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Dimensions of Adversity, Physiological Reactivity, and Externalizing Psychopathology in Adolescence: Deprivation and Threat

Daniel S. Busso, Ed.D.[1], **Katie A. McLaughlin, Ph.D.**[2], and **Margaret A. Sheridan, Ph.D.**[3]

[1]Harvard Graduate School of Education

[2]University of Washington, Department of Psychology

[3]University of North Carolina, Chapel Hill

Abstract

Objective—Dysregulation of autonomic nervous system (ANS) and hypothalamic-pituitary-adrenal (HPA) axis function is a putative intermediate phenotype linking childhood adversity (CA) with later psychopathology. However, associations of CAs with ANS and HPA-axis function vary widely across studies. Here, we test a novel conceptual model discriminating between distinct forms of CA (deprivation and threat) and examine their independent associations with physiological reactivity and psychopathology.

Methods—Adolescents (N = 169; mean age = 14.9 years; S.D.=1.4) with a range of interpersonal violence (e.g., maltreatment, community violence) and poverty exposure participated in the Trier Social Stress Test (TSST). During the TSST, electrocardiogram, impedance cardiograph, salivary cortisol and dehydroepiandrosterone-sulphate (DHEA-S) data were collected. We compared the associations of poverty (an indicator of deprivation) and interpersonal violence (an indicator of threat) on sympathetic, parasympathetic, and HPA-axis reactivity to the TSST, and assessed whether these differences mediated the association of adversity with internalizing and externalizing symptoms.

Results—Exposure to poverty and interpersonal violence was associated with psychopathology. Interpersonal violence, adjusting for poverty, was associated with blunted sympathetic (β=1.44, p=.050) and HPA-axis reactivity (β=−.09, p=.021). Blunted cortisol reactivity mediated the association of interpersonal violence with externalizing, but not internalizing, psychopathology. In contrast, poverty was not associated with physiological reactivity after adjusting for interpersonal violence.

Conclusions—We provide evidence for distinct neurobiological mechanisms through which adversity related to poverty and interpersonal violence are associated with psychopathology in adolescence. Distinguishing distinct pathways through which adversity influences mental health has implications for preventive interventions targeting youths exposed to childhood adversity.

Corresponding Author. Daniel S. Busso, Gutman 247, Appian Way, Harvard Graduate School of Education, Cambridge, MA 02138, (857)-756-3759, dab393@mail.harvard.edu.

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Keywords

interpersonal violence; poverty; physiological reactivity; externalizing disorders; psychopathology

Introduction

Childhood adversities (CA) exert a profoundly deleterious impact on development, contributing to population-wide disparities in mental health, educational attainment and economic productivity (1). Nearly 60% of US adolescents report experiencing at least one adversity, including maltreatment, poverty, parental death or divorce (2). Epidemiological and clinical studies indicate that children exposed to CAs are at elevated risk for a wide spectrum of internalizing and externalizing problems, including depression, anxiety, disruptive behavior and substance use disorders (2,3). Consequently, CAs represent salient environmental risk factors that imperil successful adjustment across multiple domains.

The past decade has witnessed a burgeoning interest in how CAs shape neurobiological development, leading to elevated risk for psychopathology (4). In particular, conceptual models propose that prolonged activation of physiological systems following chronic adversity results in a disruption of stress regulatory systems in the body (5). The autonomic nervous system (ANS) and hypothalamic-pituitary-adrenal (HPA) axis operate synergistically to orchestrate physiological responses to environmental stressors, driving long-term biological adaptations necessary for learning and survival (6). The sympathetic (SNS) and parasympathetic (PNS) branches of the ANS play an important role in maintaining the body's homeostatic balance in the face of immediate stressors (i.e. activating the 'fight or flight' response) through changes in cardiovascular tone (7). In contrast, the HPA-axis maintains homeostasis by modulating levels of slower-acting hormones (e.g. cortisol) in the bloodstream (8).

Anomalies in ANS and HPA-axis function have been documented following exposure to CAs spanning maltreatment (9), poverty (10,11), institutionalization (12), parental loss (13) and parental depression (14). However, the interpretation of this literature is complicated by the lack of a clear physiological profile associated with CA exposure. While some studies document dysregulated (hypo- and hyper-responsive) autonomic and neuroendocrine response to psychosocial stressors following adversity (12,15–17), others find discordance between autonomic and neuroendocrine responses (18), or no differences in physiological reactivity at all (19).

These divergent findings may be accounted for, in part, by variability in the types of CA investigated in prior human studies (20–22). To date, studies of CA in humans have typically focused on the effects of single forms of adversity (e.g. maternal education) that are confounded with other unmeasured exposures, such as maltreatment or community violence exposure (23). An alternative approach has been to examine associations between the number of adverse childhood experiences (e.g. 'ACEs' scores) and subsequent endocrine and cardiovascular response to stress (24,25). Neither of these approaches assesses whether specific domains of exposures result in specific patterns of ANS and HPA-axis development.

In the present study, we test a recent theoretical model that predicts variability in neurodevelopment following CA exposure (26,27). The model proposes two orthogonal dimensions of early experience, each having distinct effects on brain and biological systems: (i) *deprivation*, referring to exposures that reflect the absence of expected environmental inputs, and common in the case of neglect, poverty, or institutionalization, and (ii) *threat*, referring to interpersonal violence exposures that involve harm or threat of harm to the child, such as physical and sexual abuse or direct exposure to community violence. Importantly, both types of experience contribute to psychopathology (28), and research attempting to find specificity among types of CA and risk for internalizing and externalizing psychopathology has largely failed (see, e.g., McMahon et al. [29] for a review). However, the model predicts that deprivation and threat have differential impacts on the intervening neurobiological processes that underlie differences in psychopathology.

Specifically, evidence from human and animal studies suggests that threat exposure influences the development of cortico-limbic circuits that underlie fear learning and salience processing (30), thereby modifying physiologic responses to novel stressors (15). In rodents, exposure to threat early in development results in prolonged alteration in amygdala, hippocampal and medial prefrontal cortex (mPFC) function in response to subsequent threat cues (31), as well as hyperreactivity of the HPA-axis (32). The amygdala, hippocampus and medial prefrontal cortex (PFC) modulate behavioral and physiological responses to environmental threats via projections to the hypothalamus and brainstem (33). The amygdala signals the hypothalamus to stimulate the release of corticotrophin releasing hormone (CRH), triggering a cascade of neurochemical events that culminates in the release of cortisol by the adrenal cortex. Perception of environments threats also engages the SNS and PNS by innervating neural fibers in the brain stem (34). Human studies of interpersonal violence exposure mirror findings in animals, revealing that these exposures in childhood are associated with ANS and HPA-axis dysfunction (35,36) and disruption in function and structure of the hippocampus, amygdala, and mPFC (37–39). Given the human and animal evidence, we hypothesize that experiences of threat will be associated with disruptions in physiological reactivity after adjusting for deprivation.

