Belaise, C., Martin, J., Morris, E., & Raffi, A. (2002). Childhood abuse and later medical disorders in women. *Psychotherapy and psychosomatics*, *71*(3), 141-150.

Rooks, C., Veledar, E., Goldberg, J., Bremner, J. D., & Vaccarino, V. (2012). Early trauma and inflammation: role of familial factors in a study of twins.*Psychosomatic medicine*, *74*(2), 146.

Rosenthal, S., Feiring, C., & Taska, L. (2003). Emotional support and adjustment over a year's time following sexual abuse discovery. *Child abuse & neglect*, *27*(6), 641-661.

Romito, P., & Grassi, M. (2007). Does violence affect one gender more than the other? The mental health impact of violence among male and female university students. *Social Science & Medicine*, *65*(6), 1222-1234.

Ross, R. (1999). Atherosclerosis—an inflammatory disease. New England journal of medicine, 340(2), 115-126.

Roy, C. A., & Perry, J. C. (2004). Instruments for the assessment of childhood trauma in adults. *The Journal of nervous and mental disease*,*192*(5), 343-351.

Sansone, R. A., Wiederman, M. W., & Sansone, L. A. (1997). Health care utilization and history of trauma among women in a primary care setting.*Violence and Victims*, *12*(2), 165-172.

Sapolsky, R. M. (2000). Stress hormones: good and bad. *Neurobiology of disease*, *7*(5), 540-542.

Scarpa, A. (2004). The effects of child maltreatment on the hypothalamic-pituitary-adrenal axis. *Trauma, Violence, & Abuse*, *5*(4), 333-352.

Scher, C. D., Stein, M. B., Asmundson, G. J., McCreary, D. R., & Forde, D. R. (2001). The childhood trauma questionnaire in a community sample: psychometric properties and normative data. *Journal of traumatic stress*,*14*(4), 843-857.

Schreier, H. M., Roy, L. B., Frimer, L. T., & Chen, E. (2014). Family Chaos and Adolescent Inflammatory Profiles: The Moderating Role of SocioeconomicStatus. *Psychosomatic medicine*, *76*(6), 460-467.

Schilling, E., Aseltine, R., & Gore, S. (2008). The impact of cumulative childhood adversity on young adult mental health: measures, models, and interpretations. Social Science & Medicine, 66, 1140–1151.

Schumm JA, Stines LR, Hobfoll SE, Jackson AP. (2005) The double-barreled burden of child abuse and current stressful circumstances on adult women: the kindling effect of early traumatic experience. J Trauma Stress 2005;18:467–76.

Sedlak, A. J., Mettenburg, J., Basena, M., Peta, I., McPherson, K., & Greene, A. (2010). Fourth national incidence study of child abuse and neglect (NIS-4). *Washington, DC: US Department of Health and Human Services. Retrieved on July, 9*, 2010.

Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the human immune system: a meta-analytic study of 30 years of inquiry.*Psychological bulletin*, *130*(4), 601.

Seeman, T. E., & Crimmins, E. (2001). Social environment effects on health and aging. *Annals of the New York Academy of Sciences*, *954*(1), 88-117.

Slep, A. M. S., Foran, H. M., Heyman, R. E., & Snarr, J. D. (2010). Unique risk and protective factors for partner aggression in a large scale Air Force survey. *Journal of Community Health*, *35*(4), 375-383.

Shapiro, D. L. and A. A. Levendosky (1999). "Adolescent survivors of childhood sexual abuse: The mediating role of attachment style and coping in psychological and interpersonal functioning." Child Abuse & Neglect **23**(11): 1175-1191.

Shaw, B. A., & Krause, N. (2002). Exposure to physical violence during childhood, aging, and health. *Journal of aging and health*, *14*(4), 467-494.

Sherin, K. M., Sinacore, J. M., Li, X. Q., Zitter, R. E., & Shakil, A. (1998). HITS: a short domestic violence screening tool for use in a family practice setting. *FAMILY MEDICINE-KANSAS CITY-*, *30*, 508-512.

Slopen, N., Koenen, K. C., & Kubzansky, L. D. (2012). Childhood adversity and immune and inflammatory biomarkers associated with cardiovascular risk in youth: A systematic review. *Brain, behavior, and immunity*, *26*(2), 239-250.

