Early Origins of Adult Physiological Dysregulation

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Early Origins of Adult Physiological Dysregulation

Abstract

Most research connecting child maltreatment to adult inflammation is non-representative, theoretically limited, and does not consider gender differences. Guided by biological embedding, this study investigated the relationships among child maltreatment, adult chronic inflammation, and gender using a national sample of 1,243 midlife and older adults. Child abuse and neglect variables were used to predict adult interluekin-6 levels. The moderating effect of gender was also examined. Findings revealed that, when controlling for adult covariates, childhood physical neglect predicts elevated interluekin-6. Although gender did not moderate the effect of physical neglect, inflammation levels were higher among women than men. In addition, women were more likely to experience sexual and emotional abuse during childhood. The present study adds to the emerging literature investigating the interplay between the social environment and biological markers of health, and the importance of gender as a social structure in predicting exposure to child abuse.

Key Words: Aging and the Life Course, Child Abuse, Child Neglect, Chronic Inflammation

Early Origins of Adult Physiological Dysregulation

Introduction

Exposure to stress during childhood is a common experience, and, at times, can lead to beneficial adaptive and coping mechanisms. However, some types of childhood stress—toxic stress—entail prolonged activation of stress response systems and result in long-term physiological damage (National Scientific Council on the Developing Child 2005). One form of toxic stress is child maltreatment, which refers to any act of child abuse or neglect, and its long-term effects have been well-documented (Center for Disease Control and Prevention 2012; National Scientific Council on the Developing Child 2005). Adults who experienced maltreatment during childhood are at increased risk for several diseases, such as chronic morbidity, ischemic heart disease, hypertension, and cancer as well as premature mortality (Brown et al. 2009; Felitti et al. 1998; Greenfield and Marks 2009; Morton, Schafer, and Ferraro 2012). These empirical findings of the last two decades resonate with Herbert Ward's (n.d.) observation from working with children at St. Jude's Children's Ranch: "Child abuse casts a shadow the length of a lifetime."

To understand the enduring effects of maltreatment, scholars have begun to identify mechanisms that may help explain how child maltreatment becomes manifest as poor physical adult health. The majority of this burgeoning literature has primarily focused on behavioral, emotional, cognitive, and social pathways (Kendall-Tacket 2002). With the continual focus on psychosocial links, one fundamental question remains: How does child maltreatment "get under the skin"? Recent findings indicate that maltreatment is associated with physiological changes in the HPA axis, immune functioning, and telomere length. However, these studies are often theoretically limited (solely informed by allostatic load), focus on one type of maltreatment or combine multiple forms of maltreatment into a single measure, and utilize relatively small, non-

representative community samples (e.g., Carpenter et al. 2010; Carpenter et al. 2011; Cicchetti, Rogosch, and Oshri 2011; Danese et al. 2007; O'Donovan et al. 2011; Shalev et al. 2013). Although these studies have significantly contributed to our knowledge of the biological mechanisms of maltreatment, a theoretically-drive population study can help explain how child maltreatment "gets under the skin" and whether these initial studies are generalizable to the U.S. population. Therefore, the purpose of this paper is to provide a more comprehensive and systematic approach to elucidate how experiences of child maltreatment transpire on a physiological level, impacting the health and aging process of adults over the life course. Specifically, I ask whether child maltreatment is associated with higher levels of inflammation in adulthood in a national study of U.S. adults. In doing so, I examine multiple domains of child maltreatment. I also ask whether the relationship between child maltreatment and adult chronic inflammation varies by gender. To answer these questions, the present study uses an interdisciplinary approach to discuss the biological mechanisms of maltreatment and their interrelated relationships in light of Hertzman and Boyce's (2010) biological embedding—a comprehensive framework into which concepts of allostatic load and life course epidemiology can be incorporated. This paper begins with a discussion of prior literature, followed by Hertzman and Boyce's (2010) biological embedding, which provides a theoretical platform for the main thesis of the paper: child maltreatment gets under the skin because it entails biological consequences that are long-lasting.

