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The Longitudinal Impact of Exposure to Violence on Cortisol Reactivity in Adolescents

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Abstract

Purpose—The purpose of this report is to examine the effect of exposure to violence on cortisol reactivity in children with no identified serious mental health problems or reports of maltreatment. Exposure to violence was hypothesized to influence development of the stress system in this sample of youth as has been demonstrated in maltreated youth.

Methods—The sample consisted of 124 adolescents, ages 8 to 13 years. Data were collected at two waves of measurement, 12 months apart. Exposure to violence was operationalized as the number of different violent events each child was exposed to as a witness or victim. Cortisol reactivity was evaluated in relation to the Trier Social Stress Test for Children.

Results—Exposure to violence occurring over the 12 months prior to the first assessment is predictive of cortisol reactivity 12 months later in males, even after controlling for age and Time 1 symptoms of psychopathology, cortisol reactivity, and lifetime exposure to violence. Lifetime exposure to violence at Time 1 is positively correlated with symptoms of Major Depressive Disorder and Generalized Anxiety Disorder in both sexes.

Conclusions—The unique aspect of the current findings is that typically, research studying the effects of exposure to violence is conducted with a clinical or maltreated sample. The findings show that recent exposure to violence has an effect on reactions to a laboratory stressor and has longer-term negative mental health consequences. Further study is needed to determine whether these effects are enduring or a shorter-term adaptive response to exposure to violence.

Keywords

Cortisol; Stress; Violence; Adolescence

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Implications and Contribution

This report is unique in that it describes the longitudinal association between exposure to violence and cortisol reactivity in a non-clinical sample of adolescents. These findings indicate the importance of violent experiences on mental health and the neurobiological development of the stress system.

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Introduction

Children exposed to violence are more likely to experience maladaptive endocrine and metabolic changes including hypothalamic-pituitary-adrenal (HPA) axis alterations [1], impairment in cognitive and academic functioning [2], and mental health problems such as depression, posttraumatic stress disorder, anxiety [3], and externalizing behaviors [4]. Prior studies on the effects of exposure to violence (ETV) or other traumatic experiences have been conducted primarily with individuals that have already experienced chronic traumas, such as child maltreatment. Little is known about how exposure to a less severe, acute and unexpected stressor(s) affects neurobiological development in non-maltreated, non-clinical children.

Exposure to trauma and other stressors is related to elevated cortisol reactivity (CR) in adults [5] and adolescents [6] in some instances and blunted CR in adults [7, 8] and adolescents [9] in other instances. In addition, basal levels of cortisol have been found to be both attenuated [10–14] and elevated [10–13, 15] in maltreated or other traumatized youth. The inconsistency in direction of reactivity and basal cortisol may be explained by the type of stressor measured, such as childhood maltreatment [6–9] or major life events [5, 6], whether the stressor was chronic or acute, or the length of time between the stressor and data collection [16]. Animal and human model studies suggest that repeated exposure to acute stressors elicits a habituated stress response to similar acute stressors [16, 17]. Based on these theories and findings, CR was hypothesized to be attenuated in children exposed to violence.

The theoretical rationale for considering ETV and CR in a community-based sample was that, first, exposure to acute and unexpected stressors compared to the more chronic and anticipated childhood maltreatment may have enduring adverse effects on the HPA system similar to that of emotional and physical abuse. Recent evidence shows that early life experiences that are considered minor stressors compared to maltreatment, specifically maternal insensitivity and the experience of out-of-home care in early childhood, were predictive of lower awakening cortisol during adolescence [18]. Insensitive parenting without harsh punishment and child care experiences, although chronic, generally are not considered major traumatic or stressful experiences; nonetheless, each was predictive of cortisol levels more than a decade later. In this study, ETV was assessed at two waves of measurement, capturing acute and unexpected violent events that occurred over the child's lifetime up until the first assessment and the violent events that occurred over the 12-month period between the child's first and second assessment. By controlling for lifetime ETV at Time 1, we were able to make conclusions based on recent acute ETV and their association with the child's reactivity to a social and cognitive stressor.

