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Social Disadvantage, Severe Child Abuse, and Biological Profiles in Adulthood

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Abstract

Guided by the stress process model and the life course perspective, we hypothesize: (1) that childhood abuse is concentrated, in terms of type and intensity, among socially disadvantaged individuals, and (2) that experiencing serious abuse contributes to poor biological profiles in multiple body systems in adulthood. Data came from the Biomarker subsample of Midlife in the United States (2004–2006). We used latent class analysis to identify distinct profiles of childhood abuse, each reflecting a combination of type and severity. Results indicate that disadvantaged groups, women, and those from disadvantaged families are at greater risk of experiencing more severe and multiple types of abuse. Those with more severe and multifaceted childhood abuse show greater physiological dysregulation. Childhood abuse experiences partially accounted for the social status differences in physiological profiles. Our findings underscore that differential exposure to serious childhood stressors plays a significant role in gender and class inequalities in adult health.

Keywords

biomarkers, childhood abuse, gender, health, life course, social class

Life course approaches to health suggest that an individual's early life experiences are precursors to later health outcomes (Kuh et al. 2003). Child abuse is one of the most widely studied adverse experiences in early life (Gilbert et al. 2009), and socially disadvantaged individuals, including women and those in lower classes, are at greater risk of being abused (Sedlak, Mettenburg, et al. 2010). Although the deleterious effects of child abuse have been studied extensively and are linked to a wide array of health outcomes, including biological risk (Danese and McEwen 2012; Felitti et al. 1998), previous research is limited in several ways.

First, differential exposure to stressful life experiences is a principal mechanism that contributes to social status differences in health (Pearlin et al. 2005; Turner 2013). Yet, few studies have investigated whether social characteristics (gender, race, childhood socioeconomic status [SES]) shape the nature of abuse that individuals encounter in their early lives and the extent to which childhood abuse contributes to social status differences in physiological dysregulation.

Second, despite the importance of using comprehensive measures of stressors to investigate health disparities (Thoits 2010), prior studies have relied on narrow definitions of abuse. These studies use either binary indicators (i.e., abuse occurred or did not occur) or broad summary indices, which may fail to capture the complex nature of abuse—for example, the type, severity, and frequency (Bernstein et al. 2003). Explicating the specific nature of child abuse is thus important for investigations into the social origins and biological consequences of abuse.

Third, prior work noted that stressful life experiences are associated with dysregulation in a number of physiological systems, yet the results vary by physiological system and measures of biological risk (Goldman et al. 2005). These findings convey

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the importance of investigating a broad spectrum of biomarkers across multiple systems to better capture the adverse effects of childhood abuse. However, previous studies have been limited to a single biomarker or physiological pathway. Two recent studies have investigated cumulative dysregulation across multiple systems (Carroll et al. 2013; Friedman et al. 2015), but these inquiries focused on only emotional and physical abuse.

Finally, prior studies have not fully taken into account that dysregulation in stress hormones may present in different ways. For example, some studies have reported that early life adversities increase hypothalamic-pituitary-adrenal (HPA) axis and sympathetic nervous system (SNS) activities, thus raising levels of stress hormones (De Bellis et al. 1999; Gonzalez et al. 2009). In contrast, others found that severe or chronic stress leads to down-regulation of the primary stress systems and hence below-normal levels of stress hormones (Yehuda et al. 1993). Moreover, there is evidence that individuals who experience chronic and extreme stress can exhibit an imbalance of stress hormones, such as elevated norepinephrine in conjunction with reduced epinephrine and cortisol secretion, which has the potential to lead to adverse health consequences, including chronic fatigue and pain syndromes (Loevinger et al. 2007; Mason et al. 1988). Our study builds on and extends prior research by incorporating diverse evidence (e.g., hyper- and hyposecretion of cortisol) and novel indices (e.g., the ratio of norepinephrine to cortisol), along with conventional biomarker indicators.

Using middle-aged adults from the National Survey of Midlife Development in the United States (MIDUS), the current study has three goals: (1) to identify different categories of childhood abuse by incorporating unique combinations of type and severity of abuse; (2) to assess whether socially disadvantaged groups, women, nonwhites, and individuals who grew up in disadvantaged families are at greater risk of exposure to more severe and multiple types of abuse; and (3) to test whether childhood abuse contributes to the relationship between social characteristics and physiological dysregulation in adulthood.

BACKGROUND

Social Status, Stress, and Health

Almost everyone encounters stressful life experiences, yet not everyone is equally exposed to or affected by stressors. According to the social process model (Pearlin 1989), the risk of experiencing stressors is significantly impacted by social characteristics, like race, gender, and SES. Specifically, socially disadvantaged individuals, including women, members of racial-ethnic minority groups, and low-SES individuals, tend to encounter more frequent and intense stressors across the life course (Lantz et al. 2005; Turner, Wheaton, and Lloyd 1995). Moreover, faced with similar stressors, individuals with lower SES are more likely to experience elevated emotional distress compared to those with higher SES (McLeod and Kessler 1990), and women, compared to men, tend to rate life adversities as less controllable (Matud 2004) and having a more negative impact on their lives (C. Lee, Glei, et al. 2014; C. Lee, Rodríguez, et al. 2014). Such susceptibility among socially disadvantaged individuals is partially attributed to fewer stress-dampening resources and less effective coping strategies as well as to lower psychological strength (e.g., self-esteem; Pearlin et al. 2005). Exposure to negative life events and ongoing stressors has been linked with a variety of deleterious health outcomes, and cumulative evidence shows that health disparities, particularly in terms of mental health, can be partially explained by differential exposure to stressors between disadvantaged and advantaged individuals (Thoits 2010). For example, using comprehensive measures of stressors, Turner and colleagues reported that almost half of the SES gap and one fifth of the gender gap in depressive symptoms were explained by life events (Turner et al. 1995; Turner and Avison 2003), indicating the significant role of stressors as a key pathway to poor health outcomes.