The Biological Imprint of Child Maltreatment

Over time, maltreated children exhibit biological signs of deterioration in adulthood. Emerging literature has identified three physiological systems that can be altered by experiencing maltreatment. First, the stress response system, which has been given the most attention, can

display signs of dysregulation. Most of the research on child maltreatment and the stress response system has focused on the hypothalamic-pituitary-adrenal (HPA) axis, using cortisol as the main indicator of dysregulation. Adults who were maltreated during childhood show disruptions to both basal levels and stress reactivity regarding the HPA axis. Maltreatment has consistently been associated with flattened diurnal cortisol levels in adulthood (Cicchetti et al. 2011; Trickett, Noll, Susman, Shenk, and Putnam 2010; van der Vegt, van der Ende, Kirschbaum, Verhulst, and Tiemeier 2009). In addition, abnormal stress reactivity has also been cited in the maltreatment literature. Similar to the diurnal cortisol studies, several studies have found maltreatment to be indicative of a blunted cortisol response system in adulthood (Carpenter et al. 2009; Carpenter, Shattuck, Tyrka, Geracioti, and Price 2011). These studies reveal that the presence of abnormal stress reactivity is common among survivors of maltreatment. Since an impaired HPA axis can lead to cardiovascular disease (CVD), type 2 diabetes, and stroke (Rosmond and Bjorntrop 2000), maltreatment may indirectly influence adult health through an impaired HPA axis.

Second, child maltreatment can also compromise immune functioning as illustrated by its association with adult inflammation. Adults who were maltreated as children show increased production of the proinflammatory cytokine interleukin-6 (IL-6) (Carpenter et al. 2010). Elevated levels of C-reactive protein, fibrinogen, white blood cells, and lymphocytes have also been observed in adults who experienced maltreatment during childhood (Carpenter et al. 2010; Danese et al. 2007; Surtees et al. 2003). A compromised immune system can negatively interfere with wound healing and vaccine response as well as increase the risk of cancer and CVD (Dandona, Alijada, and Bandyopandyay 2004; Marketon and Glaser 2008; Yudkin, Kumari, Humphries, and Mohamed-Ali 2000). Thus, child maltreatment may lead to poor adult health via a compromised immune system.

Third, recent advancements in telomere research have revealed that maltreatment may biologically embed on a cellular level. Most health and aging research has focused on telomere length, suggesting that the shortening of telomeres, which occurs over time due to molecular events such as damage to DNA or cell division, leads to cellular senescence, which is part of the aging process (Balckburn 2005; Blasco 2005; Stewart and Weinberg 2000). Whereas this degradation of telomeres is a natural process, premature erosion of telomeres can be indicative of accelerated aging. Adults who were maltreated as children appear to have shorter telomeres than adult who were not maltreated as children (O'Donovan et al. 2011; Shalev et al. 2013; Tyrka et al. 2010). Although there is no set threshold of telomere length to indicate cellular senescence, the process of telomere erosion has been linked to disease (Blasco 2005; Stewart and Weinberg 2000). Shortened telomeres are found in cases of CVD, cancer, and diabetes as well as a precursor to death (Cawthon, Smith, O'Brien, Sivatchenko, and Kerber 2003; Chang and Harley 1995; Ma et al. 2011; Sampson, Winterbone, Hughes, Dozio, and Hughes 2006; Yang et al. 2011). Therefore, a third pathway through which child maltreatment may influence adult health is telomere shortening.