The second rationale for considering ETV and CR was that cortisol is considered a sensitive and reliable indicator of exposure to laboratory stressors in children and adults [19] and children's reactions to laboratory stressors are considered to generalize to how children will react to challenges in general [20]. Specifically, atypical reactions to laboratory stressors (elevated or attenuated CR) are considered an index of poor self-regulation of the stress system that extends to other areas of functioning as well. An additional consideration was that multiple exposures to stressors, such as violent events, lead to maladaptive HPA axis functioning that have deleterious consequences on psychological health status in adults [21].

A long-standing yet unanswered question in the psychobiology of stress and ETV is whether CR measured at one point in time is indicative of the current psychological state of the organism, if it is reflective of a more enduring trait, or if reactivity is dependent on the current situation [22]. The unanswered question regarding the relation between the stability

of CR is a consequence of the few longitudinal studies that included cortisol measures. Thus, CR as a stable marker of HPA axis functioning and ETV is unknown. Furthermore, adolescence is a key period of development for the HPA axis and CR to a stressor has been shown to be more pronounced in adolescence than in childhood [23]. The current study assessed CR across a one-year period at two time points to establish whether CR is stable across time or is adaptive to a child's experiences and environment, and whether there are developmental influences on reactivity to a stressor.

In addition, sex differences in CR were examined because sex-specific biopsychosocial pathways are expected to contribute to sex differences in CR. These differences may result from variations in psychological responses to interpersonal stress in females and males resulting in differences in biomarkers of stress [24], differences in neuroendocrine responses involving interactions of the HPA axis with sex steroids, as well as brain-related dimorphisms between the sexes [25]. In past studies, CR is greater in non-maltreated adult males than non-maltreated adult females [19] but there are no comparable findings for children. Sex differences also are reported for CR to the TSST-C [24, 25].

In summary, this report assessed the longitudinal relations between self-reported exposure to different forms of violence occurring over the child's lifetime and in the 12 months prior to the second data collection (Time 2) and CR in response to a controlled stressor (Time 2) in a community-based sample of adolescents. The rationale for including this age group is that there is evidence for remodeling of stress response systems during puberty [26], increased physiological reactivity to stress relative to earlier developmental periods [23], higher rates of mental health problems [27], and physical size and proportions and social roles change dramatically [28]. Consistent with research that found stressful situations lead to an attenuated stress response in novel or threatening situations in clinical or maltreated youth, ETV in the past year was expected to negatively predict CR in this community-based sample. In addition, depression, anxiety and post traumatic symptoms were examined in relation to CR so as to rule out the possible confounding influence of these symptoms.

Method

Participants

Participants were 124 ($n = 64$ females) adolescents from a small city and rural communities. In the first wave of measurement at Time 1, girls were ages 8 ($n = 21$), 10 ($n = 20$) or 12 ($n = 23$) years and boys were ages 9 ($n = 19$), 11 ($n = 21$) or 13 ($n = 20$) years (Table 1). The cross-sequential design provides a broader picture of development and accounts for the difference in timing of puberty between the sexes, as females typically mature earlier than males. The study controlled for day of menstrual cycle in females. Data were collected for females on day 5 through 9 of the menstrual cycle [29]. The body mass index (BMI) at Time 1 was calculated for males ($M = 20.85$, $SD = 4.66$) and females ($M = 19.75$, $SD = 5.03$).

The racial/ethnic background of the sample is predominantly White/non-Hispanic (89.6%) and also includes Hispanic (3.7%), African American/Black (2.2%), Asian (2.2%), and biracial (2.1%) participants. The socioeconomic status (SES) for participants was determined using the Hollingshead scale (1975) and is a composite of the child's head of household's income and highest earned academic degree. In this sample, SES ranged from 17 to 66 ($M = 50.47$, $SD = 12.73$) and a score of 50 is representative of middle class. Based on public records, the sample is representative of youth living in the suburban neighborhoods from which the sample was drawn.