Social Status and Exposure to Childhood Abuse

Some stressful life experiences that shape trajectories of individuals' health and well-being occur in early life. Childhood abuse is one of the most widely studied early life adversities (Gilbert et al. 2009), and like other life events, exposure to childhood abuse is not randomly distributed throughout the population. Children who grow up in families with low income (Cancian, Yang, and Slack 2013), unemployed parents (Gillham et al. 1998), poorly educated parents (e.g., less than high school degree; Cancian et al. 2013), or single parents (Mersky et al. 2009) are at greater risk of being exposed to abuse or neglect than their counterparts. Gender is a significant sociodemographic predictor. While findings related to physical or emotional abuse have been mixed, girls are at greater risk of being sexually abused than boys (Maikovich-Fong and Jaffee

2010; Thompson, Kingree, and Desai 2004). Although the stigma against submissiveness and same-sex sexual contact may render boys reluctant to report sexual abuse (Holmes and Slap 1998), the disproportionately greater risk of sexual abuse among girls has been stable across different assessments, times, and societies (Gilbert et al. 2009).

The prevalence of childhood abuse also varies by race, with greater risk for African Americans than non-Hispanic whites (Sedlak, Mettenburg, et al. 2010). However, racial differences may be confounded with other characteristics. National studies show that racial differences in abuse weaken or are no longer statistically significant when controlling for other sociodemographic characteristics (Hussey, Chang, and Kotch 2006) or when limited to children living in low-income households (Sedlak, McPherson, and Das 2010). In sum, social characteristics, particularly, gender and childhood SES, are significantly associated with an individual's risk of exposure to childhood abuse. Few studies have investigated how such social characteristics are associated with heterogeneity of childhood abuse in terms of severity and types of abuse. Based on the differential exposure hypothesis, we examined whether women, nonwhites, and those from disadvantaged families are at greater risk of exposure to multiple types of severe abuse in childhood.

Childhood Abuse, Adult Health, and Biological Profiles

Experiencing abuse during childhood can have persistent and negative consequences on adult health (Felitti et al. 1998). Mounting evidence indicates that adults who experienced abuse as children suffer from more chronic diseases than those who were not traumatized (Norman et al. 2012). In particular, victims of child abuse are at greater risk of developing stress-related immune disorders, including allergies, asthma, and autoimmune diseases (Dube et al. 2009) as well as metabolic and vascular diseases, including type 2 diabetes, hypertension, and heart disease (Dong et al. 2004; Fuller-Thomson, Stefanyk, and Brennenstuhl 2009; C. Lee, Tsenkova, and Carr 2014; Rich-Edwards et al. 2010; Riley et al. 2010). Adult survivors of childhood abuse are overrepresented in many clinical patient groups, including irritable bowel syndrome, fibromyalgia, and extreme obesity (Drossman et al. 1990; Walker et al. 1997). Diverse health risks likely arise from the cumulative impact of biological dysregulation beginning during early development and continuing into adulthood (Danese and McEwen 2012).

Childhood abuse tends to carry a cumulative biological toll into adulthood, impacting multiple body systems, yet evidence varies by individual systems. The association is pronounced with indicators of secondary stress systems, including inflammatory, cardiovascular, and metabolic function biomarkers (Carroll et al. 2013; Friedman et al. 2015). For example, individuals who experienced abuse as children show higher adiposity, increased glucose levels, elevated blood pressure, and a greater risk of developing metabolic syndrome (Danese et al. 2009; Danese and Tan 2013; C. Lee, Tsenkova, et al. 2014; Thomas, Hypponen, and Power 2008). Similarly, adults with a history of childhood abuse often present with signs of subclinical inflammation and infection, including elevated white blood cell counts, C-reactive protein (CRP), and fibrinogen, along with higher circulating levels of proinflammatory cytokines, such as interleukin-6 (IL-6; Slopen et al. 2010; Taylor et al. 2006).

Evidence regarding the effects of early life adversities on the primary stress system has been mixed. In the association with SNS activity, some studies have documented hyperactivity (Oosterman et al. 2010), while others did not find significant association (Carroll et al. 2013; Friedman et al. 2015). Inconsistent findings have also been reported for cortisol as an indicator of activity of HPA axis. Various studies have found (1) hyperactivity (Cicchetti and Rogosch 2001; Wismer Fries, Shirtcliff, and Pollak 2008), (2) attenuated or blunted activity (Trickett et al. 2010), and (3) no significant association (Friedman et al. 2015). Some of these inconsistencies could be attributed to methodological differences, including measures of early life adversities, differing ways of assessment of stress hormones (e.g., daytime salivary versus 12-hour overnight urinary cortisol), and sampling (e.g., clinical versus population-based samples). At the same time, there is strong evidence for the effects of trauma on the atypical secretion of stress hormones. Contrary to the conventional assumption that more stress always leads to higher levels of stress hormones, childhood trauma has been associated with both low and high levels of cortisol in adulthood (Heim, Ehlert, and Hellhammer 2000; Miller, Chen, and Zhou 2007; Yehuda et al. 1993). Similarly, both elevated and reduced levels of epinephrine have been found in adults with a history of extreme or chronic stress (De Bellis et al. 1994; Delahanty et al. 2005; Loevinger et al. 2007). Other investigations suggest that individuals with childhood trauma-related conditions (e.g., posttraumatic

stress disorder and fibromyalgia) may evince an imbalance between stress hormones, such as a higher norepinephrine/epinephrine ratio and a higher norepinephrine/cortisol ratio (Loevinger et al. 2007; Mason et al. 1988).