Slopen, N., McLaughlin, K. A., Dunn, E. C., & Koenen, K. C. (2013). Childhood adversity and cell-mediated immunity in young adulthood: Does type and timing matter?. *Brain, behavior, and immunity*, *28*, 63-71.

Sompayrac, L. (2012). *How the immune system works*. John Wiley & Sons.

Spinazzola, J., Hodgdon, H., Liang, L. J., Ford, J. D., Layne, C. M., Pynoos, R., ... & Kisiel, C. (2014). Unseen wounds: The contribution of psychological maltreatment to child and adolescent mental health and risk outcomes.*Psychological Trauma: Theory, Research, Practice, and Policy*, *6*(S1), S18.

Stith, S. M., Liu, T., Davies, L. C., Boykin, E. L., Alder, M. C., Harris, J. M., ... & Dees, J. E. M. E. G. (2009). Risk factors in child maltreatment: A meta-analytic review of the literature. *Aggression and violent behavior*, *14*(1), 13-29.

Straus, M. A. (1979). Measuring intrafamily conflict and violence: The conflict tactics (CT) scales. *Journal of Marriage and the Family*, 75-88

Straus, M. A., Hamby, S. L., Boney-McCoy, S., & Sugarman, D. B. (1996). The revised conflict tactics scales (CTS2) development and preliminary psychometric data. *Journal of family issues*, *17*(3), 283-316.

Straus, M. A. (2010). Prevalence, societal causes, and trends in corporal punishment by parents in world perspective. *Law and Contemporary Problems*, *73*(2), 1-30.

Straus, M. A. (2005). Women's violence toward men is a serious social problem. *Current controversies on family violence*, *2*, 55-77.

Tarullo, A. R. and M. R. Gunnar (2006). "Child maltreatment and the developing HPA axis." Hormones and Behavior **50**(4): 632-639.

Taylor, S. E., Lerner, J. S., Sage, R. M., Lehman, B. J., & Seeman, T. E. (2004). Early environment, emotions, responses to stress, and health.*Journal of personality*, *72*(6),

1365-1394.
Thaller, V., Vrkljan, M., Hotujac, L., & Thakore, J. (1999). The Potential Role of Hypocortisolism in the Pathophysiology of PTSD and. *Coll. Antropol*, *23*(2), 611-619.
Thompson, M. P., Kingree, J. B., & Desai, S. (2004). Gender differences in long-term health consequences of physical abuse of children: data from a nationally representative survey. *American Journal of Public Health*, *94*(4), 599-604.
Tietjen, G. E., Khubchandani, J., Herial, N. A., & Shah, K. (2012). Adverse childhood experiences are associated with migraine and vascular biomarkers.*Headache: The Journal of Head and Face Pain*, *52*(6), 920-929.
Timpson, N. J., Lawlor, D. A., Harbord, R. M., Gaunt, T. R., Day, I. N., Palmer, L. J., ... & Smith, G. D. (2005). C-reactive protein and its role in metabolic syndrome: mendelian randomisation study. *The Lancet*,*366*(9501), 1954-1959.
Tjaden, P. G., & Thoennes, N. (2000). Extent, nature, and consequences of intimate partner violence: Findings from the National Violence Against Women Survey (Vol.181867). Washington, DC: National Institute of Justice.
Treiman, D. J. (2009). *Quantitative data analysis: Doing social research to test ideas* (Vol. 27). John Wiley & Sons.
Turner, R. J. (2009). Understanding health disparities: The promise of the stress process model. In Advances in the Conceptualization of the Stress Process (pp. 3-21). Springer New York.
Tyler, K. A. (2002). Social and emotional outcomes of childhood sexual abuse: A review of recent research. *Aggression and Violent Behavior*, *7*(6), 567-589.
Ullman, S. E., & Filipas, H. H. (2005). Gender differences in social reactions to abuse disclosures, post-abuse coping, and PTSD of child sexual abuse survivors. *Child abuse & neglect*, *29*(7), 767-782.
Upchurch, D. M., Lillard, L. A., Aneshensel, C. S., & Li, N. F. (2002). Inconsistencies in reporting the occurrence and timing of first intercourse among adolescents. *Journal of sex research*, *39*(3), 197-206.
Urtasun, R., Lopategi, A., George, J., Leung, T. M., Lu, Y., Wang, X., ... & Nieto, N. (2012). Osteopontin, an oxidant stress sensitive cytokine, up- regulates collagen- I via integrin αVβ3 engagement and PI3K/pAkt/NFκB signaling. Hepatology, 55(2), 594-608.
US Department of Health and Human Services, 2014 http://www.acf.hhs.gov/programs/cb/pubs/cm10/index.htm
Veltman, M. W., & Browne, K. D. (2001). Three decades of child maltreatment research Implications for the School Years. *Trauma, Violence, & Abuse*, *2*(3), 215-239.
Wagle, U. (2002). Rethinking poverty: definition and measurement. International Social Science Journal, 54(171), 155-165.
Wassertheil-Smoller, S., Shumaker, S., Ockene, J., Talavera, G. A., Greenland, P., Cochrane, B., ... & Dunbar-Jacob, J. (2004). Depression and cardiovascular sequelae in postmenopausal women: the Women's Health Initiative (WHI). *Archives of internal medicine*, *164*(3), 289-298.
Wegman, H. L., & Stetler, C. (2009). A meta-analytic review of the effects of childhood abuse on medical outcomes in adulthood. *Psychosomatic Medicine*,*71*(8), 805-812.
Wells, K., & Guo, S. (1999). Reunification and reentry of foster children.*Children and Youth Services Review*, *21*(4), 273-294.