Recruitment

The sample was recruited using the American Student List (ASL), a database of school-age children, selected from designated ZIP codes. Randomly selected parents of children on the list were initially contacted by mail and the parents either requested additional information from the lab or were contacted by telephone to determine if the child met eligibility requirements and if the child was interested in participating in the study. The screening questionnaire was designed by nurse practitioners based on similar screening questionnaires developed in medical adolescent clinics. Children were eligible to participate if they met the following criteria: boys and girls ages 9, 11, or 13 years old and 8, 10, or 12 years old, respectively; no current chronic health or severe mental health problems as assessed by a screening questionnaire completed by the parent or guardian that would limit the child's ability to sit through the interview, such as physically handicapped children with gross motor or speech problems or mentally challenged children; and not currently taking medication that influence hormone levels (e.g. corticosteroids). Parents also were asked if the family had any experience with a child protective service agency and no contacts were reported. Twenty-six additional participants were recruited from the community via posted flyers and e-mails. Further details are provided in a previous publication [30].

Procedures

Adolescents who met criteria for the study visited a General Clinical Research Center (GCRC) at a large research university with his or her parent or guardian. The visits were scheduled between the hours of 2:30 PM and 5:30 PM ($M = 3:38$ PM, $SD = .58$). The study was approved by a university institutional review board and a GCRC advisory board. A consent form was signed by the parent and an assent form was signed by the adolescent prior to data collection.

Measures

Trier Social Stress Test for Children (TSST-C)—CR was measured in response to the TSST-C [31], a laboratory social and cognitive stressor. The TSST-C consisted of a story completion task followed by mental, serial subtraction while being monitored by two confederate judges. Different story completion tasks and judges were used at Time 1 and Time 2. Participants were told their performance would be compared to others of the same age.

Cortisol—Children were instructed not to eat, exercise, or smoke for two hours prior to saliva collection and compliance was confirmed verbally by parent and child. At the lab, children were not allowed to eat or drink any liquid other than water during the procedure. Children passively drooled into a 5-mL tube until 4-mL of saliva was collected or 5 minutes passed. The samples were collected 20 min and 5 min pre-TSST-C, and 0 min, 10 min, and 20 min post-TSST-C.

Saliva samples were assayed by enzyme immunoassay (Cat. No. 1-0102/1-0112; Salimetrics, State College, PA) in 25 μ l of saliva. Sensitivity of the test ranges from 0.007 to 1.2 μ g/dL and the average intraassay and interassay coefficients of variation are 5.34% and 9.86%, respectively. Each sample was assayed in duplicate and the mean value was used in the analysis.

CR was determined by calculating Area Under the Curve with respect to Increase (AUC_I) using samples 2, 3, and 4, and is a measure of the total change in cortisol over time [32]. The fifth sample of saliva was not utilized because it captured the recovery period at Time 1 (sample 4 $M = 0.19$ μ g/dL; sample 5 $M = 0.16$ μ g/dL) and Time 2 (sample 4 $M = 0.20$ μ g/dL; sample 5 $M = 0.18$ μ g/dL) rather than reactivity to the stressor [33].

My Exposure to Violence (MyETV)—The MyETV is a self-report measure of ETV occurring within the past 12 months and over the child's lifetime [34]. The MyETV is a well-validated measure consisting of six scales (Lifetime and past year exposure to: witnessed violence, victimization, and total exposure) with high test-retest reliability ($r = .75$ to $.94$) and internal consistency was judged to be acceptable (*Cronbach's alpha* = $.68$ to $.93$) [34]. The MyETV measures the number of different violent events that occurred over the child's lifetime and within the past year (Table 3) and the experiences tapped by this measure are not usually thought of as those meeting DSM criteria. The present analysis assessed total ETV over the child's lifetime at Time 1 and within the past 12 months at Time 2.