In sum, individuals who experience extreme or chronic abuse as children are likely to secrete abnormal levels of hormones from the primary stress systems (HPA axis and SNS), which appears to be related to poor biological profiles in other body systems associated with stress physiology (glucose regulation, lipid metabolism, cardiovascular function, immune competence). Yet, few population-based studies of childhood abuse and adult health have attempted to incorporate such biological evidence from the trauma literature. By combining various perspectives from prior work, we will explicitly test the effects of childhood abuse on multiple body systems and the contribution of childhood abuse to the association of social status and physiological dysregulation across body systems.

DATA AND METHODS

Sample

The current study included participants in the Biomarker subsample of the MIDUS study (n = 1,255). MIDUS began in 1995 with a sample of noninstitutionalized, English-speaking residents of the contiguous United States, ages 25 to 78 (N = 7,108). National random-digit dialing was used to select the main sample (n = 3,487) and a sample of twin pairs (n = 1,914). The study also included a random subsample of siblings of individuals in the main sample (n = 950) and oversamples of those living in urban areas using five cities in the United States (n = 757). Between 2004 and 2006, 4,963 of the original respondents completed a follow-up telephone survey and self-assessment questionnaires. At this time, a supplemental sample of 592 African Americans from Milwaukee, Wisconsin, was recruited to enhance the racial diversity of the sample (Radler and Ryff 2010). Respondents in Wave 2 and the Milwaukee samples were invited to participate in the Biomarker study. Around 39% of those eligible for participation were able to participate in a two-day visit to a clinical research center for a physical exam that included a 12-hour urine collection, a fasting blood sample, and measurements of height, weight, waist–hip circumference, and blood pressure. Respondents participating in the Biomarker subsample were more likely to have higher levels of education than those from the full MIDUS II sample. For more details, see Love et al. (2010).

Measures

Social characteristics. We included five social characteristics: *gender* (male = 0, female = 1), *race* (white = 0, nonwhite = 1, with around 90% of nonwhites being black), and three dichotomous measures to capture childhood disadvantage: parental education, poverty, and family instability. We coded *parental education* as 1 if neither parent had a high school degree. *Childhood poverty* was coded as 1 if the respondent's family had ever received welfare or Aid to Families with Dependent Children in childhood. A dichotomous measure of *family instability* was based on the question, "Did you live with both of your biological parents up until you were 16?" Possible explanations for a response of "no" included parents being separated or divorced, parents not being legally married, the death of a parent, and adoption. We also calculated a sum of the three indicators representing *childhood disadvantage*.

Childhood abuse. The current study focused on severe forms of maltreatment, which were measured with 15 items from the Childhood Trauma Questionnaire (CTQ; Bernstein and Fink 1997). The CTQ, which assessed trauma in early life (younger than 18 years of age), included five items within each of several domains, and three of those domains were included in this analysis: (1) *emotional abuse* (called names by family, parents wished was never born, family said hurtful things, felt hated by family, was emotionally abused), (2) *physical abuse* (hit hard enough to see doctor, hit hard enough to leave bruises, punished with hard objects, hit hard enough to be noticed, was physically abused), and (3) *sexual abuse* (touched sexually, hurt if did not do something sexual, made to do sexual things, was molested, was sexually abused). The CTQ also contained a three-item scale for minimization and denial to identify respondents who were more likely to underreport negative events in childhood (e.g., "I had the best family in the world"). Response categories ranged from 1 ("never true") to 5 ("very often true"). Using latent class analysis (LCA), we created five categories of childhood abuse, based on distinct profiles of severity and type of abuse: (1) no abuse, (2) moderate abuse, (3) severe sexual abuse, (4) severe emotional and physical abuse, and (5) severe emotional, physical, and sexual abuse (for details, see the Analytic Strategy and Results sections and Table 1).

Table 1. Item Response Probabilities of Five-class Model of Childhood Abuse, National Survey of Midlife Development in the United States (MIDUS) Biomarker Subsample, 2004–2006 (n = 1,103).

	No Abuse	Moderate Abuse	Severe Abuse		
Variable	Class 1 (68%)	Class 2 (15%)	Class 3 (7%)	Class 4 (5%)	Class 5 (5%)
Emotional abuse					
Called names by family	.03	.56	.20	.75	.64
Parents wished was never born	.01	.17	.03	.56	.55
Family said hurtful things	.02	.81	.26	.83	.94
Felt hated by family	.02	.28	.08	.68	.82
Was emotionally abused	.02	.43	.40	.78	.98
Physical abuse					
Hit hard enough to see doctor	.00	.03	.00	.17	.15
Hit hard to enough to leave bruises	.02	.08	.13	.91	.61
Punished with hard objects	.22	.43	.36	.99	.87
Hit hard enough to be noticed	.00	.01	.00	.31	.23
Was physically abused	.00	.03	.16	.81	.81
Sexual abuse					
Was touched sexually	.01	.03	.92	.02	.95
Hurt if did not do something sexual	.00	.01	.24	.02	.43
Made to do sexual things	.01	.00	.69	.00	.80
Was molested	.00	.01	.73	.00	.91
Was sexually abused	.00	.01	.65	.00	.92

Note: Individual Childhood Trauma Questionnaire (CTQ) items are described with abbreviations. Item response probabilities indicate the probability of responding "sometimes," "often," or "very often" true vs. "never" or "rarely" true to each CTQ item.

