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Cortisol Response to Stress in Female Youths Exposed to Childhood Maltreatment: Results of the Youth Mood Project

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Abstract

Background—Few studies have examined stress reactivity and its relationship to major depressive disorder (MDD) and posttraumatic stress disorder (PTSD) among maltreated youth. We examined differences between maltreated and control participants in heart rate and cortisol resting and reactivity levels in response to a psychosocial stressor.

Methods—We recruited 67 female youths aged 12 to 16 with no prior history of depression from child protection agencies and a control group of 25 youths matched on age and postal code. Child maltreatment was measured with two self-report instruments. Psychiatric status was assessed using the Schedule for Affective Disorders and Schizophrenia for School-Aged Children.

Results—Piecewise multilevel growth curve analysis was used to model group differences in resting and reactivity cortisol levels and heart rate in response to the Trier Social Stress Test (TSST). During the resting period, both the maltreated and control groups showed a similar decline in levels of cortisol. During the reactivity phase, youth in the control group showed an increase in cortisol levels following the TSST and a gradual flattening over time; maltreated youth exhibited an attenuated response. This blunted reactivity was not associated with current symptoms of MDD or PTSD. There were no group differences in resting and reactivity levels of heart rate.

Conclusions—These findings provide further support for hypothalamic-pituitary-adrenal axis dysregulation among maltreated youth. Since the ability to respond to acute stressors by raising

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cortisol is important for health, these findings may assist in understanding the vulnerability of maltreated youth to experience physical and mental health problems.

Keywords

Child maltreatment; hypothalamic-pituitary-adrenal axis

It is now well recognized that exposure to child maltreatment, which includes physical, sexual, and emotional abuse and neglect, is associated with psychiatric impairment in childhood, adolescence, and adulthood (1–5). Major depressive disorder (MDD) has a strong association with maltreatment, especially among female individuals (4,6,7). It remains unclear, however, why some individuals exposed to child abuse or neglect subsequently experience psychiatric impairment and others do not.

De Bellis *et al.* (8), De Bellis (9), and Heim *et al.* (10,11) have suggested that alterations in corticotropin-releasing hormone (CRH) mediate the development of MDD in response to child maltreatment. Work from animal studies provides support for the hypothesis that increased activation of the hypothalamic-pituitary-adrenal (HPA) axis as manifested by increased secretion of CRH is linked to severe stress in early life (12,13). Hypothalamic-pituitary-adrenal axis and autonomic hyperreactivity have been observed in response to a psychosocial stress test among a group of women with a history of child physical and/or sexual abuse compared with control subjects (14).

Tarullo and Gunnar (15) found that most studies of HPA activity involving children exposed to maltreatment examined basal levels rather than stress reactivity (for a review, see reference 15). Results of the few investigations examining HPA axis response to stress among maltreated children and adolescents are conflicting (16). De Bellis *et al.* (8) showed that girls with a past history of sexual abuse and higher rates of dysthymia manifested decreased adrenocorticotropic hormone (ACTH) response to CRH but no differences in cortisol secretion compared with a control group. Kaufman *et al.* (17) examined HPA axis responses to CRH in a group of male and female depressed youth (abused and nonabused) compared with control children. There were no differences in cortisol secretion, but depressed, abused youth who were continuing to experience chronic adversity showed increased ACTH secretion. Both studies provide important information about HPA responses to CRH but had limited numbers of maltreated youths in the sample.

The purpose of the present study was to examine whether resting levels of cortisol, heart rate, and pattern of stress response to a psychosocial stressor differed between youth exposed to maltreatment and a control group. To understand the effects of MDD/posttraumatic stress disorder (PTSD), we examined whether stress reactivity differed among female youth based on the presence or absence of MDD and PTSD symptoms.

Methods and Materials

Participants

Female youths between the ages of 12 and 16 from three local child protection agencies (CPA) qualified for eligibility review. Two trained undergraduate research assistants, who signed confidentiality agreements with the agencies, reviewed the 243 qualifying cases and summarized the files of the 201 female youths who had open cases with a history of maltreatment. Two members of the research team and two clinical researchers independently adjudicated the summaries for eligibility and to determine abuse exposures and severity. About half (108/201) were ineligible, based on these exclusion criteria: a positive history of depression (25%); current or past use of antidepressants or medication affecting the HPA

axis (31%); cognitive impairment (20%); living in an unstable environment (12%); or unconfirmed abuse (6%). Also, 6% could not be contacted. A letter describing the study was mailed to the 93 eligible youths and their parent/guardian; 76 (82%) agreed to participate. Nine were subsequently excluded because of age, medication, or inability or unwillingness to attend the clinic visit.