Psychopathology—Given that symptoms of psychopathology are associated with HPA axis functioning, a psychiatric interview was administered to rule out the influence of psychopathology on CR. The Diagnostic Interview for Children-IV R (DISC-IV R) is a structured interview used to assess symptoms of psychopathology in children and corresponds with the Diagnostic and Statistical Manual of Mental Disorders [35]. Senior faculty and research assistants were trained by one of the psychiatrists who assisted in the development of the DISC-IV R. Children completed the DISC-IV R and the three scales assessing major depressive disorder (MDD), generalized anxiety disorder (GAD), and posttraumatic stress disorder (PTSD) symptoms occurring within the past 12 months were utilized to evaluate the child's current symptomatology (Table 1) because depression, anxiety, and PTSD have been shown to be positively associated with violence exposure [3].

Pubertal Stage—A pediatric nurse assessed pubertal stage using Tanner criterion by performing a physical exam of the adolescent. A self-rating of pubertal stage was used if the adolescent did not consent to the examination ($n = 8$). Breast (females) and genital (males) stage was used in the analysis.

Data Analysis Plan—CR values were found to be positively skewed; therefore, a constant was added to CR values and a square root transformation was performed. Two multiple linear regression models were developed and Time 2 CR was regressed on age, Time 1 lifetime ETV, Time 1 CR, and symptoms of psychopathology (Model 1), and Time 1 covariates and ETV occurring in the 12 months before Time 2 (Model 2). Symptoms of MDD, GAD, and PTSD were included in order to examine the association between ETV and depression and anxiety, and to rule out the effect of symptoms of psychopathology on CR.

Results

Descriptive Statistics

Descriptive information for all variables appears in Table 1 and Pearson correlations by child's sex appear in Table 2. There were no sex differences in symptoms of MDD, GAD, and PTSD. Mean ETV and CR did not differ significantly between males and females at Time 1 or Time 2, and CR was significantly greater at Time 2 in males and females (Table 1). Lifetime ETV at Time 1 was positively correlated with symptoms of MDD in males and females (Table 2).

Hypothesis Tests

A multiple linear regression model tested the association between ETV and CR in males and females. Covariates (age, Time 1 lifetime ETV, CR, and symptoms of MDD, GAD, and PTSD) and ETV occurring in the 12 months before Time 2 were entered into the model in two separate steps. The findings were similar for males and females when sample 5 was

included in the calculation of cortisol AUCi. The F statistics, betas, and p-values are presented in Table 4.

Males

Model 1 was not significant for males, such that age, Time 1 lifetime ETV and CR, and symptoms of MDD, GAD, and PTSD did not explain the variation in Time 2 CR. When ETV occurring in the 12 months before Time 2 was added to the model (Model 2), the overall model was significant and the analysis revealed that boys with greater ETV, after controlling for Time 1 covariates, have lower CR at Time 2 (Table 4).

Females

Model 1 and Model 2 were not significant for females (Table 4).

Discussion

The purpose of this report is to test longitudinal hypotheses regarding the effect of ETV on CR in adolescents. The role of psychiatric symptoms of depression, anxiety, and PTSD in CR also was examined. The hypothesis that adolescents with greater recent ETV would have significantly lower CR was supported for males. The findings held even after controlling for symptoms of psychiatric disorders, age, and CR and lifetime ETV at Time 1. Symptoms of MDD and GAD were positively associated with lifetime ETV at Time 1, indicating that adolescents with greater lifetime ETV report more symptoms of MDD and GAD.