In contrast, we hypothesize that poverty, a proxy for deprivation, is unlikely to result in disrupted physiological reactivity after adjusting for threat. While several studies have demonstrated associations between poverty and physiological reactivity indicators in children and adolescents (11,40–42), these studies did not account for co-occurring exposure to threat, such as interpersonal violence. Children living in poverty are at higher risk for exposure to many forms of interpersonal violence, including maltreatment (43,44), intimate partner violence (45), and community violence (46). Although some studies examining the impact of interpersonal violence exposure on physiological reactivity have controlled for poverty or socioeconomic status (23,36,47), almost no investigations of the impact of poverty on physiological reactivity have controlled for interpersonal violence. The purpose of the current study, then, is to test these hypothesized associations between threat (adjusting for deprivation) and deprivation (adjusting for threat) with autonomic (SNS and PNS) and neuroendocrine (salivary cortisol, dehydroepiandrosterone sulphate; DHEA-S) responses to a laboratory stressor.

Notably, we use the ratio of cortisol to DHEA-S as a measure of HPA-axis function. While cortisol has been extensively studied in the stress literature, DHEA, and its sulphate, DHEA-S, have received considerably less attention (48). Evidence suggests that these steroids have antiglucocorticoid properties, and may therefore counterbalance the effects of cortisol on stress-induced neurotoxicity (49). Accordingly, it has been suggested that the ratio of cortisol to DHEA may represent a better index of neuroendocrine imbalance than cortisol alone (50).

Next, given the robust associations between CA exposure and psychopathology (2), we examined whether effects of CAs on internalizing and externalizing symptoms were mediated by physiological reactivity. A robust research literature suggests that alterations in HPA-axis or ANS function are associated with both internalizing and externalizing difficulties in childhood and adolescence. Youth with internalizing problems generally exhibit elevated cortisol (51) and SNS (52) response to psychosocial stress, whereas externalizing problems are characterized by blunted ANS arousal and HPA-axis reactivity (53,54). Together, these data suggest that both hyper- and hypo-activation of physiological systems are associated with risk for later psychopathology.

Threat exposure was operationalized as exposure to physical abuse, sexual abuse, or direct exposure to community violence. Poverty was operationalized as whether or not a family's income fell above or below the federal poverty line. Although poverty is not a direct measure of deprivation (i.e. it is possible to be poor and still have exposure to enriched cognitive, social and linguistic inputs), low parental SES has been associated with exposure to fewer enriched cognitive experiences in childhood (55) including impoverished linguistic inputs and decreased overall exposure to language (56,57). As such, poverty serves as a proxy for deprivation exposure in this study. Consistent with the deprivation/threat model, we hypothesized that while deprivation and threat would be associated with internalizing and externalizing psychopathology, only threat exposure (adjusting for deprivation) would influence physiological reactivity, and in turn statistically mediate associations with psychopathology.

Methods

Sample

A sample of 169 adolescents (75 males, 94 females) was recruited from schools, after-school programs, medical clinics at Boston Children's Hospital and the wider community in Boston and Cambridge, MA, between July 2010 and November 2012. Recruitment sites were selected to oversample diverse ethnic/racial groups and those with experiences of childhood adversity (i.e. participants were recruited from neighborhoods with high levels of community violence and from clinics that served a predominantly low-SES catchment area). The sample had a mean age of 14.9 years (SD=1.4). The sample was racially diverse: 41.3% White (n=69), 18.0% Black (n=30), 18.0% Hispanic/Latino (n=30), 7.8% Asian (n=13) and 15% Biracial/Other (n=25). All females were postmenarchal. Twenty six percent of participants came from households whose income fell below the federal poverty line (n=42), and approximately one third of the sample (38.3%; n=64) was from single-parent households. Participants were excluded from the study if they were currently using

corticosteroids. Two participants declined to participate in the psychosocial stress task, resulting in an analytic sample of 167 participants. Parents provided informed consent, and adolescents provided assent. All study procedures were approved by the institutional review boards at Boston Children's Hospital and Harvard University.

Procedure

Participants completed the Trier Social Stress Test (TSST), a widely used stress induction procedure used with children and adolescents (58). Participants arrived to the laboratory with their parents, and written consent and assent was obtained from parents and adolescents, respectively. After providing saliva samples, participants completed a five-minute baseline period in which they sat quietly while physiological data (SNS and PNS) were acquired. Then, adolescents completed questionnaire and interview measures assessing adversity exposure and psychopathology. Next, after a second five-minute baseline period, participants completed three tasks in front of two experimenters: a speech preparation period, a five-minute speech to experimenters, and a mental subtraction task (see (36)). Throughout all phases of the TSST (baseline, speech preparation, speech, math), participants were seated and continuous cardiac measures were recorded non-invasively. Saliva samples were acquired at two time points: (1), immediately before the initial resting period, 20–30 minutes after participants had arrived at the lab; and (2), 15 minutes following the beginning of the speech portion of the TSST. Subjects were instructed to refrain from exercising, eating or drinking caffeinated beverages within four hours of their study visit. To account for potential diurnal effects on the HPA-axis, all participants started the laboratory session in the afternoon (between 1pm–4pm).

Autonomic Measures

Electrocardiogram (ECG) recordings were acquired using a Biopac ECG amplifier (Goleta, CA), using a modified Lead II configuration (right clavicle, left lower torso, and right leg ground). Cardiac impedance was obtained using a Bio-Impedance Technology model HIC-2500 impedance cardiograph (Chapel Hill, NC). Electrodes were placed on the neck and torso. ECG and impedance cardiography data were sampled at 1.0kHz and acquired using Biopac MP150 hardware and Acqknowledge software. Data were scored by trained RAs blind to participant identity, and were averaged into 1-minute epochs using Mindware Heart Rate Variability (HRV) software (Mindware Technologies, Gahanna, OH).

Respiratory sinus arrhythmia (RSA) was used as a measure of PNS reactivity and was calculated from the inter-beat interval time series using spectral analysis implemented in Mindware. RSA was calculated for the high-frequency band 0.12–0.40 Hz. To ensure that RSA represents a measure of pure parasympathetic cardiac control, respiration rate was derived from the basal cardiac impedance signal and included as a covariate in all PNS analyses. Greater PNS reactivity is indicated by a task-related decrease in RSA from basal levels (i.e. vagal withdrawal).

Pre-ejection period (PEP) was calculated from impedance cardiography data as a measure of SNS reactivity. PEP represents the time interval beginning with ventricular depolarization and ending when blood is ejected from the left ventricle, where shorter intervals correspond

to greater SNS activation (59). As scoring of impedance cardiography data requires manual placement of the B point (the opening of the aortic valve), these data were independently scored by two raters and differences of more than 5% were adjudicated by one of the study investigators (KM).

Neuroendocrine Measures

Cortisol (nmol/L) and dehydroepiandrosterone-sulphate (DHEA-S; ng/mL) concentrations were collected at three time points during the TSST. Neuroendocrine samples were obtained with cryovial tubes (Immuno-Biological Laboratories) using the drool method, whereby participants expectorate approximately 1.5 ml of saliva into a cryovial with a plastic straw. Saliva samples were stored immediately at −20°C until they were shipped on dry ice to a laboratory in Boston, Massachusetts. Samples were assayed for cortisol and DHEA-S using commercially available luminescence immunoassay kits (CLIA; IBL, Hamburg, Germany). Intra-assay and inter-assay coefficients of variance were acceptable (cortisol: 4.24% and 3.34%; DHEA-S: 3.96% and 4.33%; respectively). HPA-axis activity was calculated by dividing cortisol by DHEA-S for each time point.