A control group of 25 youths was recruited from an existing database of children whose mothers had agreed at the child's birth to be contacted for future participation in research studies. Control subjects were matched with the sample with respect to age and postal code (a proxy for socioeconomic status [SES]). Control subjects had no history of involvement with a CPA and no maltreatment.

Consent to participate in the study for youth in the care of the state was provided by the CPA; parental consent was provided for those in the care of their parents. Youths who were 16 years of age provided their own consent, while those under the age of 16 provided assent. This research was approved by the Hamilton Health Sciences/McMaster University Research Ethics Board. Participants received $15 for each home assessment and $25 for each clinic visit.

Measures and Procedures

This investigation was part of a larger study carried out over a 2-year period with assessments every 6 months. At the initial home visit by a public health nurse, youths self-completed demographic questions, the Childhood Experiences of Violence Questionnaire (CEVQ) (18,19), the Childhood Trauma Questionnaire (CTQ) (20,21), and the Tanner scale (22–24) (see Supplement 1 for information about the psychometric properties of these instruments).

The first clinic visit was scheduled to coincide with days 6 to 10 of the youth's menstrual cycle, since HPA responsiveness to stress is affected by menstrual cycle phase (25). All appointments were scheduled during the late afternoon (16:00 to 19:00 hours) because cortisol levels show only small spontaneous fluctuations during this time (26). Participants were also asked to refrain from eating, drinking, and smoking for at least 2 hours prior to their visit. Upon arrival at the clinic, participants filled out a questionnaire asking when they last ate, drank, and smoked; the date of their last menstrual cycle; and if they were taking any medications. A registered nurse helped them apply a Polar Vantage XL (Polar Electro Oy, Kempele, Finland) heart monitor to measure heart rate (HR). The participant then underwent the Trier Social Stress Test (TSST).

The TSST has been found to reliably induce psychological and endocrine stress responses in different populations of adults, children, and youth (27,28). The stress protocol for children and youth has a brief stress anticipation period, a public speaking task (5 minutes), and mental arithmetic (5 minutes) in front of a panel of two judges. Peak cortisol concentrations are reliably found 10 minutes after stress termination in this protocol (28).

At the end of the experiment, the participants were asked to rate how stressful the experiment was for them from "not stressful at all" to "very stressful."

Over the course of the first visit, both HR and saliva samples were recorded at −45, −25, −5, +20, +40, and +60 minutes, with the TSST at 0 minutes. During the preliminary phase of the study, a baseline salivary sample at −25 minutes was obtained (n = 16 of 67 participants), but initial analyses showed that participants had elevated cortisol in this first sample, so a sample was added at −45 minutes to have two possible true resting (basal) samples before

the stress test. The samples were obtained by passive drool into sterile plastic tubes. Saliva samples were stored at −80°C until the time of analysis.

Following the TSST, a trained clinician administered the Schedule for Affective Disorders and Schizophrenia for School-Aged Children-Epidemiologic Version 5 (K-SADS-E) (29).

Cortisol Assays

Salivary cortisol concentrations, reflecting the level of unbound cortisol, were determined by enzyme immunoassay (Salimetrics, State College, Pennsylvania). Control, standard, and unknown samples were pipetted in duplicate. The intra-assay coefficients of variation for cortisol were 8.3% for a low-concentration sample and 6.9% for a high-concentration sample. The interassay coefficient of variation was 10.6% across 10 individual runs.

Statistical Analysis

To examine group differences in sociodemographic and clinical characteristics, independent *t* tests for continuous variables and chi-square analyses for categorical variables were conducted. Piecewise multilevel growth curve modeling with MLwiN (Centre for Multilevel Modeling, University of Bristol, Bristol, United Kingdom) (30) examined differences between maltreated and control youth in resting and post-TSST levels of cortisol and HR. The analysis used measurements of HR and the natural logarithm of cortisol taken on the same individuals at the six consecutive time points described above. Data collected in this way form a hierarchical or multilevel structure: repeated assessments over time (level 1) are nested within individuals (level 2). Growth curve models are better than repeated measures analysis of variance (ANOVA), which has traditionally been used to analyze cortisol data, because the former do not require complete data for every participant or that observations be equally spaced in time (31). Growth curve modeling appropriately handles data that violate the assumption of independence (32).