Physiological dysregulation. Using 28 biological markers, we constructed summary scores of biological dysregulations. Individual biomarkers representing each domain were selected based on prior studies (e.g., Carroll et al. 2013; Glei et al. 2012; Loevinger et al. 2007; Mason et al. 1988). For the primary stress systems, *HPA-axis activity* included three hormones: urinary cortisol, blood dehydroepiandrosterone (DHEA), and its sulfated form (DHEA-S). *SNS activity* was indexed with the hormones: urinary epinephrine and urinary norepinephrine. For *imbalanced secretion of stress hormones*, we used the ratios of norepinephrine/cortisol and norepinephrine/epinephrine. *Parasympathetic nervous system* (PNS) consisted of four parameters of heart rate variability, including low- and high-frequency spectral power, the standard deviation of R–R (heartbeat to heartbeat) intervals, and the root mean square of successive differences. For secondary physiological systems associated with stress, *proinflammatory activity* included five indices: CRP, fibrinogen, IL-6, the soluble intracellular adhesion molecule-1 (sICAM-1), and endothelial leukocyte adhesion molecule-1 (E-selectin). *Cardiovascular function* included three indicators: resting heartbeat and systolic and diastolic blood pressure. *Lipid metabolism* contained six indices: waist circumference, triglycerides, high-density lipoproteins (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, and total cholesterol/HDL ratio. Finally, for *glucose metabolism*, we used three indices: glycosylated hemoglobin (HbA1c), fasting glucose, and the *homeostasis model assessment* of insulin resistance (HOMA-IR).

To construct summary scores, we defined high-risk cutoffs for biomarkers using previously established clinical cutoffs and, for biomarkers without an established high-risk cutoff, a level above the 80th or below the 20th percentile. Given that both high and low cortisol and epinephrine levels are associated with extreme trauma (De Bellis et al. 1994; Heim et al. 2000; Loevinger et al. 2007), we defined the top and bottom deciles in urinary cortisol and epinephrine as high-risk ranges. For details on the high-risk cutoffs for each biomarker, see Supplementary Table S2 available in the online version of the article. The

summary score for each biological domain reflects the number of biomarkers in the high-risk range (possible range from 0 to 6). As the number of biomarkers was different across the eight domains (ranging from two to six), each domain's potential contribution to a physiological dysregulation score varied. As in prior studies (Gruenewald et al. 2012), we created a weighted summary score (possible range from 0 to 8) by first computing a weighted score for each domain (summary score/the number of biomarkers; possible range from 0 to 1) and then calculating the total of the eight domain scores. We included three control variables: (1) age as a continuous variable after confirming that the linearity assumption was appropriate, (2) sampling status (the Milwaukee subsample), and (3) the site of data collection (West, Midwest, and East Coast).

Analytic Strategy

One fifth of the sample had missing data for at least one variable. The range of missing information for individual variables varied, for example, 3% for race, 2% for childhood abuse, 3% for parental education, and 0.2% to 8.2% for the various biomarkers. Using the *ice* command in Stata (StataCorp 2013), we performed five imputations to predict missing values. We used "multiple imputation, then deletion" by generating imputed values, including dependent variables, and then deleting observations with imputed dependent variables (Von Hippel 2007). After deleting those with information missing for at least one biomarker of interest ($n = 152$), the analytic sample included 1,103 respondents.

We used LCA to identify subgroups of respondents who had similar histories of childhood abuse. Given that most abused children experience multiple types of abuse with varying degrees of severity, latent modeling is beneficial for identifying the underlying heterogeneity of abuse experiences. LCA has been used to identify a set of heterogeneous subgroups based on the patterns of responses to questions about categorical variables (Collins and Lanza 2010). To elicit latent classes, we used 15 items from the CTQ (five items for each type of abuse). To achieve a manageable number of cells and to avoid unacceptably sparse distributions within cells in the latent analysis data matrix, we recoded the five initial response categories of the CTQ items into a dichotomized response for each item (0 = "never" and "rarely true," and 1 = "sometimes," "often," and "very often true"). For the LCA, we added a composite score of the three items of minimization/denial as a control variable to adjust for the tendency among some respondents to exaggerate positive aspects of their childhood or to deny negative experiences (Bernstein et al. 2003). To select the best-fitting model in subsequent comparisons between k and $k + 1$ class solutions, we used the Akaike information criterion (AIC), the Bayesian information criterion (BIC), entropy, and interpretability (Collins and Lanza 2010). To label these profiles, we used both the pattern of item response probabilities produced by LCA and the CTQ cutoff scores recommended in prior studies (Bernstein and Fink 1997).

We reported descriptive statistics (Table 2) and constructed multinomial logistic regression models to simultaneously estimate binary comparisons among the childhood abuse groups (Table 3). We examined whether socially disadvantaged groups were more likely to experience abuse and more severe forms of abuse in childhood. In Tables 4 through 6, we constructed a series of nested models to assess the contribution of childhood abuse to the association between three social characteristics (gender, race, and childhood disadvantage) and physiological dysregulation. Given that only a small percentage of respondents were nonwhites (18% blacks and 3% other nonwhites), and after confirming the blacks were comparable to other nonwhites in terms of biological risk, we collapsed all nonwhite individuals into one group to obtain sufficient statistical power.

In the models (Tables 4 through 6), latent classes of childhood abuse were entered as a series of dummy variables, with the reference group *no abuse*. We used Poisson regression models (count-type outcomes) for each subsystem score and ordinary least squares regression for a weighted summary score for overall stress systems. In Model 1, we assessed the extent to which social status affects dysregulation in biological systems. We then examined the extent to which childhood abuse explains the association between social status and biological profiles (Model 2). The contribution of childhood abuse to the summary score of overall stress systems (Table 6) was evaluated by (1) attenuation in the social status coefficients between Models 1 and 2, and (2) testing the significance of the indirect effects with bootstrapping methods (Hayes 2009). Based on estimates from the final model (Model 2 in Table 6), Figure 1 depicted the average levels for the summary scores of multiple biological domains across the five latent classes of childhood abuse. Because approximately 10% of respondents were from the same family, we corrected intraclass correlation by applying robust standard error estimation. LCA analyses were carried out using Mplus 6.0 and all other analyses using Stata 13.0.