Weiss, E. L., Longhurst, J. G., & Mazure, C. M. (1999). Childhood sexual abuse as a risk factor for depression in women: psychosocial and neurobiological correlates. *American journal of psychiatry*.

West, B. T., Berglund, P., & Heeringa, S. G. (2008). A closer examination of subpopulation analysis of complex-sample survey data. *Stata J*, *8*(4), 520-531.

Wheaton, B. (1994). Sampling the stress universe. In *Stress and mental health* (pp. 77-114). Springer US.

Whitaker, D. J., Haileyesus, T., Swahn, M., & Saltzman, L. S. (2007). Differences in frequency of violence and reported injury between relationships with reciprocal and nonreciprocal intimate partner violence.*American Journal of Public Health*, *97*(5), 941-947.

Whitsel,E. Cuthbertson, C. Tabor, J (2012) Measures of inflammation and immune Function, Add Health Wave IV Documentation.

Widom, C. S., & White, H. R. (1997). Problem behaviours in abused and neglected children grown up: prevalence and co-occurrence of substance abuse, crime and violence. *Criminal Behaviour and Mental Health*, *7*(4), 287-310.

Widom, C. S., Czaja, S., & Dutton, M. A. (2014). Child abuse and neglect and intimate partner violence victimization and perpetration: A prospective investigation. *Child abuse & neglect*, *38*(4), 650-663.

Woods, S. B., Farineau, H. M., & McWey, L. M. (2013). Physical health, mental health, and behaviour problems among early adolescents in foster care. *Child: care, health and development*, *39*(2), 220-227.

Wolfe, D. A., & McGEE, R. O. B. I. N. (1994). Dimensions of child maltreatment and their relationship to adolescent adjustment. *Development and Psychopathology*, *6*(01), 165-181.

Wright, M. O. D., Crawford, E., & Del Castillo, D. (2009). Childhood emotional maltreatment and later psychological distress among college students: The mediating role of maladaptive schemas. *Child Abuse & Neglect*, *33*(1), 59-68.

Yehuda, R. (2000). Biology of posttraumatic stress disorder. *The Journal of clinical psychiatry*, *61*(suppl 7), 1-478.

Yudkin, J. S., Stehouwer, C., Emeis, J., & Coppack, S. (1999). C-Reactive Protein in Healthy Subjects: Associations With Obesity, Insulin Resistance, and Endothelial Dysfunction A Potential Role for Cytokines Originating From Adipose Tissue?. *Arteriosclerosis, thrombosis, and vascular biology*, *19*(4), 972-978.