The time prior to the TSST was conceptualized as a resting measure of cortisol, while the post-TSST period was considered cortisol reactivity. A piecewise approach was necessary to account for the discontinuity in the trajectory of log cortisol and HR before and after the TSST. This approach permits separate but simultaneous modeling of the trajectories (slopes) of resting and reactivity levels of cortisol and HR. The observed values of log cortisol and HR were plotted as a function of the six assessment points, separately for the maltreated and control youth (see Figures 1 and 2, respectively). Figure 1 informed our choice of the appropriate mathematical functions for time. Prior to the TSST, time was modeled as a linear function; subsequent to the TSST, both a linear and quadratic function of time were specified, so a squared term for reactivity was added to the equation. A similar analysis was run for HR. Our approach to coding for piecewise regression is adapted from Llabre *et al.* (33). Time was centered at 5 minutes before the TSST, making this the common intercept point.

Three growth models were developed, after controlling for time of day. Model 1 characterizes baseline levels and growth trajectories in log cortisol during resting and reactivity time periods separately. Model 2 includes fixed effects to examine differences between groups at baseline and in the growth trajectories. To test for differences in slopes during resting and reactivity periods, three interaction vectors were included: group × resting, group × reactivity, and group × reactivity^2. In model 3, individual level covariates, specifically SES, Tanner pubertal stage, current MDD symptoms, and current PTSD symptoms, were included to examine whether group differences remained after taking into account these potentially confounding variables. Identical models were run to assess differences in resting and reactivity levels of HR.

Results

Table 1 presents selected sociodemographic and clinical characteristics of the maltreated and control groups. Significant group differences on all sociodemographic characteristics emerged, with the exception of age and ethnicity. Compared with the control group, youth exposed to maltreatment live in families with lower levels of SES and are less likely to live with at least one biological parent (32.8% vs. 100%). In terms of clinical characteristics, youth exposed to child maltreatment are more likely to have symptoms of MDD and PTSD. The two groups are similar in levels of pubertal development.

Table 2 summarizes the exposures of maltreated female youths. The prevalence of moderate physical abuse varied from 36% to 66%, depending on measurement (CEVQ, CTQ, K-SADS-E, adjudication). Severe physical abuse was experienced by 13% to 40% of this group. Exposure to sexual abuse of moderate severity was found in 25% to 36%; severe sexual abuse ranged from 9% to 30%. Emotional abuse was found in 27% to 72% of maltreated youths. About half of the maltreated youths reported witnessing domestic violence. Adjudicators found neglect in the majority (90%) of maltreated youths; CTQ indicated moderate and severe emotional neglect in 30% and 19% of these youths, respectively.

Self-report ratings of stress regarding participation in the TSST showed that the majority of both control and maltreated participants experienced stress with this task. There were no statistically significant differences in the proportion of control and maltreated participants who rated the TSST as at least “sort of stressful” (76.0% vs. 68.1%; Fisher’s exact test; $p = .8$) or as at least “stressful” (40% vs. 44%; Fisher’s exact test; $p = .6$), respectively. We also examined the stress ratings as main effects and interaction terms; this did not alter our findings.

Figures 1 and 2 present the observed values of log cortisol and HR. The plots reveal a decrease in levels of log cortisol and HR prior to the TSST and an increase immediately following the stressor with an eventual plateau at the end of the assessment period. The cortisol plot (Figure 1) also indicates relatively parallel downward slopes for the maltreated and control youth prior to the stressor, but cortisol reactivity appears steeper for the control youth relative to maltreated youth. The observed values for HR show roughly parallel lines during the resting and reactivity periods for the maltreated and control youth.