Table 2. Descriptive Statistics for All Analytic Variables, National Survey of Midlife Development in the United States (MIDUS) Biomarker Subsample, 2004–2006 (n = 1,103).

Variable	Mean (Standard Deviation) or %
Social characteristics	
Female	57
White	79
Black	18
Other nonwhites	3
Parental education (< high school degree)	25
Childhood poverty	9
Childhood family instability	25
Childhood disadvantage	
None	56
One	32
Two or three	12
Exposure to childhood abuse	
No abuse	67
Moderate abuse	15
Severe SA	7
Severe EA, PA	5
Severe EA, PA, SA	5
Physiological dysregulation (possible range)	
Primary stress systems	
Hypothalamic-pituitary-adrenal axis activity (0–3)	.57 (.82)
Sympathetic nervous system (0–2)	.37 (.58)
Imbalanced secretion of stress hormones (0–2)	.41 (.62)
Parasympathetic nervous system (0–4)	.79 (1.32)
Secondary stress systems	
Proinflammatory activity (0–5)	1.07 (1.20)
Cardiovascular functioning (0–3)	.55 (.74)
Lipid metabolism (0–6)	1.91 (1.67)
Glucose metabolism (0–3)	.44 (.80)
Weighted summary score of overall stress systems (0–8)	1.64 (1.11)
Control variables	
Age	53.95 (11.49)
Milwaukee subsample	16
Site of data collection	
West Coast	35
Midwest	43
East Coast	22

Note: EA = emotional abuse; PA = physical abuse; SA = sexual abuse. A weighted score was computed as the summary score divided by the number of biomarkers.

RESULTS

Latent Classes of Childhood Abuse and Their Characteristics

The results of LCA revealed that the five-class solution best fit the data. The BIC and AIC showed significant model improvement for the five-class solution (BIC = 7,798 and AIC = 8,214) when compared to the four-class solution (BIC = 8,002 and AIC = 8,332). In the comparisons between the five- and six-class solutions, the AIC supported the six-class solution (AIC = 7,726), but there was no significant improvement in BIC when using the six-class solution (BIC = 8,227). The entropies also supported the five-class over the

Table 3. Multinomial Logistic Models Predicting Exposure to Childhood Abuse, National Survey of Midlife Development in the United States (MIDUS) Biomarker Subsample, 2004–2006 (n = 1,103).

	No Abuse vs. Moderate Abuse		No Abuse vs. Severe SA		No Abuse vs. Severe EA, PA		No Abuse vs. Severe EA, PA, SA	
Variable	RRR	95% CI	RRR	95% CI	RRR	95% CI	RRR	95% CI
Female	1.35	[.94, 1.92]	5.66***	[2.89, 11.08]	1.16	[.64, 2.08]	4.37***	[2.11, 9.06]
Black	1.06	[.28, 3.95]	2.16	[.57, 7.81]	1.87	[.33, 9.08]	.88	[.08, 9.61]
Other race	2.57*	[1.08, 6.11]	2.90	[.95, 8.72]	.96	[.13, 6.49]	1.19	[.22, 6.33]
Childhood disadvantage	1.28	[.97, 1.70]	1.66**	[1.16, 2.36]	1.56†	[.98, 2.49]	3.80***	[2.46, 5.86]

Note: EA = emotional abuse; PA = physical abuse; SA = sexual abuse; 95% CI = 95% confidence interval; RRR = relative risk ratio. Models control for age, the site of data collection, and subsample status.
†$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$.

Table 4. Poisson Regression Estimates of Social Disadvantage and Childhood Abuse on Primary Stress Systems, National Survey of Midlife Development in the United States (MIDUS) Biomarker Subsample, 2004–2006 (n = 1,103).

	Hypothalamic-pituitary-adrenal Axis Activity		Sympathetic Nervous System Activity		Imbalanced Secretion of Stress Hormones		Parasympathetic Nervous System Activity	
Variable	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Female	.496***	.474***	.391***	.390***	.229*	.155	.041	.028
	(.089)	(.091)	(.098)	(.100)	(.097)	(.101)	(.098)	(1.00)
Nonwhite	.055	.068	.005	–.007	.357	.329	–.042	–.032
	(.204)	(.200)	(.224)	(.226)	(.192)	(.192)	(.252)	(.253)
Childhood disadvantage	.081	.048	.143*	.139*	.169*	.093	–.085	–.119
	(.061)	(.065)	(.068)	(.070)	(.069)	(.068)	(.082)	(.084)
Moderate abuse		–.151		.140		.176		.121
		(.127)		(.124)		(.135)		(.146)
Severe SA		.080		–.039		.351*		–.140
		(.140)		(.189)		(.165)		(.217)
Severe EA, PA		.207		.095		.569**		–.034
		(.186)		(.197)		(.176)		(.282)
Severe EA, PA, SA		.374*		.027		.666***		.464
		(.145)		(.193)		(.169)		(.238)

Note: EA = emotional abuse; PA = physical abuse; SA = sexual abuse. Models control for age, the site of data collection, and subsample status.
*$p < .05$, **$p < .01$, ***$p < .001$.

six-class solution (.930 vs. .911). In addition, the six-class model did not reveal an additional, substantively meaningful class, and one of the six classes contained less than 4% of the sample, raising concerns regarding adequate statistical power for conducting subsequent analyses.

Approximately 32% of the respondents experienced some forms of childhood abuse. Among abused individuals, emotional abuse (EA) and physical abuse (PA) were common. There were two profiles of severe sexual abuse (SA): one contained individuals who reported severe SA combined with other severe forms of abuse; the other included individuals who did not report other severe forms of abuse. The five classes were labeled as follows: Class 1 = no abuse (68%), Class 2 = moderate abuse (15%), Class 3 = severe SA (7%), Class 4 = severe EA and PA (5%), and Class 5 = severe EA,

Table 5. Poisson Regression Estimates of Social Disadvantage and Childhood Abuse on Secondary Stress Systems, National Survey of Midlife Development in the United States (MIDUS) Biomarker Subsample, 2004–2006 (n = 1,103).