Self-Report Measures

Interpersonal violence exposure—was assessed using two self-report questionnaires. First, the Childhood Trauma Questionnaire (CTQ;(60) is a 28-item self-report measure that retrospectively assesses 5 types of negative childhood experiences. Participants respond to each item in the context of “while growing up” on a 5-point Likert scale ranging from “never” to “very often”. Fifteen items corresponding to emotional abuse (e.g. “people in my family said hurtful or insulting things to me”), physical abuse (e.g. “I got hit so hard by someone in my family that I had to see a doctor or go to the hospital”), and sexual abuse (e.g. “someone molested me”) were summed to generate an overall index of childhood abuse (α =.90). We also administered the Screen for Adolescent Violence Exposure (SAVE; (61), a 32-item measure of adolescents’ exposure to direct or indirect violence in school, home or neighborhood contexts. We summed all 12 items corresponding to direct violence exposure (e.g. “someone has pulled a knife on me”), measured on a 5-point Likert scale ranging from “never” to “almost always” (α = .89). Finally, we *z*-transformed and summed the CTQ and SAVE measures (r = .23, p <.001) to create an overall interpersonal violence exposure composite, with higher scores indicating greater exposure to interpersonal violence.

Deprivation—The ratio of income to needs was computed by diving parent-reported family income by the federal poverty thresholds (as determined by the number of people in the household). Our primary measure of deprivation was a dichotomous variable indicating whether participants lived under the federal poverty line (i.e. an income to needs ratio <1). For sensitivity analyses, we also treated deprivation as a continuous measure by taking a log transformation of income-to-needs ratio (62).

Psychopathology—was assessed using the Youth Self-Report form from the Child Behavior Checklist (CBCL(63)). The CBCL is a widely used measure of youth emotional and behavioral problems, and has been population-normed to generate age-standardized

estimates of psychopathology. Here, we use the global externalizing and internalizing subscales of the YSR.

Data Analysis

A log transformation was used to normalize the distribution of cortisol/DHEA-S prior to analysis. Autonomic reactivity to the TSST was modeled by predicting values of PEP and RSA measured during the first minute of the speech and math portions of the TSST, while adjusting for these parameters during the first minute of baseline. Similarly, neuroendocrine reactivity was modeled by predicting cortisol/DHEA following the speech, adjusting for baseline levels. Multiple regression models were conducted with the *sem* package in Stata 13.1 (StataCorp, College Station, TX), using full information maximum likelihood (FIML) to account for occasional missing data due to noise in the ANS waveforms, equipment malfunctions, etc. Less than 8% of the data were missing on any one variable. Following Little (64), data were determined to be missing completely at random.

We used standard tests of mediation (65) to determine whether physiological reactivity mediated the association between our two adversity measures (poverty and interpersonal violence) and psychopathology. First, we tested associations between the predictors (interpersonal violence and poverty) and the outcomes (internalizing and externalizing symptoms) (C path). Interpersonal violence and poverty were entered in separate regression models, and then included together. Next, we tested the association between the predictors and the proposed mediators (PEP, RSA and cortisol/DHEA during the speech and math portions of the TSST) (A path). As above, interpersonal violence and poverty were entered separately and then together. Finally, we tested associations between the mediators and the outcomes (B path). If these criteria were met for a single path, the significance of the indirect path (through the mediator) was tested using a bootstrapping approach (66). This approach generates bias-corrected, bootstrapped confidence intervals for total and specific indirect effects of the predictors, on the outcome, through the mediators. Confidence intervals that do not include zero indicate statistically significant mediation. Age and gender were included as covariates in all models, and respiration rate was included as a covariate for all models using RSA. Unstandardized betas are presented in the results.

Results

Physiological Reactivity

We first examined task-related changes in physiological reactivity from baseline using paired samples *t*-tests. Significant increases in SNS activity (i.e. smaller PEP than baseline) were observed for 90% of participants for the speech ($t = 13.78$; $p < .001$) and 82% of participants for the math ($t = 11.52$; $p < .001$) portions of the TSST. Similarly, vagal suppression (i.e. smaller RSA than baseline) was observed for 70% of participants for speech ($t = 6.29$; $p < .001$) and 61% of participants for math ($t = 4.25$; $p < .001$). Finally, increased HPA-axis reactivity (i.e. greater cortisol:DHEA-S than baseline) occurred in 78% of participants ($t = 7.06$; $p < .001$).

Adversity Exposure and Psychopathology

Table 1 displays means and standard deviations for key study variables. Bivariate correlations are presented in Table 2. Following examination of the uncontrolled, bivariate associations, we ran multiple regression models testing the association between CAs (poverty and violence exposure) and internalizing and externalizing psychopathology. Poverty was associated with externalizing (β=4.74, p=.005), but not internalizing symptoms (β=2.40, p=.18). Children whose household income fell below the federal poverty line exhibited greater levels of externalizing symptoms than those above the poverty line. This pattern of results did not change when adjusting for violence exposure. Exposure to violence was significantly associated with both externalizing (β=3.06, p<.001) and internalizing symptoms (β=2.87, p<.001), which did not change when adjusting for poverty. Children with greater exposure to violence had higher levels of psychopathology.

Adversity Exposure and Physiological Reactivity

Figure 1 displays SNS, PNS and HPA-axis reactivity to the TSST, by poverty and interpersonal violence exposure. Analysis of baseline physiological data revealed that poverty was associated with significantly higher baseline HPA-axis activity (β=.60, p=.013) and marginally higher baseline PNS activity (β=.46, p=.065). In contrast, no associations were observed between poverty and baseline SNS activity, or between interpersonal violence and any physiological measure.

Next, we examined associations between adversity exposure and physiological reactivity. Table 3 displays coefficients, standard errors, and significance values for hierarchical linear regression models predicting physiological reactivity (RSA, PEP, cortisol:DHEA-S) from poverty and interpersonal violence. Neither violence nor poverty predicted PNS reactivity to speech or math. Exposure to violence was associated with blunted SNS reactivity to the speech and math portions of the TSST. Findings did not change when adjusting for poverty. In contrast, poverty was unassociated with SNS reactivity, with or without adjusting for violence. Finally, interpersonal violence was associated with blunted HPA-axis response to the TSST, even when accounting for poverty. In contrast, poverty was unrelated to HPA-axis reactivity, even when accounting for interpersonal violence.

Physiological Reactivity and Psychopathology

Next, we tested the hypothesis that physiological reactivity would be associated with symptoms of psychopathology. Symptoms of externalizing psychopathology were associated with blunted physiological reactivity. Blunted SNS and PNS reactivity to math (SNS: β=.15, p=.017; PNS: β=1.14, p=.050) and decreased HPA-axis activation following the TSST (β=−2.55, p=.008) were associated with greater externalizing symptoms. Neither SNS, PNS, nor HPA-axis reactivity were associated with internalizing symptoms.