This is the first known report to test the relation between CR and ETV in a community-based sample of adolescents using more than one wave of measurement. In previous studies, samples generally consisted of adults recruited from clinical or maltreated populations without the benefit of data collection at more than one time point. In addition, previous studies on childhood experiences generally were retrospective, increasing the possibility that a confounding influence could explain the difference in CR between the maltreated and non-maltreated individuals. In this report, adolescents were asked about lifetime and concurrent ETV. The current longitudinal design allowed for the measurement of short-term change in laboratory CR and the evaluation of longer-term effects of ETV on CR in adolescence.

Existing literature supports the findings in this report and adds to the DSM findings regarding exposure and psychiatric diagnoses. Exposure to psychological stressors has been shown to affect components of the HPA axis in multiple studies [36]. A substantial portion of the literature on HPA axis function and exposure to stressors focuses on CR in an adult population reporting childhood maltreatment [37]. Adults with a history of childhood maltreatment and no current psychopathology were found to have decreased CR in response to a social stressor [7]. Similarly, in a non-clinical adult sample, CR was attenuated in individuals with multiple adverse life events compared to those with one or no reported adverse life events [38]. Studies focusing on children and cortisol predominantly assessed CR in maltreated youth with few studies examining CR in non-maltreated youth. Overall, our findings are consistent with studies showing attenuated CR in maltreated youth [1, 39, 40] in males.

Sex differences were apparent; ETV was predictive of CR in males only. One theoretical explanation for this difference is that, although males and females may show the same physiological response to an acute stressor, their behavioral response may differ [24]. This difference may affect responsivity to future stressors, although we were unable to test this hypothesis in the current study. Age may also explain the sex difference as adolescents are shown to have greater reactivity to a stressor than children [23]; however, this has been ruled out by controlling for age in the analysis. We suggest our findings be considered with

respect to multiway interactions of sex, age, violence type and biological response, and future studies could explore these interactions in larger samples.

There were limitations to this study as well. The sample size is modest and the lack of power may explain non-significant findings for girls. In addition, the HPA response to different forms of violence was not assessed given our sample size. Nonetheless, in this study the small sample size should not diminish the importance of the novel findings, as we were able to collect multiple waves of data providing an empirical basis for developing future longitudinal hypotheses. The sample was primarily White/non-Hispanic descent and predominantly recruited from suburban neighborhoods, limiting the generalizability of the findings to White/non-Hispanic children living in nonurban or large city, suburban communities. Future research should include a larger, more diverse sample over an extended time period. In summary, the findings provide evidence for a relation between self reported ETV, a generally recognized life stressor for young adolescents, and dampened CR.

The findings of this study attest to the importance of early youth experiences on the development of the neurobiological stress system in adolescents. The unique aspect of the current findings is that they support the notion that even past ETV in which the child was directly or indirectly involved appear to have at least a one-year effect on reactions to a controlled laboratory stressor. Future research should address questions regarding duration of exposure, time elapsed following exposure, and lower CR and health consequences of exposure.

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Table 1

Descriptive Statistics and Sex Differences

Measure	M (SD)		Total Sample (n = 124)	Males (n = 60)	Females (n = 64)	T value	p-value
Time 1							
Age (years)			10.49 (1.68)	10.94 (1.61)	10.06 (1.64)	3.15	<.01
Pubertal (Tanner) Stage			2.34 (1.08)	2.44 (1.02)	2.24 (1.13)	1.03	0.31
BMI			20.29 (4.87)	20.85 (4.66)	19.75 (5.03)	1.32	0.19
Cortisol Reactivity (µg/dL/min)			3.13 (3.98)	2.98 (3.70)	3.28 (4.25)	-0.42	0.68
Transformed Cortisol Reactivity (µg/dL/min)			3.19 (0.65)	3.18 (.58)	3.20 (0.72)	-0.16	0.87
Lifetime Exposure to Violence			2.03 (1.84)	2.30 (1.91)	1.80 (1.75)	1.61	0.11
Symptoms of Major Depressive Disorder			5.13 (4.66)	5.14 (4.80)	5.12 (4.56)	0.03	0.98
Symptoms of Generalized Anxiety Disorder			2.46 (2.18)	2.45 (2.37)	2.47 (2.00)	-0.05	0.96
Symptoms of Posttraumatic Stress Disorder			0.02 (0.26)	0.00 (0.00)	0.04 (0.36)	-0.97	0.33
Time 2							
Cortisol Reactivity (µg/dL/min)			11.27 (12.44)	9.17 (9.53)	13.24 (14.46)	-1.86	0.07
Transformed Cortisol Reactivity (µg/dL/min)			5.72 (1.16)	5.54 (1.10)	5.88 (1.21)	-1.61	0.11
Past 12 months Exposure to Violence			0.38 (0.70)	.42 (0.80)	0.33 (0.59)	0.75	0.45