Table 3 shows the beta coefficients and standard errors for fixed parameters and the variance and covariance components of random parameters from the piecewise multilevel growth curve modeling with repeated assessments of log cortisol as the dependent variable. Only significant covariance terms were retained in the model. In model 1, at 5 minutes before the introduction of the TSST, the predicted mean value of the log cortisol is 1.055 nmol/L (model 1, intercept). During the resting period of the assessment, there was an average decrease of .108 nmol/L in log cortisol per 10-minute interval (model 1, resting). Following the TSST, levels of cortisol initially increased by an average of .116 nmol/L per 10-minute interval, but the increase was gradually reduced by an average of .004 per 10-minute intervals ($2 \times -.002$). The variance component for each of the parameters in the model (i.e., the intercept, resting, reactivity, and reactivity^2) indicates that there is significant variability between individuals in the intercept and slopes for the resting and reactivity time periods that warrant the inclusion of level 2 predictors. The variance estimates are displayed under random parameters, level 2. The parameter labeled intercept/intercept shows the variation across individuals in levels of cortisol at 5 minutes pre-TSST. The parameters labeled resting/resting and reactivity/reactivity indicate the extent to which there is between-individual variation in cortisol slopes during the resting and reactivity time periods,

respectively. Covariance among parameter estimates indicates that the baseline was significantly related to resting (covariance = .032; correlation = .34), with higher intercept values associated with flatter resting slopes. There was considerable correlation (.78) between the linear and quadratic components of reactivity.

In model 2, we examine group differences in mean levels of cortisol at the intercept/baseline (5 minutes pre-TSST) and in the growth trajectories during resting and reactivity. Relative to the control group, youth exposed to maltreatment do not have elevated levels of cortisol at baseline. During the resting period, both the maltreated and control group show a similar decline in levels of cortisol: the interaction term group × resting level does not reach statistical significance.

Differences in slopes between the maltreated and control group are evident during the reactivity phase. Youth in the control group show an increase in levels of cortisol following the TSST and a gradual flattening over time, while maltreated youth do not show an increase in levels of cortisol, nor do they have a gradual flattening of response. In model 3, pubertal development was the only significant predictor of mean levels of cortisol at baseline: youth who were more advanced in their pubertal development had higher log cortisol. The inclusion of these covariates in the model did not alter the magnitude of the group differences in reactivity. To further examine the association between cortisol reactivity and current symptoms of MDD and PTSD, we reanalyzed our data excluding all cases who met the criteria for current MDD and/or PTSD symptoms. The results remained the same, suggesting that the blunted response evident among the maltreated youth was not due to the presence of PTSD or MDD symptoms. We also included two interaction terms to test for differences in slopes during the reactivity period for those who had current symptoms of MDD or PTSD and those who did not (reactivity × MDD; reactivity × PTSD). The interaction terms were not statistically significant, suggesting that the slopes were parallel for those who had symptoms and those who did not.

A total of 12 female participants (1 control subject and 11 exposed to maltreatment) reported taking oral contraceptives at intake. We redid our analyses excluding all 12 participants on oral contraceptives. We continued to find a blunted response to the TSST among the maltreated group and no evidence of a flattening of response over time. Specifically, the beta coefficients (and standard errors) in this reduced sample for the interaction terms, maltreated × reactivity and maltreated × reactivity^2, are $\beta = -.233$ (.067), $p < .001$ and $\beta = .026$ (.08), $p < .01$, respectively, both of which are comparable with the estimates obtained on the full sample (Table 3, model 2). In both the reduced sample (i.e., participants on oral contraceptives excluded) and in the full sample, we found evidence of a blunted response to the TSST among the maltreated youth, suggesting that our findings are not a result of a subset of females taking oral contraceptives.

Identical models were run with the repeated assessment of HR as the dependent variable. The results from these analyses indicated that there were no group differences in resting and reactivity levels. Both groups showed a general decline in HR during the resting period, an increase following the TSST, and a gradual flattening over time. The table of results is available from the authors upon request.

Discussion

We found a blunted cortisol response (but no difference in heart rate response) to a psychosocial stressor among maltreated female youth even after controlling for SES and Tanner pubertal staging, as well as current symptoms of MDD and PTSD. This suggests that in adolescents, the blunted cortisol reactivity to a psychological stressor is associated with

exposure to child maltreatment, regardless of the presence of internalizing symptoms. Our findings support the habituation effect discussed by Murali and Chen (34) where chronic exposure to stressful events may be linked with reduced physiological responsiveness to new stressors over time (35). These authors evaluated the association of exposure to violence with biological reactivity measures in adolescents recruited from a public high school. The acute stressor in their study was an interaction with the experimenter that involved either a debate or a verbal puzzle. Exposure to violence was related to decreased cardiovascular reactivity in terms of blood pressure, HR, and HR variability but not cortisol, whereas our results showed a blunted cortisol response but no difference in HR.