Mediation Analysis

Finally, we ran separate mediation models to test whether physiological reactivity to the TSST mediated the association of violence exposure with internalizing and externalizing symptoms. Adjusting for poverty, a significant indirect effect of violence exposure through

HPA-axis reactivity was observed (N=156; 95% CI: .03, .45) (Figure 2). We found no significant indirect effect of violence through SNS reactivity to speech or to math.

Sensitivity Analysis

We conducted sensitivity analyses to determine whether our dichotomous operationalization of poverty explained our findings. The pattern of findings was identical when using income-to-needs ratio as a continuous measure.

Discussion

The present study provides evidence for distinct effects of deprivation and threat on ANS and HPA-axis development. Disturbances in autonomic and neuroendocrine system function are a putative mechanism underlying the association between CA and psychopathology (8,67). However, findings from previous studies have been highly mixed, suggesting the need to disentangle the relative impacts of different forms of CA.

We extend previous research in a number of ways. First, we tested a novel theoretical model that differentiates between experiences of deprivation and threat in shaping neurobiological development (26,27). Consistent with this model, our data support the hypothesis that while both forms of CA—reflected in poverty and interpersonal violence exposure—are associated with higher levels of psychopathology, only threat is associated with differences in physiological reactivity. Specifically, we found that threat was associated with blunted SNS and cortisol reactivity to the TSST, and that HPA-axis reactivity mediated the association between threat and externalizing psychopathology. In contrast, we found no association between threat and PNS reactivity, or any associations between physiological reactivity and internalizing symptoms. These findings are consistent with previous studies reporting blunted physiological reactivity to stress in individuals with exposure to interpersonal violence, such as maltreatment (15,17,23) and with those reporting blunted physiological reactivity in children with externalizing disorders (53,54). Blunted cortisol reactivity could reflect down-regulation of corticotropin- releasing hormone (CRH) receptors in the pituitary gland following iterative exposure to interpersonal violence. In turn, this pattern of reduced reactivity to environmental stressors may contribute to externalizing pathology by chronically reducing arousal levels and increasing sensation seeking (68). Notably, despite finding differences in baseline HPA-axis activity among poverty-exposed adolescents, there was no association between poverty and physiological reactivity, with or without adjusting for interpersonal violence. This departs from a number of prior studies of poverty-exposed youth (11,69). These findings suggest that studies examining poverty and physiological reactivity to stress should assess and control for exposure to violence, which may be a critical confounder.

Second, our findings show that interpersonal violence was associated with blunted SNS reactivity to the TSST. To date, surprisingly few prior studies have explored the impact of adversity exposure on ANS reactivity, and these have often used nonspecific measures such as heart rate (15,70). Our use of pre-ejection period is advantageous as it represents a measure of ‘pure’ sympathetic response. The importance of differentiating between contributions of the SNS and PNS is underscored by our finding that only SNS indicators

are associated with exposure to threat. In contrast, in previous studies we (and others) have found that PNS reactivity to psychosocial stress functions as a *moderator,* such that risk of internalizing psychopathology following exposure to CAs varies as a function of PNS activity (71,72). Taken together, these results suggest that measures of ANS activity, particularly of the SNS, may be used as a clinically useful index of stress sensitivity following exposure to violence in childhood alongside commonly used HPA-axis measures such as cortisol.

The findings from this study should be viewed in light of several limitations. First, our data are cross-sectional, precluding us from determining the directional and transactional pathways linking CA exposure, physiological reactivity and externalizing psychopathology. Our mediation analyses assume a temporal ordering of relationships, but it is possible that externalizing symptoms precede rather than follow from blunted physiological reactivity to stress. It is plausible that youths with externalizing problems may put themselves at increased risk for exposure to interpersonal violence (e.g. by engaging in community violence). Future prospective studies are needed to explore bidirectional associations between these variables. Second, our assessment of psychopathology used a self-report questionnaire. Replication of these findings using a structured clinical interview is therefore warranted. Third, our model focuses on only two aspects of early experience, and there are likely to be numerous others. For example, other characteristics of adversity, including the developmental timing and chronicity of exposure, have been conceptualized as important components of CA that explain differences in neuroendocrine activity and psychopathology in later life (73–75). Further studies are needed to examine the impact of additional dimensions of adversity on neurobiological development and mental health. Fourth, we did not collect information on females' use of oral contraceptives, which may have affected analyses involving HPA-axis reactivity. However, given the age of the sample, we find this to be unlikely. Finally, our study design did not allow us to examine moderators of the association between childhood adversity and psychopathology. An enhanced understanding of factors that may buffer individuals from early adversity is another important goal for future research.

Conclusions

We provide evidence for differential influences of threat and deprivation on physiological reactivity to stress. Although both deprivation and threat exposure were associated with greater levels of externalizing symptoms, only threat exposure was associated with differences in physiological reactivity. Blunted physiological reactivity, in turn, mediated only the association of threat with externalizing problems. These findings provide preliminary support for the deprivation/threat model as a useful theoretical framework through which to understand the association of childhood adversity with neurobiological development. Moreover, these findings highlight the importance of distinguishing between different forms of adversity. Such an approach may eventually yield information that can be used to targeting intervention approaches based on disorder etiology, in addition to symptomatology.

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Abbreviations

CA	childhood adversity
DHEA-S	dehydroepiandrosterone sulphate
ECG	electrocardiogram
HPA	hypothalamic-pituitary-adrenal
SNS	sympathetic nervous system
PNS	parasympathetic nervous system
TSST	Trier Social Stress Test
PEP	pre-ejection period

References

1. Shonkoff JP, Garner AS, Siegel BS, Dobbins MI, Earls MF, Garner AS, McGuinn L, Pascoe J, Wood DL. The Lifelong Effects of Early Childhood Adversity and Toxic Stress. Pediatrics. 2012 Jan 1; 129(1):e232–e246. [PubMed: 22201156]
2. McLaughlin KA, Green JG, Gruber MJ, Zaslavsky AM, Kessler RC. Childhood adversities and first onset of psychiatric disorders in a national sample of us adolescents. Arch Gen Psychiatry. 2012 Nov 1; 69(11):1151–1160. [PubMed: 23117636]
3. Kessler RC, Avenevoli S, Ries Merikangas K. Mood disorders in children and adolescents: an epidemiologic perspective. Biol Psychiatry. 2001; 49(12):1002–1014. [PubMed: 11430842]
4. Teicher MH, Andersen SL, Polcari A, Anderson CM, Navalta CP, Kim DM. The neurobiological consequences of early stress and childhood maltreatment. Neurosci Biobehav Rev. 2003 Mar; 27(1–2):33–44. [PubMed: 12732221]
5. McEwen BS. Brain on stress: How the social environment gets under the skin. Proc Natl Acad Sci U S A. 2012 Oct 16; 109(Suppl 2):17180–17185. [PubMed: 23045648]
6. Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. Psychol Bull. 2004 May; 130(3):355–391. [PubMed: 15122924]
7. Sapolsky RM, Romero LM, Munck AU. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocr Rev. 2000 Feb; 21(1):55–89. [PubMed: 10696570]
8. Gunnar MR, Quevedo K. The neurobiology of stress and development. Annu Rev Psychol. 2007; 58:145–173. [PubMed: 16903808]
9. Cicchetti D, Rogosch FA. Diverse patterns of neuroendocrine activity in maltreated children. Dev Psychopathol. 2001; 13(3):677–693. [PubMed: 11523854]
10. Evans GW. A multimethodological analysis of cumulative risk and allostatic load among rural children. Dev Psychol. 2003 Sep; 39(5):924–933. [PubMed: 12952404]
11. Evans GW, Kim P. Childhood Poverty and Health Cumulative Risk Exposure and Stress Dysregulation. Psychol Sci. 2007 Nov 1; 18(11):953–957. [PubMed: 17958708]
12. McLaughlin KA, Sheridan MA, Tibu F, Fox NA, Zeanah CH, Nelson CA. Causal effects of the early caregiving environment on development of stress response systems in children. Proc Natl Acad Sci. 2015 Apr 20. 201423363.