Biological embedding of child maltreatment

Among the many explanations for how child maltreatment gets under the skin, the most cogent theoretical framework is Hertzman and Boyce's (2010) biological embedding, which posits that certain circumstances, events, and experiences can cause permanent, long-term damage via their physiological effects. Two components of biological embedding are essential in understanding the long-arm of child maltreatment. First, childhood is seen as a sensitive period, distinguishing biological embedding from allostatic load. Childhood comprises a substantial period of growth and development during which biological systems are calibrating

and, therefore, are susceptible to external influences. During this time, environmental stressors have the potential to disrupt these developing systems. For instance, the HPA axis is particularly malleable during the early years of life as regulatory stress response systems are being set (Tarullo and Gunnar 2006). As demonstrated by allostatic load research, chronic or severe exposure to stress during childhood can cause permanent damage to the HPA axis that incurs over a lifetime (McEwen 1998). By framing childhood as a sensitive period, biological embedding strengthens the case for allostatic load by providing theoretical plausibility for how childhood events can manifest on a physiological level.

Second, biological embedding posits that these biological disturbances alter biological processes in stable ways that endure throughout life. As revealed above by the prior literature, maltreatment appears to have met this requirement. Unlike allostatic load, biological embedding explicates how these environmental insults can incur damage via latency, accumulation, and pathways over time. This conceptualization is similar to life course epidemiologists' conceptual model of accumulation of risks, reflecting the integrative nature of biological embedding (Ben-Shlomo and Kuh 2002). One specific aspect of life course epidemiology's accumulation model that pertains to the process of biological embedding is chain of risks.

The physiological unfolding of child maltreatment via a chain of risks

How poor health accumulates over the life span can often be explained by the life course epidemiological concept, chain of risk concept, which is analogous to the domino effect: the occurrence of one event is likely to lead to a subsequent event (Ben-Shlomo and Kuh 2002). As mentioned above, biological embedding may occur through an accumulation process, such as a chain of risks, or "pathways" as labeled by Hertzman and Boyce (2010). Whereas the bulk of prior research has depicted a long latency between exposure to child maltreatment and onset of

disease, it can be argued that there is a biological chain of risks occurring behind the scenes that has, until recently, gone undetected. Thus, maltreatment may lead to an unfolding process of detrimental physiological processes that can impact health in later in life. There is evidence to suggest that this process begins with the stress response system, which is responsible for setting off a cascade of physiological reactions in response to stress that appear to infiltrate the other aforementioned biological systems.

In response to stress, the hypothalamus releases cortocotropin-releasing hormone (CRH), which signals the anterior pituitary gland to release adrenocorticotropic hormone (ACTH), which, in turn, signal the adrenal gland to release glucocorticoids (Schury and Kolassa 2012). These glucocorticoids released by the HPA axis modulate proinflammatory immune signaling (Webster, Tonelli, and Sternberg 2002). Whereas elevated levels of glucocorticoids suppress immune functioning and lead to risk of disease, suppressed levels of glucocorticoids can lead to increased inflammation (Webster et al. 2002). Initially, the HPA axis may flood the system with glucocorticoids, increasing the risk of disease, but later, as the HPA axis becomes blunted due to negative feedback at the hypothalamic level and decreased levels of glucocorticoids are released, inflammation becomes more prevalent. Through the modulatory effects of persistently elevated glucocorticoids, chronic exposure to stress can result in increased levels of inflammation (Webster et al. 2002). Thus, higher levels of inflammation that have been observed in adults who were maltreated during childhood may be attributed to a dysregulated HPA axis. Whereas cortisol levels fluctuate daily and, often, lower measures indicate a blunted, dysregulated system, the present study focuses on inflammation since chronic high inflammation is indicative of a poorly regulating immune system as well as an indirect measure of poor HPA axis functioning. In addition, compared to research on cortisol, relatively few studies have investigated adult inflammation as a consequence of child maltreatment.

Given the toxic nature of maltreatment and its implications as a precursor to many adult diseases that often have biological underpinnings, child maltreatment is a suitable candidate to demonstrate biological embedding. To test the biological embedding hypothesis and extend prior literature, the following hypotheses are proposed:

H1: Child abuse is associated with higher levels of adult inflammation.

H2: Child neglect is associated with higher levels of adult inflammation.