Note. Mean (Standard Deviation); Time 1 = first wave of measurement; Time 2 = second wave of measurement; Lifetime Exposure to Violence and Past 12 months Exposure to Violence are from MyETV [34]; Symptoms of Major Depressive Disorder, Generalized Anxiety Disorder, and Posttraumatic Stress Disorder are from the DISC IV [35]; Cortisol Reactivity is in response to the TSST-C [31] and is calculated as Area Under the Curve Increase [32]; Transformed Cortisol Reactivity is a square root transformation.

Table 2

Pearson Correlation Analysis for Males and Females

	1	2	3	4	5	6	7	8	9	10
1: Cortisol Reactivity (Time1)	--	.140	.984**	.134	-.311*	-.068*	.335**	-.175	-.121	-.046
2: Cortisol Reactivity (Time 2)	.038	--	.151	.932**	-.046	-.257*	.087	.068	.078	.034
3: Transformed Cortisol Reactivity (Time 1)	.951**	.080	--	.139	-.319**	-.041	.332**	-.179	-.115	-.047
4: Transformed Cortisol reactivity (Time 2)	.046	.992**	.089	--	-.022	-.379**	.097	.108	.117	.076
5: Lifetime Exposure to Violence (Time 1)	-.087	-.174	-.090	-.179	--	.205	-.024	.151	.299*	.311*
6: Past 12 months Exposure to Violence (Time 2)	-.174	-.039	-.179	-.017	.254*	--	.020	-.139	-.030	.000
7: Age	.297*	.157	.337**	.188	-.150	-.020	--	-.284*	-.214	-.141
8: Pubertal Stage	-.135	.054	-.099	.047	.096	-.011	.005	--	.222	.041
9: GAD Symptoms	.068	-.034	.108	-.027	.448**	.104	.042	.113	--	.588**
10: MDD Symptoms	.120	-.134	.127	-.131	.567**	.185	-.113	.103	.654**	--
11: PTSD Symptoms	.065	-.019	.073	-.010	.084	.138	.144	-.140	.094	.051

Note.

* = $p < .05$;** = $p < .01$;

Pearson correlation; Male correlations appear above the diagonal, female correlations below the diagonal; Time 1 = first wave of measurement; Time 2 = second wave of measurement; Cortisol Reactivity is in response to the TSST-C [31] and is calculated as Area Under the Curve Increase [32]; Transformed Cortisol Reactivity is a square root transformation; Lifetime and past 12 months Exposure to Violence is from MyETV [34]; GAD = Generalized Anxiety Disorder; MDD = Major Depressive Disorder; PTSD = Post-traumatic Stress Disorder; GAD, MDD, PTSD symptoms measured by the DISC IV [35].