13. Tyrka AR, Wier L, Price LH, Ross N, Anderson GM, Wilkinson CW, Carpenter LL. Childhood Parental Loss and Adult Hypothalamic-Pituitary-Adrenal Function. Biol Psychiatry. 2008 Jun 15; 63(12):1147–1154. [PubMed: 18339361]

14. Halligan SL, Herbert J, Goodyer IM, Murray L. Exposure to postnatal depression predicts elevated cortisol in adolescent offspring. Biol Psychiatry. 2004 Feb 15; 55(4):376–381. [PubMed: 14960290]

15. Heim C, Newport DJ, Heit S, Graham YP, Wilcox M, Bonsall R, Miller AH, Nemeroff CB. Pituitary-adrenal and autonomic responses to stress in women after sexual and physical abuse in childhood. JAMA. 2000 Aug 2; 284(5):592–597. [PubMed: 10918705]

16. Kaufman J, Birmaher B, Perel J, Dahl RE, Moreci P, Nelson B, Wells W, Ryan ND. The corticotropin-releasing hormone challenge in depressed abused, depressed nonabused, and normal control children. Biol Psychiatry. 1997 Oct 15; 42(8):669–679. [PubMed: 9325560]

17. Gunnar MR, Frenn K, Wewerka SS, Van Ryzin MJ. Moderate versus severe early life stress: Associations with stress reactivity and regulation in 10–12-year-old children. Psychoneuroendocrinology. 2009 Jan; 34(1):62–75. [PubMed: 18835102]

18. Gordis EB, Granger DA, Susman EJ, Trickett PK. Asymmetry between salivary cortisol and alpha-amylase reactivity to stress: relation to aggressive behavior in adolescents. Psychoneuroendocrinology. 2006 Sep; 31(8):976–987. [PubMed: 16879926]

19. DeSantis SM, Baker NL, Back SE, Spratt E, Ciolino JD, Moran-Santa Maria M, Dipanker B, Brady KT. Gender differences in the effect of early life trauma on hypothalamic-pituitary-adrenal axis functioning. Depress Anxiety. 2011 May; 28(5):383–392. [PubMed: 21328636]

20. Humphreys KL, Zeanah CH. Deviations from the expectable environment in early childhood and emerging psychopathology. Neuropsychopharmacol Off Publ Am Coll Neuropsychopharmacol. 2015 Jan; 40(1):154–170.

21. Kuhlman KR, Geiss EG, Vargas I, Lopez-Duran NL. Differential associations between childhood trauma subtypes and adolescent HPA-axis functioning. Psychoneuroendocrinology. 2015 Apr. 54:103–114. [PubMed: 25704913]

22. Miller GE, Chen E, Zhou ES. If it goes up, must it come down? Chronic stress and the hypothalamic-pituitary-adrenocortical axis in humans. Psychol Bull. 2007 Jan; 133(1):25–45. [PubMed: 17201569]

23. MacMillan HL, Georgiades K, Duku EK, Shea A, Steiner M, Niec A, Tanaka M, Gensey S, Spree S, Vella E, Walsh CA, De Bellis MD, Van der Meulen J, Boyle MH, Schmidt LA. Cortisol Response to Stress in Female Youths Exposed to Childhood Maltreatment: Results of the Youth Mood Project. Biol Psychiatry. 2009 Jul 1; 66(1):62–68. [PubMed: 19217075]

24. Lovallo WR, Farag NH, Sorocco KH, Cohoon AJ, Vincent AS. Lifetime Adversity Leads to Blunted Stress Axis Reactivity: Studies from the Oklahoma Family Health Patterns Project. Biol Psychiatry. 2012 Feb 15; 71(4):344–349. [PubMed: 22112928]

25. Voellmin A, Winzeler K, Hug E, Wilhelm FH, Schaefer V, Gaab J, La Marca R, Pruessner JC, Bader K. Blunted endocrine and cardiovascular reactivity in young healthy women reporting a history of childhood adversity. Psychoneuroendocrinology. 2015 Jan.51:58–67. [PubMed: 25290347]

26. McLaughlin KA, Sheridan MA, Lambert HK. Childhood adversity and neural development: Deprivation and threat as distinct dimensions of early experience. Neurosci Biobehav Rev. 2014 Nov.47:578–591. [PubMed: 25454359]

27. Sheridan MA, McLaughlin KA. Dimensions of early experience and neural development: deprivation and threat. Trends Cogn Sci. 2014 Nov; 18(11):580–585. [PubMed: 25305194]

28. Nelson, C., Sheridan, M. Lessons from Neuroscience Research for Understanding Causal Links between Family and Neighborhood Characteristics and Educational Outcomes. In: Duncan, G., Murnane, R., editors. Whither Opportunity: Rethinking the Role of Neighborhoods and Families on Schools and School Outcomes for American Children. New York: Russell Sage Foundation Press; 2011.

29. McMahon SD, Grant KE, Compas BE, Thurm AE, Ey S. Stress and psychopathology in children and adolescents: is there evidence of specificity? J Child Psychol Psychiatry. 2003 Jan 1; 44(1): 107–133. [PubMed: 12553415]