The role of gender

Although gender is often considered an individual ascribed status, the present study draws from recent theoretical developments that conceptualize gender as a social structure (Risman 2004). Structures are, by definition, embedded within multiple societal dimensions—individual, organizational, institutional, and international. The pervasiveness of gender throughout social processes and organizations as well as its ability to provide opportunities or constraints for agency qualifies it as social structures (2004). Conceiving of gender as social structures, this study investigates how the social structure of gender may influence the relationship between childhood misfortune and adult inflammation.

Although limited attention has been given to gender differences in the effects of childhood maltreatment on adult chronic inflammation, patterns of childhood misfortune and adult inflammation appear to vary by gender. Prior research on the childhood origins of adult health suggests that the effect of childhood disadvantage on adult health is stronger for women than men (Hamil-Luker and O'Rand 2007). A possible explanation is that gender socially patterns how individuals respond to misfortune: women have reported similar events as more upsetting and requiring a longer recovery period than men (Surtees and Wainwright 2007). Related to the present study, the effect of childhood disadvantage on adult inflammation appears

to vary by gender (Brummett et al. 2013). Given the emerging evidence indicating that gender may influence the relationship between childhood experiences and adult inflammation, the present study investigates the moderating effect of gender and proposes the following hypothesis:

H3: The relationship between child maltreatment and adult chronic inflammation varies by gender.

Methods

Sample

The data come from the National Survey of Midlife Development in the United States (MIDUS). MIDUS participants were selected from working telephone banks in the contiguous United States using a national random digit-dialing sample. During 1995, MIDUS surveyed 3,032 English-speaking, non-institutionalized men and women aged 25-74 with an over-sample of adults aged 65-74 and men. After completing the initial computer assisted telephone interview, respondents were mailed self-administered questionnaires. The response rate for completing both the telephone interview and mail questionnaires was 61%. Approximately ten years later, between 2004 and 2006, participants were re-interviewed with similar telephone interview and self-administered mail questionnaires. Of the 3,032 original respondents, 2,101 participated in the follow-up telephone interview, yielding a response rate of 69.5% (71% mortality-adjusted response rate). A subsample was selected from the 2004-2006 follow-up to assess biological measures (N=1,243), which were gathered between 2004 and 2009. This study draws from that subsample.

Measures

Adult inflammation. To assess inflammation levels, the present study used interluekin-6. IL-6 was measured by a morning fasting blood test drawn from respondents while under hospital observation per the study's protocol. Blood samples were then stored for assay. The laboratory's intra-assay coefficient of variance was 4.09% and the inter-assay coefficient of variance was 13%. IL-6 is a continuous variable, ranging from 0.16-23 pg/mL. To adjust for skewness, IL-6 was log-transformed.

Childhood maltreatment. Drawing from prior conceptualization research, child maltreatment variables were created following the Childhood Trauma Questionnaire (Bernstein and Fink 1998). The first set of childhood maltreatment variables comprised two variables of child abuse: emotional abuse and physical abuse. For both variables, participants were asked how often they experienced five different items during childhood. Responses for each item of physical and emotional abuse range from (1) never to (5) very often. For emotional abuse, respondents indicated how often family members called him/her names; said hurtful or insulting things; felt hated; parents wished child never born; were emotionally abused. For physical abuse, respondents indicated how often family members hit him/her hard enough to go to hospital, leave a bruise, or noticed by someone (e.g., teach, neighbor, doctor); punished with hard object (e.g., belt, cord); were physically abused.

The second set of variables assesse child neglect, and also consisted of two variables: emotional neglect and physical neglect. For both variables, participants were asked how often they experienced five different items during childhood. For emotional neglect, respondents were asked if a family member made him/her feel important or special; felt loved; family members looked out for each other; family felt close to each other; and family source of strength and support. For physical neglect, respondents were asked if he/she didn't have enough to eat; knew

someone could protect him/her; parents too drunk/high to provide care; had to wear dirty clothes; and if someone could take him/her to doctor if need arose.