	Proinflammatory Activity		Cardiovascular Function		Lipid Metabolism		Glucose Metabolism	
Variable	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Female	.194**	.171*	–.080	–.114	–.181**	–.228***	–.241*	–.271*
	(.071)	(.072)	(.082)	(.083)	(.054)	(.056)	(.110)	(.113)
Nonwhite	.262	.252	.207	.170	–.156	–.178	.623**	.614*
	(.138)	(.138)	(.171)	(.171)	(.136)	(.134)	(.231)	(.234)
Childhood disadvantage	.121*	.101*	.144*	.113	.053	.012	.043	.024
	(.048)	(.048)	(.058)	(.059)	(.042)	(.042)	(.081)	(.083)
Moderate abuse		–.014		.356**		.132		–.007
		(.098)		(.102)		(.077)		(.158)
Severe SA		.196		.211		.320**		.207
		(.118)		(.134)		(.095)		(.196)
Severe EA, PA		.384**		.216		.365**		.087
		(.128)		(.178)		(.105)		(.245)
Severe EA, PA, SA		.138		.258		.373**		.199
		(.114)		(.183)		(.111)		(.223)

Note: EA = emotional abuse; PA = physical abuse; SA = sexual abuse. All models control for age, the site of data collection, and subsample status. Robust standard errors are in parentheses.
*p < .05, **p < .01, ***p < .001.

Table 6. Ordinary Least Squares Estimates of Social Disadvantage and Childhood Abuse on Weighted Summary Score, National Survey of Midlife Development in the United States (MIDUS) Biomarker Subsample, 2004–2006 (n = 1,103).

	Overall Stress Systems	
Variable	Model 1	Model 2
Female	.142*	.092
	(.065)	(.066)
Nonwhite	.207	.185
	(.175)	(.174)
Childhood disadvantage	.146**	.092
	(.054)	(.054)
Moderate abuse		.164
		(.087)
Severe SA		.275*
		(.129)
Severe EA, PA		.449*
		(.179)
Severe EA, PA, SA		.581**
		(.167)

Note: EA = emotional abuse; PA = physical abuse; SA = sexual abuse. All models control for age, the site of data collection, and subsample status. Robust standard errors are in parentheses.
*p < .05, **p < .01, ***p < .001.

PA, and SA (5%). The findings for the five classes are summarized in Table 1. To confirm the robustness of these findings, we repeated LCA using the alternative sample without any missing information. The analysis generated results similar to those described above (see Supplementary Table S1 available in the online version of the article).

Descriptive statistics for all variables are shown in Table 2. The average age of respondents was 54 years old, and the majority of the sample was white (78%). A quarter of the respondents reported that neither parent graduated from high school. Around 9% of respondents reported that their family had been on welfare at some point during their childhood, and a quarter of the respondents did not live with both of their biological parents up to age 16.

Social Status and Exposure to Childhood Abuse

Table 3 shows that disadvantaged individuals, women, and individuals who grew in disadvantaged families are at greater risk of experiencing abuse, particularly, severe abuse of multiple types. Results from multinomial logistic models indicated that the risk of experiencing severe SA and all types of severe abuse was more than three times higher

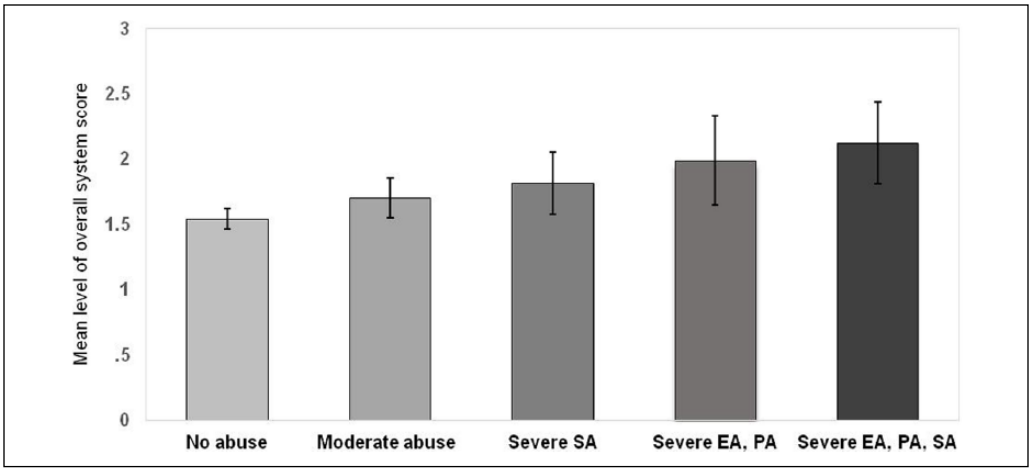

Figure 1. Mean Levels of Overall System Scores by Latent Class of Childhood Abuse, National Survey of Midlife Development in the United States (MIDUS) Biomarker Subsample, 2004–2006 (n = 1,103).
Note: SA = sexual abuse; EA = emotional abuse; PA = physical abuse. Figure 1 is based on Model 2 in Table 6. The estimated values represent the predicted scores of overall physiological dysregulation (weighted) by latent class of childhood abuse. Compared to no abuse, the two severe abuse groups (severe EA, PA and severe EA, PA, and SA) have significantly higher levels of physiological dysregulation.