Although normal levels of cortisol secretion were observed in response to a pharmacological challenge in the study by De Bellis *et al.* (8), their abused subjects secreted significantly lower ovine-CRH-stimulated levels of ACTH compared with control subjects. We were unable to measure ACTH in our study; only salivary samples could be collected according to the agreement with participating CPAs. The youths in our study varied in age from 12 to 16 years at the time of recruitment with an average age of 14 years, whereas the De Bellis *et al.* (8) sample was aged 7 to 15 with an average age of 11 years. Tarullo and Gunnar (15) point to the fact that normally developing children manifest an increase in basal cortisol levels from childhood to adolescence related to pubertal status. These authors also comment that most investigations of maltreated children have examined basal cortisol, while studies involving adult participants have focused on stress reactivity. Our study is one of a few to examine stress reactivity in a maltreated female sample without the confound of prior psychotropic medications that may have influenced the HPA axis findings.

Based on findings from a longitudinal study of sexually abused girls, Putnam (36) suggests that the effect of puberty on abused girls may result in a transition from hypercortisolism to hypocortisolism; it would be important to examine the effect of maltreatment on HPA functioning from childhood through puberty to adulthood (15).

In studies of adult women, Heim *et al.* (10,14) examined ACTH, cortisol, and heart rate responses to the TSST (14) and CRH challenge (10) among four groups of women: maltreated, depressed; maltreated, nondepressed; nonmaltreated, depressed; and nonmaltreated, nondepressed. Interestingly, both the psychosocial stressor and pharmacologic challenge led to an increase in ACTH but normal cortisol response among maltreated women with no diagnosis of current MDD. Unlike ACTH, maximum cortisol concentrations were not related to adult trauma or an interaction with adult trauma and child abuse; rather, severity of daily hassles over the past month was negatively associated with cortisol (11). These contrasting findings may be related to the age differences between our samples.

Our study has several limitations. We had hoped to examine stress reactivity across different subtypes of maltreatment, but almost all participants had experienced neglect, thereby precluding any opportunity to compare exposure with abuse with neglect. We recruited only female youth, so we are not able to comment on stress reactivity in male youth. In their study of adolescents, Murali and Chen (34) found gender differences in physiological measures; male youth had increased diastolic blood pressure reactivity but reduced cortisol reactivity compared with female youth. It would be useful to examine the responses of maltreated male youth to a psychosocial stress test such as the TSST. Ideally, psychiatric status of the participants would have been determined based on multiple informant data; however, given that many of the maltreated group were living in foster care and only one third had a biological parent living in the home, we were not able to obtain such information from parents.

It was not possible to obtain information on maternal prenatal stress because of the nature of our sample and so we were not able to control for the potential influence of prenatal programming—the effect of the maternal HPA axis on these youth in utero (37). Results of animal studies examining alterations in HPA reactivity related to gestational stress (38) raises the question of whether subsequent HPA dysregulation years later could, in fact, be associated with prenatal stress, rather than postnatal events, or a combination. Given the history of exposure to domestic violence as well as high rates of poverty in our sample, it is quite possible that the mothers of the maltreated group experienced greater prenatal stress than those in the control group. A prospective study of mothers beginning prenatally that follows their infants longitudinally for exposure to adverse events including maltreatment would be the only way to fully address this issue.

Despite the limitations, this is one of the few studies to examine stress reactivity in youth. We were able to control for a number of important factors, including SES. A study of school children in Quebec varying in age from 6 to 10 years shows that children of low SES have significantly higher salivary cortisol levels than those with high SES, with the largest differences becoming apparent at about age 10 (39). We also gathered information directly from the participants regarding how stressful they found the TSST to be. Murali and Chen (34) explored the hypothesis of whether adolescents exposed to violence might experience a laboratory stress as less stressful than those who have not been exposed to such violence, thereby explaining the reactivity patterns. They found no significant associations between stress appraisal of the laboratory task and the exposure to violence. As outlined above, the majority of youths in both groups reported the TSST as at least somewhat stressful, and the proportions were similar.