Scales for emotional abuse (α=0.878), physical abuse (α=0.794), emotional neglect (α=0.892), and physical neglect (α=0.698) were created by summing across all items for each respective measure. Items for emotional neglect were first reverse-coded, so that higher scores indicate higher standing. Similarly, two items for physical abuse were also reverse-coded prior to scaling. For cases with only one missing value, mean substitution was used. Raw scales ranged from 5-25, but the log-transformation of each variable was taken to adjust for skewness.

Gender. A binary variable of gender was created based on respondents' self-reports of gender. A dummy variable for female was created (1=female; 0=male).

Covariates. To adjust for several factors that may explain variation in inflammation levels, covariates included age, race, marital status, anxiety, cardiovascular disease (CVD), and obesity, smoking, and prescription medications. Age was a continuous variable, ranging from 34-84. Dummy variables were created for race (1=nonwhite), marital status (1=married). Dummy variables were also created for several health indicators: CVD (1=physician diagnosis, 0=otherwise), obesity (1= BMI $\geq$ 30), smoker (1=current smoker), and prescription medications (1=currently taking prescription medication). Three kinds of prescription medications were included in the final analyses. These included prescription medications for high blood pressure, cholesterol, and corticosteroids, and a dummy variable was included for each medication. To adjust for potential psychosocial influences, anxiety was included. The scale for anxiety was created using 9 items that asked respondents how often they felt fear or anxiety when talking to people; going to a party; working under observation; calling, talking, or disagreeing with someone he/she doesn't know well; being center of attention; returning goods to a store; and

resisting high-pressure salesperson (Fresco et al. 2001). For respondents with <2 missing items, a scale was constructed by computing the mean across all 9 items (α=0.852).

Analytic design

Analyses were conducted using Stata, version 14.0. Descriptive statistics were conducted for the full sample and by gender. For the final analyses, models first included only the child maltreatment variables as predictors to establish a baseline relationship between child maltreatment and adult inflammation. Next, adult covariates were entered into the models. Preliminary analyses examined gender as a moderator of child maltreatment and adult inflammation. However, there was no statistical evidence for moderation, and these results are not presented (but available upon request). Therefore, final models presented are estimated on the full sample. In addition, sensitivity analyses investigated alternative specifications of child maltreatment, including a summary score and latent variables assessing constructs of abuse and neglect. Results indicated that each variable of abuse and neglect was a distinct construct. Because none of the independent variables had more than 5% missing, listwise deletion was used for item-missing data.

Results

Descriptive statistics

Descriptive statistics of key variables are presented in Table 1. As shown, the average value of logged IL-6 was 0.849 for women and 0.750 for men. Emotional neglect was the most commonly type of maltreatment reported for men and women. The average values of logged sexual, emotional, physical abuse were, respectively, 1.862, 2.034, and 1.879 for women. For men, average values of logged sexual, emotional, physical abuse were 1.689, 1.919, and 1.881,

respectively. The average values of logged emotional and physical neglect for women were 2.183 and 1.879, respectively. For men, average values of logged emotional and physical neglect were 2.172 and 1.860, respectively. Inflammation, sexual abuse, and emotional abuse varied by gender, with women have higher levels of each. Although not shown, health-related covariates indicated that the sample, in general, was comprised of relatively healthy adults. Most respondents were non-smokers without CVD; less than half were obese, and approximately less than a third were taking any of the prescription medications.

TABLE 1 APPROXIMATELY HERE

Child maltreatment and adult inflammation

As stated above, the first model only included the child maltreatment variables as predictors of adult inflammation. Among the five variables of child maltreatment, only child sexual abuse and physical neglect predict adult inflammation. More frequent sexual abuse in childhood was associated with higher levels of IL-6 in adulthood (b=0.188, p<0.01). Similarly, more frequent physical neglect was associated with higher levels of IL-6 in adulthood (b=0.318, p<0.001).