30. McCrory EJ, De Brito SA, Sebastian CL, Mechelli A, Bird G, Kelly PA, Viding E. Heightened neural reactivity to threat in child victims of family violence. Curr Biol. 2011 Dec 6; 21(23):R947–R948. [PubMed: 22153160]
31. Chen Y, Fenoglio KA, Dubé CM, Grigoriadis DE, Baram TZ. Cellular and molecular mechanisms of hippocampal activation by acute stress are age-dependent. Mol Psychiatry. 2006 Nov; 11(11): 992–1002. [PubMed: 16801951]
32. Liu D, Diorio J, Tannenbaum B, Caldji C, Francis D, Freedman A, Sharma S, Pearson D, Plotsky PM, Meaney MJ. Maternal care, hippocampal glucocorticoid receptors, and hypothalamic-pituitary-adrenal responses to stress. Science. 1997 Sep 12; 277(5332):1659–1662. [PubMed: 9287218]
33. Chiang JJ, Taylor SE, Bower JE. Early adversity, neural development, and inflammation. Dev Psychobiol. 2015 Sep 15.
34. Thayer JF, Åhs F, Fredrikson M, Sollers JJ III, Wager TD. A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. Neurosci Biobehav Rev. 2012 Feb; 36(2):747–756. [PubMed: 22178086]
35. Tarullo AR, Gunnar MR. Child maltreatment and the developing HPA axis. Horm Behav. 2006 Nov; 50(4):632–639. [PubMed: 16876168]
36. McLaughlin KA, Sheridan MA, Alves S, Mendes WB. Child maltreatment and autonomic nervous system reactivity: identifying dysregulated stress reactivity patterns by using the biopsychosocial model of challenge and threat. Psychosom Med. 2014 Sep; 76(7):538–546. [PubMed: 25170753]
37. McLaughlin KA, Peverill M, Gold AL, Alves S, Sheridan MA. Child Maltreatment and Neural Systems Underlying Emotion Regulation. J Am Acad Child Adolesc Psychiatry. 2015 Sep; 54(9): 753–762. [PubMed: 26299297]
38. Gorka AX, Hanson JL, Radtke SR, Hariri AR. Reduced hippocampal and medial prefrontal gray matter mediate the association between reported childhood maltreatment and trait anxiety in adulthood and predict sensitivity to future life stress. Biol Mood Anxiety Disord. 2014; 4:12. [PubMed: 25408863]
39. Herringa RJ, Birn RM, Ruttle PL, Burghy CA, Stodola DE, Davidson RJ, Essex MJ. Childhood maltreatment is associated with altered fear circuitry and increased internalizing symptoms by late adolescence. Proc Natl Acad Sci U S A. 2013 Nov 19; 110(47):19119–19124. [PubMed: 24191026]
40. Lupien SJ, King S, Meaney MJ, McEwen BS. Child's stress hormone levels correlate with mother's socioeconomic status and depressive state. Biol Psychiatry. 2000 Nov 15; 48(10):976–980. [PubMed: 11082471]
41. Blair C, Granger DA, Willoughby M, Mills-Koonce R, Cox M, Greenberg MT, Kiblighan KT, Fortunato CK. Salivary Cortisol Mediates Effects of Poverty and Parenting on Executive Functions in Early Childhood. Child Dev. 2011; 82(6):1970–1984. [PubMed: 22026915]
42. Evans GW, Schamberg MA. Childhood poverty, chronic stress, and adult working memory. Proc Natl Acad Sci. 2009 Apr 21; 106(16):6545–6549. [PubMed: 19332779]
43. Trickett PK, Lawrence J, Carlson V, Cicchetti D. Relationship of socioeconomic status to the etiology and developmental sequelae of physical child abuse. Dev Psychol. 1991; 27(1):148–158.
44. Coulton CJ, Crampton DS, Irwin M, Spilsbury JC, Korbin JE. How neighborhoods influence child maltreatment: A review of the literature and alternative pathways. Child Abuse Negl. 2007 Nov; 31(11–12):1117–1142. [PubMed: 18023868]
45. Cunradi CB, Caetano R, Clark C, Schafer J. Neighborhood Poverty as a Predictor of Intimate Partner Violence Among White, Black, and Hispanic Couples in the United States: A Multilevel Analysis. Ann Epidemiol. 2000 Jul; 10(5):297–308. [PubMed: 10942878]
46. Buka SL, Stichick TL, Birdthistle I, Earls FJ. Youth exposure to violence: prevalence, risks, and consequences. Am J Orthopsychiatry. 2001 Jul; 71(3):298–310. [PubMed: 11495332]
47. Carpenter LL, Shattuck TT, Tyrka AR, Geracioti TD, Price LH. Effect of childhood physical abuse on cortisol stress response. Psychopharmacology (Berl). 2010 Sep 14; 214(1):367–375. [PubMed: 20838776]

48. Van Voorhees EE, Dennis MF, Calhoun PS, Beckham JC. Association of DHEA, DHEAS, and cortisol with childhood trauma exposure and posttraumatic stress disorder. Int Clin Psychopharmacol. 2014 Jan; 29(1):56–62. [PubMed: 23907073]

49. Kimonides VG, Spillantini MG, Sofroniew MV, Fawcett JW, Herbert J. Dehydroepiandrosterone antagonizes the neurotoxic effects of corticosterone and translocation of stress-activated protein kinase 3 in hippocampal primary cultures. Neuroscience. 1999 Mar; 89(2):429–436. [PubMed: 10077325]

50. Goodyer IM, Park RJ, Netherton CM, Herbert J. Possible role of cortisol and dehydroepiandrosterone in human development and psychopathology. Br J Psychiatry. 2001 Sep 1; 179(3):243–249. [PubMed: 11532802]

51. Goodyer IM, Park RJ, Herbert J. Psychosocial and endocrine features of chronic first-episode major depression in 8–16 year olds. Biol Psychiatry. 2001 Sep 1; 50(5):351–357. [PubMed: 11543738]

52. Nock MK, Mendes WB. Physiological arousal, distress tolerance, and social problem-solving deficits among adolescent self-injurers. J Consult Clin Psychol. 2008 Feb; 76(1):28–38. [PubMed: 18229980]

53. Beauchaine TP, Gatzke-Kopp L, Neuhaus E, Chipman J, Reid MJ, Webster-Stratton C. Sympathetic- and parasympathetic-linked cardiac function and prediction of externalizing behavior, emotion regulation, and prosocial behavior among preschoolers treated for ADHD. J Consult Clin Psychol. 2013 Jun; 81(3):481–493. [PubMed: 23544677]

54. Beauchaine TP, Gatzke-Kopp L, Mead HK. Polyvagal Theory and Developmental Psychopathology: Emotion Dysregulation and Conduct Problems from Preschool to Adolescence. Biol Psychol. 2007 Feb; 74(2):174–184. [PubMed: 17045726]

55. Bradley RH, Corwyn RF, McAdoo HP, Coll CG. The home environments of children in the United States part I: variations by age, ethnicity, and poverty status. Child Dev. 2001 Dec; 72(6):1844–1867. [PubMed: 11768149]

56. Fernald A, Marchman VA, Weisleder A. SES differences in language processing skill and vocabulary are evident at 18 months. Dev Sci. 2013 Mar; 16(2):234–248. [PubMed: 23432833]

57. Hart, B., Risley, TR. Meaningful Differences in the Everyday Experience of Young American Children. Baltimore, MD: Paul H. Brookes Publishing; 1995. p. 268

58. Kudielka, BM., Hellhammer, DH., Kirschbaum, C. Ten years of research with the Trier Social Stress Test-Revisited. In: Harmon-Jones, E., Winkielman, P., editors. Social neuroscience: Integrating biological and psychological explanations of social behavior. New York, NY: Guilford Press; 2007. p. 56-83.