This research adds to the existing literature in providing information about stress reactivity among an extremely vulnerable group of maltreated female youth—the majority had experienced neglect and many had been exposed to multiple types of maltreatment. Our results suggest that the blunted cortisol response is related to the exposure to maltreatment and not the presence of internalizing symptoms, an important finding in view of the evidence for the association between internalizing problems and increased basal cortisol levels among maltreated children (15,40). Of note, however, in a sample of children (average age 9 years) attending a summer camp, those with a history of maltreatment and comorbid internalizing and externalizing problems of a clinical level were more likely not to show the typical diurnal decrease in cortisol compared with maltreated youth without such comorbidity (40).

The clinical relevance of the blunted cortisol response in this group of maltreated youth is not clear but may put them at risk for impairment in both physical and emotional health; the ability to respond to acute stressors by raising cortisol is important for health (40). It would have been useful to recruit this sample before puberty, to explore whether this blunted cortisol response is related to pubertal onset. We hope further studies of this nature will lead to knowledge about prediction of risk for mental and physical health conditions in this vulnerable population.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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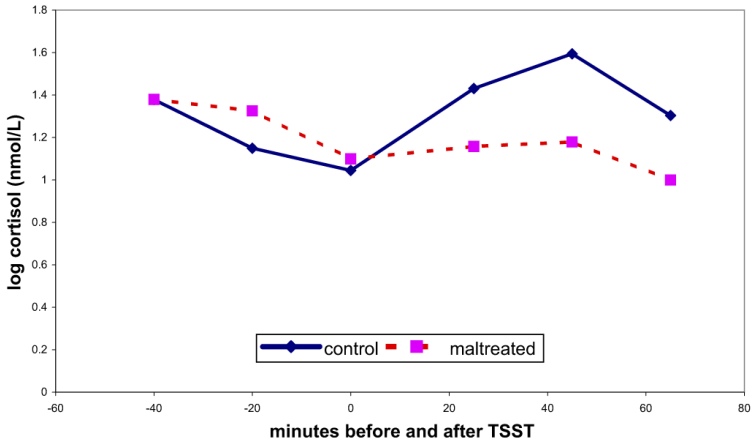

Figure 1.
Observed log cortisol (nmol/L) by group over time. TSST, Trier Social Stress Test.

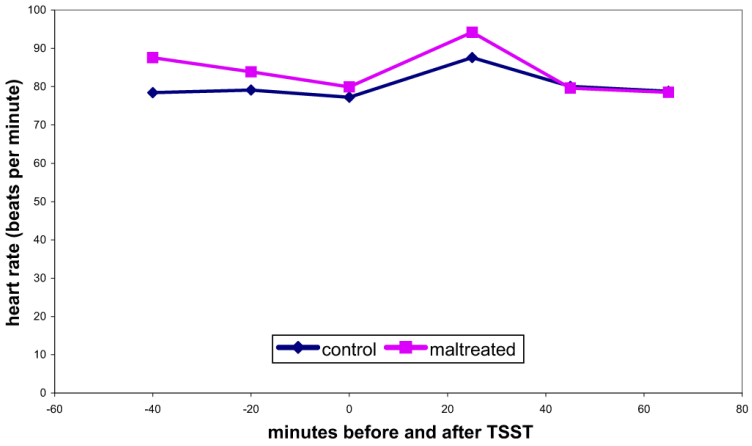

Figure 2.
Observed heart rate (beats per minute) by group over time. TSST, Trier Social Stress Test.

Table 1

Sociodemographic and Clinical Characteristics of Maltreated and Control Youth

Variable	Control Group (*n* = 25)	Maltreated Group (*n* = 67)
Sociodemographic Characteristics		
Hollingshead Four Factor Index SES, M (SD)	47.30 (10.52)	31.92 (9.67)[a,e]
Caucasian, %	96.00	79.10
At least one biological parent in home, %	100.00	32.80%[e]
Age in years, M (SD)	14.00 (1.50)	14.18 (1.15)
Clinical Characteristics		
Pubertal development		
Tanner breast development (stages 4 and 5), %	58.30[b]	75.80
Tanner pubic hair (stages 4 and 5), %	66.70[b]	78.80[a]
Current MDD symptoms, %	.00	22.40[d]
Current PTSD symptoms, %	.00	26.20[c,d]
Clinical Visit Characteristics		
Time of day (hours:minutes), M (SD)	16:55 (39.39 minutes)	17:20 (29.51 minutes)[b,e]

MDD,major depressive disorder; M, mean; PTSD, posttraumatic stress disorder; SD, standard deviation; SES, socioeconomic status.