TABLE 2 APPROXIMATELY HERE

In Model 2, adult covariates were introduced into the model. After adjusting for potential adult risk factors and confounders, the effect of child sexual abuse on adult inflammation was fully attenuated and became non-significant. The effect of child physical neglect was attenuated, but remained significant (b=0.189, p<0.05). More frequent physical neglect was associated with higher levels of IL-6 in adulthood.

Discussion

The present study adds to the emerging literature investigating the interplay between the social environment and biological markers of health. Utilizing a national sample of American adults, this study revealed a relationship between child maltreatment and adult inflammation on a population level, as hypothesized by biological embedding. In doing so, three main hypotheses were tested. The first hypothesis stated that child abuse was associated with adult inflammation levels. Among the three variables of child abuse, only sexual abuse predicted higher levels of IL-6 in adulthood. However, once adult covariates were added to the model, child sexual abuse was no longer significant. Although this does not support H1 per biological embedding, some of the adult covariates may be potential mediators of child sexual abuse and adult inflammation, such as smoking, BMI, and education.

The second hypothesis stated that child neglect would be associated with higher levels of inflammation in adulthood. Between the two variables of child neglect—physical and emotional neglect—physical neglect was associated with higher levels of IL-6 in adulthood. This relationship remained, net of adult demographics, health behaviors, and potential confounding factors, supporting Hertzman and Boyce's (2010) biological embedding hypothesis. Thus, among the five variables of child maltreatment, only child physical neglect appears to biologically embed. Several potential reasons are given to explain the inconsistency between the findings presented herein and those in prior studies.

First, respondents in the biomarker subsample of MIDUS were, in general, relatively healthy. Less than a fifth, for instance, were current smokers or had been diagnosed with a heart condition. Moreover, less than a third were on commonly prescribed medications. However, this selection issue would be expected—unhealthy people would be less likely to want or be able to participate. In addition, the MIDUS subsample was taken at the second time period; the

unhealthiest participants most likely attrited due to death or illness. Moreover, those who experienced the most or severest child maltreatment are also more likely to be affected by poor health and premature mortality. Second, basal biological measures may not be the best measures of dysregulation. Emerging research on animal models suggests that physiological measures should be taken after stress systems are challenged (e.g., Waters 2013). Response to challenge is perhaps a better measure of dysregulation. Other limitations of the present study include its cross-sectional design and the retrospective nature of the child maltreatment measures.

Nonetheless, the present study contributes to the current literature on child maltreatment and biomarkers by demonstrating that not all experiences of child maltreatment are equal in their impact. As revealed, child abuse and neglect appear to have differential effects on adult physiology, with child neglect, and more specifically physical neglect, appearing to have more salient and lasting effects on adult physiology.

The third hypothesis expected that the relationship between child maltreatment and adult inflammation would vary by gender. Although the relationship between child maltreatment and adult inflammation did not vary by gender, bivariate relationships indicated that the likelihood of child abuse varied by gender. Women were more likely than men to experience sexual and emotional abuse during childhood. Regarding the lack of gender differences in models predicting adult inflammation, a possible explanation for these null findings may be the kind of childhood adversity examined. Whereas most of the childhood disadvantage-adult health research citing gender difference has used childhood SES (e.g., Brummet et al. 2013; Hamil-Luker and O'Rand 2007), the present study investigated the long-term effect of child maltreatment. Perhaps some groups may be more resilient to less toxic forms of childhood adversity, whereas other kinds of childhood experience that are more toxic, such as child maltreatment, exert inexorable effects regardless of gender.

Given the limited literature on child maltreatment and biological mechanisms, there several recommendations for future research. First, the chain of risks following maltreatment is a multifaceted process that can also include health behaviors, SES, and genetic predispositions among many other potential factors. Scholars from various fields investigating the biological effect of maltreatment may often focus on covariates that are only specific to their discipline. For instances, whereas medical sociologists give priority to socioeconomic status, many studies do not account for its effect (e.g., Carpenter et al. 2011). These studies may be over-estimating the effect of child maltreatment. Future research can also be informed by biological studies, such as the epigenetic findings in the animal studies. Indeed, life course health research requires an interdisciplinary approach in which scholars from different academic backgrounds work together to help bridge the gaps of the current research.