59. Sherwood A, Allen MT, Fahrenberg J, Kelsey RM, Lovallo WR, van Doornen LJP. Methodological Guidelines for Impedance Cardiography. Psychophysiology. 1990 Jan 1; 27(1):1–23. [PubMed: 2187214]

60. Bernstein DP, Ahluvalia T, Pogge D, Handelsman L. Validity of the Childhood Trauma Questionnaire in an Adolescent Psychiatric Population. J Am Acad Child Adolesc Psychiatry. 1997 Mar; 36(3):340–348. [PubMed: 9055514]

61. Hastings TL, Kelley ML. Development and validation of the Screen for Adolescent Violence Exposure (SAVE). J Abnorm Child Psychol. 1997 Dec; 25(6):511–520. [PubMed: 9468111]

62. Noble KG, Houston SM, Brito NH, Bartsch H, Kan E, Kuperman JM, Akshoomoff N, Amaral DG, Bloss CS, Libiger O, Schnork NJ, Murray SS, Casey BJ, Chang L, Ernst TM, Frazier JA, Gruen JR, Kennedy DN, Van Zijl P, Mostofsky S, Kaufmann WR, Kenet T, Dale AM, Jernigan TL, Sowell ER. Family income, parental education and brain structure in children and adolescents. Nat Neurosci. 2015 May; 18(5):773–778. [PubMed: 25821911]

63. Achenbach, TM. Integrative Guide for the 1991 CBCL/4–18, YSR, and TRF Profiles. Burlington, VT: Department of Psychiatry, University of Vermont; 1991. p. 211

64. Little RJA. A Test of Missing Completely at Random for Multivariate Data with Missing Values. J Am Stat Assoc. 1988 Dec 1; 83(404):1198–1202.

65. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: conceptual, strategic, and statistical considerations. J Pers Soc Psychol. 1986 Dec; 51(6): 1173–1182. [PubMed: 3806354]

66. Preacher KJ, Hayes AF. SPSS and SAS procedures for estimating indirect effects in simple mediation models. Behav Res Methods Instrum Comput. 2004 Nov 1; 36(4):717–731. [PubMed: 15641418]

67. Lupien SJ, McEwen BS, Gunnar MR, Heim C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. Nat Rev Neurosci. 2009 Jun; 10(6):434–445. [PubMed: 19401723]

68. van Goozen SHM, Fairchild G, Snoek H, Harold GT. The evidence for a neurobiological model of childhood antisocial behavior. Psychol Bull. 2007 Jan; 133(1):149–182. [PubMed: 17201574]

69. Rudolph KE, Gary SW, Stuart EA, Glass TA, Marques AH, Duncko R, Merikangas KR. The association between cortisol and neighborhood disadvantage in a U.S. population-based sample of adolescents. Health Place. 2014 Jan.25:68–77. [PubMed: 24367996]

70. Orr SP, Lasko NB, Metzger LJ, Berry NJ, Ahern CE, Pitman RK. Psychophysiologic assessment of women with posttraumatic stress disorder resulting from childhood sexual abuse. J Consult Clin Psychol. 1998 Dec; 66(6):906–913. [PubMed: 9874903]

71. McLaughlin KA, Rith-Najarian L, Dirks MA, Sheridan MA. Low Vagal Tone Magnifies the Association Between Psychosocial Stress Exposure and Internalizing Psychopathology in Adolescents. J Clin Child Adolesc Psychol. 2013; 0(0):1–15.

72. McLaughlin KA, Alves S, Sheridan MA. Vagal regulation and internalizing psychopathology among adolescents exposed to childhood adversity. Dev Psychobiol. 2014 Jul 1; 56(5):1036–1051. [PubMed: 24338154]

73. Bosch NM, Riese H, Reijneveld SA, Bakker MP, Verhulst FC, Ormel J, Oldehinkel AJ. Timing matters: Long term effects of adversities from prenatal period up to adolescence on adolescents' cortisol stress response. The TRAILS study. Psychoneuroendocrinology. 2012 Sep; 37(9):1439–1447. [PubMed: 22365483]

74. Fries E, Hesse J, Hellhammer J, Hellhammer DH. A new view on hypocortisolism. Psychoneuroendocrinology. 2005 Nov 1; 30(10):1010–1016. [PubMed: 15950390]

75. Jonson-Reid M, Kohl PL, Drake B. Child and Adult Outcomes of Chronic Child Maltreatment. Pediatrics. 2012 May 1; 129(5):839–845. [PubMed: 22529281]

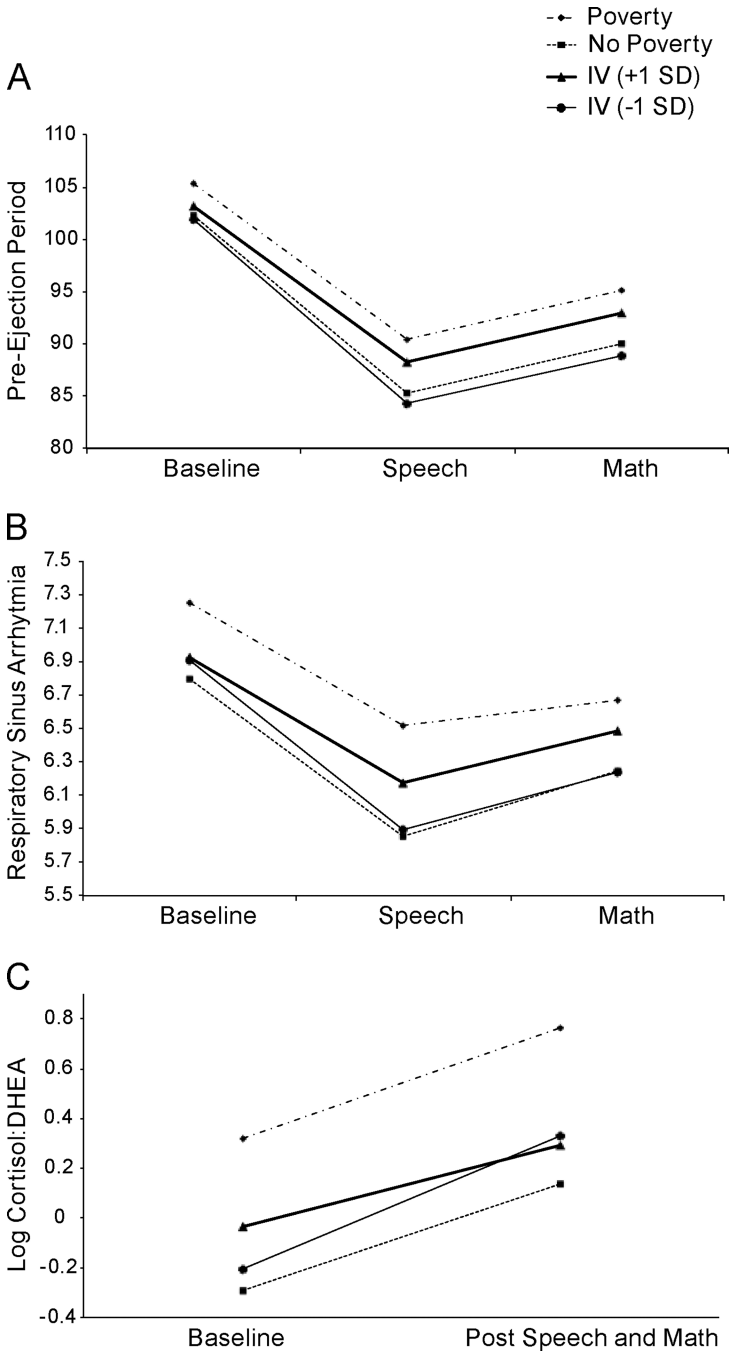

Figure 1.