Table 3

Percent Reporting “Yes” to Each Type of Exposure

Exposure Type	Lifetime at Time 1			Past 12 months at Time 2		
	Total Sample	Males	Females	Total Sample	Males	Females
Witnessed someone being chased	16.3	15.2	17.4	1.5	3.0	0.0
Victim of being chased	8.1	10.6	5.8	.7	1.5	0.0
Witnessed someone being hit, slapped, punched, or beaten up	25.2	25.8	24.6	5.9	10.6	1.4
Victim of being hit, slapped, punched, or beaten up	17.0	21.2	13.0	4.4	6.1	2.9
Witnessed someone being attacked with a weapon	0.0	0.0	0.0	0.0	0.0	0.0
Victim of being attacked with a weapon	3.7	6.1	1.4	0.0	0.0	0.0
Witnessed someone being shot	0.0	0.0	0.0	0.0	0.0	0.0
Witnessed someone being shot at but not wounded	.7	0.0	1.4	0.0	0.0	0.0
Heard gunfire nearby	16.3	18.2	14.5	5.9	3.0	8.7
Witnessed a serious accident where someone was hurt very badly/died	28.1	31.8	24.6	5.1	4.5	5.8
Present in a serious accident where you or someone else was hurt very badly/died	6.7	9.1	4.3	0.0	0.0	0.0
Witnessed someone being threatened	8.1	9.1	7.2	1.5	1.5	1.4
Victim of being threatened	2.2	3.0	1.4	.7	1.5	0.0
Present in a natural disaster	12.6	18.2	7.2	1.5	1.5	1.4
Other traumatic situation not listed	9.6	10.6	8.7	.7	1.5	0.0
Found out someone you knew had been killed	18.5	19.7	17.4	2.2	3.0	1.4
Found out someone you knew died suddenly/serious injured	31.3	31.8	30.9	7.4	4.5	10.1

Note. Time 1 = first wave of measurement; Time 2 = second wave of measurement; Lifetime at Time 1 and Past 12 months at Time 2 is from MyETV [34].

Table 4

Regression of Time 2 Cortisol Reactivity on Exposure to Violence, Age, and Psychopathology (Males)

Variable	B	SE	β	F	R ²	ΔR^2
Model 1						
Age (years)	0.89	0.67	0.21			
Time 1 Cortisol Reactivity ($\mu\text{g/dL/min}$)	0.15	0.30	0.08			
Time 1 Lifetime Exposure to Violence	-0.28	0.54	-0.08			
MDD Symptoms	0.02	0.24	0.01			
GAD Symptoms	0.46	0.49	0.16			
PTSD Symptoms ^a	--	--	--			
Full Model				0.78	0.07	0.07
Model 2						
Time 2 Exposure to Violence Past 12 months	-3.78	1.10	-0.45 ^{**}			
Full Model				2.76 [*]	0.25	0.18

Note.

^{*} $p < .05$.^{**} $p < .01$.

^a = Males reported no symptoms of PTSD; Cortisol Reactivity is in response to the TSST-C [31] and is calculated as Area Under the Curve Increase [32] and is a square root transformation; MDD, GAD, PTSD symptoms measured by the DISC IV [35].

Table 5

Regression of Time 2 Cortisol Reactivity on Exposure to Violence, Age, and Psychopathology (Females)

Variable	B	SE	β	F	R ²	Δ R ²
Model 1						
Age (years)	1.68	1.23	0.19			
Time 1 Cortisol Reactivity (μ g/dL/min)	-0.00	0.48	-0.00			
Time 1 Lifetime Exposure to Violence	-1.21	1.36	-0.15			
MDD Symptoms	-0.33	0.61	-0.11			
GAD Symptoms	0.71	1.26	0.10			
PTSD Symptoms	-1.41	5.02	-0.04			
Full Model				0.86	0.09	0.09
Model 2						
Time 2 Exposure to Violence Past 12 months	0.61	3.36	0.03			
Full Model				0.73	0.09	0.00

Note.

* $p < .05$;

** $p < .01$;

Cortisol Reactivity is in response to the TSST-C [31] and is calculated as Area Under the Curve Increase [32] and is a square root transformation; MDD, GAD, PTSD symptoms measured by the DISC IV [35].