Second, data should be longitudinal with large, representative samples (e.g., Price et al. 2013). Most studies on maltreatment and biological measures are cross-sectional and comprised of small samples. Within the past few decades, some promising representative, longitudinal studies have been appearing, such as the Health and Retirement Study that have measures of childhood as well as biological markers. Future research should take advantage of this new era of biomarker collection in social surveys.

Third, longitudinal studies would also enable tests of mediation to empirically show how the effect of maltreatment on adult health and mortality operates through biological changes. Tests of mediation that map out the trajectory of disease are necessary, as we are reminded by Hertzman and Boyce (336:2010) that "to meet the test of biological embedding not only must the experiences have biological effects, but also these effects must, in turn, influence long-term human developmental outcomes and the expression of gradients in human development".

Testing these components of biological embedding will likely call for a multidisciplinary approach that maps out the life course of child maltreatment.

Policy implications

As average life expectancy increases, the focus of many gerontological fields has shifted toward "optimal aging". Whereas much is known about adult risk factors that may threaten optimal aging, a burgeoning literature has been identifying risk factors that occur much earlier in life. Thus, linking child maltreatment to biomarkers associated with poor adult health and aging provides evidence that interventions geared toward optimal aging need to be implemented before adulthood. Health policymakers should intervene to reduce exposure to child maltreatment, especially for women who are more likely to experience certain types of abuse. Despite current efforts to reduce the incidence of child maltreatment, the epidemic of child maltreatment continues, with approximately 20% of children in America experiencing some form of maltreatment (Finkelhor et al. 2009). As scholars continue to learn more about the enduring effects of child maltreatment, this information must be disseminated and interventions implemented to the public at every level—from public policy to primary care physicians and social workers.

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Table 1. Descriptive Statistics for Key Variables,
Midlife Development in the U.S. Biomarker Study

Variables[a]	Range	Female Mean(SD)	Male Mean(SD)
Inflammation			
IL-6	-1.833-3.135	*0.849(0.766)*	*0.750(0.726)*
Child Abuse			
Sexual Abuse	1.609-3.219	*1.862(0.454)*	*1.689(0.246)*
Emotional Abuse	1.609-3.219	*2.034(0.455)*	*1.919(0.363)*
Physical Abuse	1.609-3.219	1.879(0.351)	1.881(0.300)
Child Neglect			
Emotional Neglect	1.609-3.219	2.183(0.466)	2.172(0.419)
Physical Neglect	1.609-3.045	1.879(0.347)	1.860(0.309)
N		704	539

[a]Variables log-transformed to adjust for skewness.

Note: Italicized mean(SD) indicates significant gender difference at 0.05 level.

Table 2. Regression of Interluekin-6 on Child Maltreatment, MIDUS (N=1,243)

	Model 1		Model 2	
Variables	*b*[a]	SE[b]	*b*[a]	SE[b]
Child Abuse				
Sexual Abuse	0.188	0.064**	0.033	0.064
Emotional Abuse	-0.040	0.078	0.009	0.078
Physical Abuse	-0.065	0.089	-0.124	0.089
		0.069		0.069
Child Neglect				
Emotional Neglect	-0.091	0.086	-0.026	0.086
Physical Neglect	0.318	0.002***	0.189	0.002*
R2	0.023		0.174	
F-test	5.82***		15.32***	

*p<0.05; **p<0.01; ***p<0.001 (two-tailed tests).

[a]Unstandardized coefficient.

[b]Standard error.

Note: Model 2 adjusted for age, gender, race, education, marital status, BMI, smoking, health status, and medications.