Physiological reactivity to the TSST, by exposure to poverty and interpersonal violence. Panels display SNS (top), PNS (middle) and HPA-axis (bottom) reactivity. Interpersonal violence (IV) is shown at +1 and −1 standard deviations from the mean. Data are unadjusted for covariates.

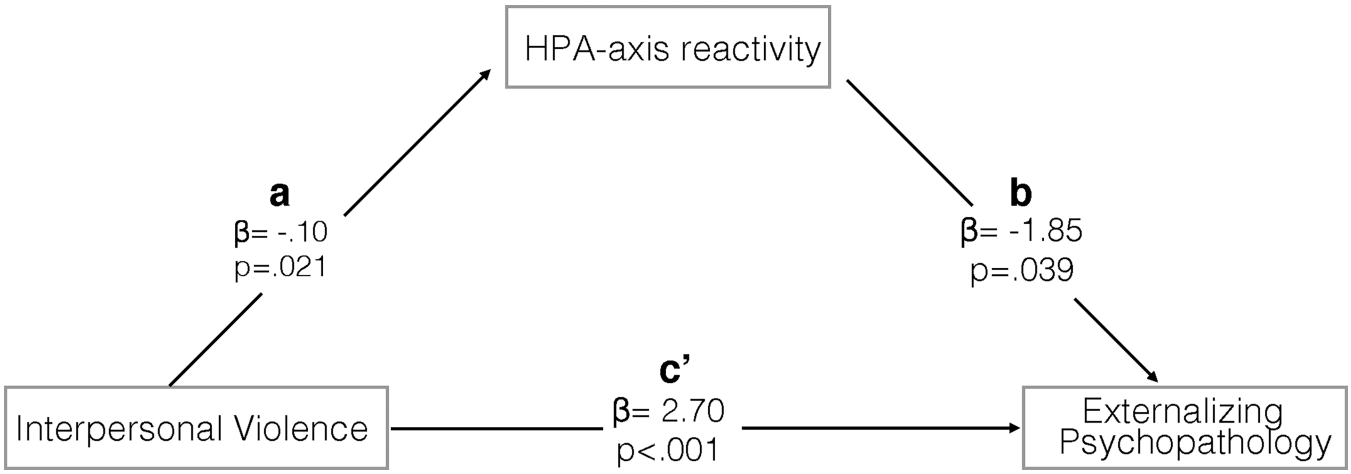

Figure 2.

HPA-axis reactivity mediates the association between interpersonal violence exposure and externalizing psychopathology. The significance of the indirect effect was tested using a bootstrapping approach, and analyses adjust for age, sex, poverty, and HPA-axis activity at baseline. Note: c' = direct effect.

Table 1

Distribution for Childhood Adversity, Physiological Reactivity and Psychopathology Variables (N=167)

	Mean	SD
SAVE Traumatic Violence	13.40	2.57
CTQ Abuse	19.64	7.03
RSA - Baseline	6.92	1.34
RSA - Speech	6.03	1.41
RSA - Math	6.38	1.43
PEP - Baseline	102.61	14.77
PEP - Speech	86.39	19.58
PEP - Math	90.99	18.30
Cortisol - Baseline	6.59	6.00
Cortisol – Post Speech and Math	10.98	8.09
DHEA-S - Baseline	16.61	34.62
DHEA-S – Post Speech and Math	17.44	33.95
YSR Externalizing	52.12	9.63
YSR Internalizing	52.92	10.26

Note: SAVE = Screen for Adolescent Violence Exposure; CTQ = Childhood Trauma Questionnaire; YSR = Youth Self Report; RSA = Respiratory sinus arrhythmia; DHEA-S = Dehydroepiandrosterone sulphate; PEP = Pre-ejection period.

Table 2

Bivariate Correlations Among Physiology, Childhood Adversity, Psychopathology, and Demographic Variables (N=167)

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. Age	--												
2. Female	−.12	--											
3. Poverty	−.01	−.06	--										
4. Interpersonal Violence	.22**	−.05	.21**	--									
5. PEP (Baseline)	.29**	−.09	.10	.07	--								
6. PEP (Speech)	.18*	−.19*	.12	.16*	.68**	--							
7. PEP (Math)	.18*	−.10	.13	.18*	.74**	.90**	--						
8. RSA (Baseline)	−.10	.08	.15	.01	−.02	.01	−.04	--					
9. RSA (Speech)	−.02	−.15	.21*	.16*	.20*	.46**	.43**	.16*	--				
10. RSA (Math)	−.05	.01	.13	.14	.10	.30**	.33**	.37**	.67**	--			
11. LogCortisol:DHEA (Baseline)	.00	.12	.20*	.10	−.07	−.08	−.08	.13	−.04	.06	--		
12. LogCortisol:DHEA (Post Speech)	−.09	.13	.20*	−.02	−.05	−.15	−.14	.17*	−.07	.04	.84**	--	
13. YSR Externalizing	.15	.07	.21**	.48**	.03	.09	.13	.07	.15	.18*	−.01	−.13	--
14. YSR Internalizing	.19*	.04	.10	.44**	−.07	−.00	−.01	−.04	.11	.02	−.09	−.15	.54**

Note:

* $p < .05$;

** $p < .01$;

YSR = Youth Self Report; PEP = Pre-ejection period; RSA = Respiratory sinus arrhythmia

Table 3

Summary of hierarchical regression analysis for effects of poverty and interpersonal violence on physiological reactivity (N=167)

	Speech			Math		
	β	**S.E**	***p*-value**	**β**	**S.E**	***p*-value**
RSA						
IV	.09	.07	.19	.09	.07	.19
Poverty	.45	.25	.071	.15	.25	.55
IV (controlling for poverty)	.07	.07	.32	.08	.07	.22
Poverty (controlling for IV)	.40	.25	.11	.09	.25	.70
PEP						
IV	1.51	.72	.035	1.65	.61	.007
Poverty	2.48	2.64	.35	2.47	2.30	.28
IV (controlling for poverty)	1.44	.73	.050	1.59	.63	.012
Poverty (controlling for IV)	1.33	2.67	.62	1.20	2.31	.60
	Post Speech and Math					
	β	S.E	*p*-value			
Cortisol:DHEA-S						
IV	−.08	.04	.033			
Poverty	.09	.14	.50			
IV (controlling for poverty)	−.09	.04	.021			
Poverty (controlling for IV)	.15	.14	.28			

Note: RSA = respiratory sinus arrhythmia; PEP=pre-ejection period; DHEA-S=dehydroepiandrosterone sulphate; IV = interpersonal violence. Analyses control for age, sex, baseline reactivity and respiration rate (for models with RSA).