among women than among men (relative risk ratio [RRR] = 4.37, $p < .001$). Yet, there were no significant gender differences in two other forms of abuse (moderate abuse and severe EA, PA). Disadvantaged individuals were at greater risk for experiencing severe SA (RRR = 1.66, $p < .01$); severe EA, PA (RRR = 1.56, $p = .06$); and all types of severe abuse (RRR = 3.80, $p < .001$).[1]

Social Status, Childhood Abuse, and Physiological Dysregulation

Estimates from Model 1 in Table 4 show biological risk in the primary stress systems differed by social status. Women had higher risk scores for HPA-axis activity ($b = .496$, $p < .001$), SNS activity ($b = .391$, $p < .001$), and imbalanced secretion of stress hormones ($b = .229$, $p < .05$). Individuals from disadvantaged families also had high risk scores in SNS activity ($b = .143$, $p < .05$) and imbalanced stress hormones ($b = .169$, $p < .05$). After including childhood abuse (Model 2), the coefficients of gender and childhood disadvantage on the imbalanced secretion of stress hormones were substantially reduced. Individuals in the severest abuse groups had higher risk score for HPA axis ($b = .374$, $p < .05$) than those in the no-abuse group. There were large differences in the ratios of several stress hormones: those with more severe and multifaceted abuse were more prone to secrete stress hormones in an imbalanced and atypical manner. For SNS activity, there was no significant difference across the groups.

Table 5 shows substantial and significant gender differences in the secondary stress systems, which varied by biological systems: women had a higher inflammatory burden than men ($b = .194$, $p < .01$) but had lower scores for lipid metabolism ($b = –.181$, $p < .01$) and glucose metabolism ($b = –.241$, $p < .05$). These patterns are partially attributed to biological sex differences in immune and metabolic function, with women being at greater risk for inflammatory disorders and men being more prone to cardiovascular disease (Carr 2003; Oertelt-Prigione 2012). Individuals from disadvantaged families were significantly more prone to higher inflammatory burden ($b = .121$, $p < .05$) and greater cardiovascular dysfunction ($b = .144$, $p < .05$). After adjusting for childhood abuse, the gender gap weakened for proinflammatory activity but became stronger for cardiovascular function, lipid metabolism, and glucose metabolism. Similar to findings from the primary stress system, experience of severe abuse was significantly associated with higher risk scores for elevated proinflammatory activity and poor lipid metabolism. There were fewer significant associations for childhood abuse and cardiovascular function and glucose metabolism.

Table 6 portrays the results from the analysis testing whether childhood abuse significantly contributes to the association between social status and

the summary risk score of overall stress systems. From Models 1 and 2, the coefficient of gender was changed substantially from .142 to .092 (35%). Similarly, there were substantial reductions in the coefficient of childhood disadvantage between two models (37%). The results of bootstrapping support statistically significant contribution of childhood abuse. Specifically, childhood abuse significantly linked the two pathways: (1) being female and risk scores of the overall stress system (95% confidence interval [CI] = [.024, .113]) and (2) childhood disadvantage and risk scores of the overall stress system (95% CI = [.033, .107]).

After controlling for social status, the relationships between childhood abuse and physiological dysregulation in overall stress systems were in the predicted direction with a dose-response association: more severe and multiple types of abuse were associated with higher risk scores (Figure 1). Specifically, those with severe abuse of all types had the highest levels of physiological dysregulation (2.12), followed by severe EA and PA (1.99), severe SA (1.82), moderate abuse (1.70), and no abuse (1.54). Post hoc pairwise comparisons of adjusted means show that compared to no abuse, the two severe abuse groups (severe EA, PA and severe EA, PA, and SA) have significantly higher levels of physiological dysregulation.

DISCUSSION

Several key findings emerge from our analyses. First, by using LCA to create distinct profiles of childhood abuse, we found that individuals in lower social positions are at greater risk of experiencing more severe and multiple types of abuse. Women are more likely than men to be exposed to severe SA alone and severe abuse of all types. These findings are consonant with prior studies documenting gender differences in SA (Gilbert et al. 2009), but we showed further that for women, exposure to severe SA may underlie a greater risk of being exposed to other types of severe abuse as well. In addition, there were substantial associations between early life disadvantage and childhood abuse: having poorly educated parents, experiencing poverty, and/or experiencing family instability increased the risk of being severely abused (see Note 1 and Supplementary Table S3 available in the online version of the article). Overall, our findings support an established pattern in sociological research indicating that socially disadvantaged individuals experience not only more stress but also more intense forms of stress compared to the general population (Pearlin et al. 2005; Turner et al. 1995).

Second, childhood abuse plays a significant role in linking social status and biological profiles in adulthood, explaining roughly a third of the effects of gender and childhood disadvantage on the dysregulation of the physiological stress systems we studied. Our analytic approach did not take into account unobserved variables, for example, genetic and environmental factors and other lifestyle risk factors that can vary by gender and social class and influence biological profiles. Thus, the observed associations explained by childhood abuse should be interpreted with caution. Nevertheless, given the statistically significant indirect pathways from social status to dysregulation in physiological systems in midlife *via* childhood abuse, childhood abuse may explain at least part of the gender and childhood disadvantage differences in physiological dysregulation. Overall, our findings indicate that socially disadvantaged individuals are more likely to be exposed to early life trauma, which engenders dysregulation across multiple physiological systems, predisposing for the onset and perpetuation of illness in adulthood.

Third, our analyses incorporated some unique perspectives from the trauma literature, namely, the importance of looking not only for elevated physiological activation but also for atypical secretion patterns of stress hormones, both above and below normal range. By defining both extremes of the distribution of cortisol (top and bottom deciles) as high-risk ranges, we found that individuals who experienced all types of severe abuse exhibited a high-risk score for HPA activity. This significant association did not appear when considering only a conventional risk cutoff on the high end (e.g., a level above the 80th percentile; see Supplementary Table S4, available in the online version of the article). In addition, using two ratios of stress-related hormones, we found that victims of childhood abuse were more likely than nonvictims to exhibit an imbalanced pattern. Our findings are consistent with evidence from trauma-related chronic pain and fatigue conditions, showing that individuals who experience extreme trauma or chronic stress are prone to higher norepinephrine/cortisol and higher norepinephrine/epinephrine ratios, and tend to secrete relatively low levels of epinephrine from the adrenal gland (Loevinger et al. 2007; Mason et al. 1988). The potential health consequences of this imbalance are not well understood, yet they appear to play a role in chronic fatigue and the development of insulin resistance, metabolic syndrome, and

other stress-related physical disorders (Björntorp and Rosmond 2000; Heim et al. 2000; Loevinger et al. 2007). Further studies are needed to more clearly define the parameters of atypical hormone secretion and the extent to which it helps explain the association between childhood trauma and health conditions common in adulthood, including diabetes and cardiovascular disease.

Fourth, consistent with prior work (Carroll et al. 2013; Friedman et al. 2015), we found the effects of childhood abuse remain significant even after controlling for several social characteristics (e.g., gender, race, childhood disadvantage). Both the severity and types of childhood abuse determined the magnitude of the poor biological profile. However, the dose-response association varied by system and fewer significant associations were evident when looking for incremental increases in cardiovascular dysfunction and poor glucose regulation. Given that the use of medications, antihypertensives, are commonly used to treat for these health conditions (Egan, Zhao, and Axon 2010), we created alternative samples by reclassifying individuals taking certain prescription medications as being at risk. However, these analyses did not produce a dose-response association (see Supplementary Table S5, available in the online version of the article).

Such findings may be driven in part by a ceiling effect. That is, a large percentage of participants may have been concentrated at or near high-risk cutoff, which may lead to little variation across the five abuse groups. Specifically, around 35% of respondents were taking antihypertensive mediations, and around 60% of respondents were considered at high risk for poor cardiovascular function (receiving a score of 1+ or taking antihypertensive mediations). A similar tendency was evident when considering the indices of glucose metabolism. Although only 30% of respondents were in the high-risk category, around 60% of those who received a score of 0 in glucose metabolism would be still considered prediabetic (HbA1c value of 5.7% to 6.4%; American Diabetes Association 2010). In addition, we could not exclude explanations related to measurement errors. The observed differences in physiological dysregulation are likely to be a function of various factors, some of which are unobservable (Glei et al. 2013; Goldman et al. 2005). Our findings support using a broader spectrum of biomarkers to better understand the effects of early life trauma on biological profiles in adulthood. Future investigations that use longitudinal biomarker data may be more informative and help reduce some of the noise inherent in cross-sectional data.

Limitations and Future Directions

There are some limitations to the study. First, questions about childhood trauma were asked in midlife, requiring retrospective evaluations of experiences that may have occurred as many as 50 years earlier. Individuals with current mood disorders may be particularly prone to recall bias, exaggerating or misrepresenting adversities experienced during childhood (Widom and Morris 1997). Nonetheless, self-reports are the most common way to document a history of early abuse, and memories of specific childhood experiences are highly stable over time (Yancura and Aldwin 2009). In addition, recollections of traumatic events in childhood tend to be fairly accurate (reviewed in Hardt and Rutter 2004). In supplementary analyses, we confirmed strong reliability between reports of PA measured across the 10-year gap between Waves 1 and 2.

Second, unobserved and unmeasured factors in this study may potentially affect our estimates. Victims with similar experiences of abuse may respond very differently depending on other attributes, such as genetics and environmental background (Danese and McEwen 2012). For example, a family genetic liability may contribute to harsh parenting and disease vulnerability (D. Lee et al. 2013; Walter and Emery 2005). Risk of having elevated levels of physiological dysregulation may also be affected by environmental factors, such as exposure to chemicals, crowded housing, and other adverse neighborhood characteristics (Schulz et al. 2012).

Third, the majority of the African American participants in the Biomarker subsample (85%) were recruited as part of the Milwaukee supplement sample; thus our findings on racial differences cannot be generalized beyond this city-specific sample.

Fourth, nonrandom attrition for victims of childhood abuse (e.g., greater mortality risk of victims between Waves 1 and 2) may create some biased estimates. Yet, if missing data have caused any bias, it is likely to be in a conservative direction (i.e., underestimation).

Despite these limitations, our study contributes to the literature on health inequality by identifying significant links between social disadvantage, severe child abuse, and biological dysregulation in adulthood. More work is needed to further disentangle the pathways to adult risk whereby individuals who experienced severe abuse as children tend to be more vulnerable to subsequent adversities

over the life course. Fortunately, not all individuals who experienced childhood abuse are at greater risk of physiological dysregulation. We observed wide variation in the magnitude of the biological toll, even among victims of severe abuse. Future research should investigate the sources of this heterogeneity in health outcomes and identify factors that foster resilience, buffering against the adverse impact of early life trauma. Finally, our study indicates that employing novel biomarker indices can improve our understanding of early life trauma and its long-term consequences. We encourage social scientists and biomedical researchers to incorporate diverse methods and knowledge from multiple disciplines to advance our understanding of the relationship between the social stress process and dynamics of stress-response physiological systems played out over the life course.

SUPPLEMENTAL MATERIAL

The online Tables S1 through S5 are available in the online version of the article.

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NOTE

1. We found significant associations between each category of early life disadvantage and risk of experiencing childhood abuse. For example, individuals whose parents were poorly educated (relative risk ratio [RRR] = 2.79, $p < .01$), those who were in poverty during childhood (RRR = 3.93, $p < .01$), and those with family instability (RRR = 3.22, $p < .01$) were at greater risk for experiencing all types of severe abuse (see Supplementary Table S3, available in the online version of the article).

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