[a] $n = 66$.

[b] $n = 24$.

[c] $n = 65$.

[d] $p < .01$.

[e] $p < .001$.

Table 2

Prevalence of Maltreatment Type by CEVQ, CTQ, CPA, and K-SADS-E at Time 1

Abuse Type/Measurement	Any Indicators *N* (%)	CEVQ *N* (%)	CTQ-Cutoff Moderate[a] *N* (%)	CTQ-Cutoff Severe[b] *N* (%)	CPA *N* (%)	K-SADS-E *N* (%)
Physical Abuse	54 (80.6)	31 (46.3)	24 (35.8)		44 (65.7)	35 (52.2)
Sexual Abuse	40 (59.7)	24 (35.8)	22 (32.8)		17 (25.4)	21 (31.3)
Severe Physical Abuse	30 (44.8)	27 (40.3)		14 (20.9)	9 (13.4)	
Severe Sexual Abuse	23 (34.3)	20 (29.9)		11 (16.4)	6 (9.0)	
Emotional Abuse	55 (82.1)		28 (41.8)	18 (26.9)	48 (71.6)	
Emotional Neglect			20 (29.9)	13 (19.4)		
Neglect[c]	64 (95.5)				60 (89.6)	
Witnessing Domestic Violence	45 (67.2)				34 (50.8)	31 (46.3)
Physical Assault					3 (4.5)	
Sexual Assault					10 (14.9)	

N = 67 (maltreated group).

CEVQ, Childhood Experiences of Violence Questionnaire; CPA, child protection agencies adjudication; CTQ, Childhood Trauma Questionnaire; K-SADS-E, Schedule for Affective Disorders and Schizophrenia for School-Aged Children-Epidemiologic Version 5.

[a] Moderate cutoff based on the selected items by CTQ-Short-Form.

[b] Severe cutoff based on the selected items by CTQ-Short-Form.

[c] Includes emotional neglect.

Table 3

Fixed Effects Estimates and Variance-Covariance Estimates from Piecewise Multilevel Growth Curve Models of Resting and Reactivity Levels of Cortisol

	Model 1	Model 2	Model 3
Fixed Effects, β (SE)			
Intercept	1.055 (0.082)	.795 (.168)	.560 (.214)
Time of day [a]	−.305 (.122)[c]	−.441 (.136)[d]	−.403 (.131)[d]
Resting [b] (linear)	−.108 (.017)[e]	−.087 (.030)[d]	−.087 (.030)[d]
Reactivity [b] (linear)	.116 (.030)[e]	.274 (.053)[e]	.273 (.053)[e]
Reactivity[2b] (quadratic)	−.002 (.0004)[e]	−.003 (.001)[e]	−.003 (.001)[e]
Maltreated		.357 (.202)	.353 (.230)
Maltreated by resting		−.031 (.036)	−.032 (.036)
Maltreated by reactivity		−.218 (.062)[e]	−.218 (.062)[e]
Maltreated by reactivity^2		.003 (.001)[d]	.003 (.001)[d]
Socioeconomic status			−.001 (.007)
Tanner pubic hair (Stage ≥ 4)			.512 (.163)[d]
Current MDD symptoms			−.108 (.230)
Current PTSD symptoms			−.240 (.207)
Random Parameters			
Between individual (level 2)			
Variances, σ^2_u (SE)			
Intercept/intercept	.537 (.088)[e]	.521 (.085)[e]	.453 (.075)[e]
Resting/resting	.016 (.004)[e]	.015 (.003)[e]	.015 (.003)[e]
Reactivity/reactivity	.051 (.012)[e]	.041 (.010)[e]	.041 (.010)[e]
Reactivity^2/reactivity^2	.0001 (.00001)[e]	.0001 (.00001)[e]	.0001 (.00001)[e]
Covariances, σ_u (SE)			
Intercept/resting	.032 (.013)[c]	.033 (.013)[c]	.030 (.012)[c]
Reactivity/reactivity^2	−.0006 (.0002)[e]	−.0005 (.0001)[e]	−.0005 (.0001)[e]
Within individual (level 1)			
Variance, σ^2_e (SE)			
Intercept/intercept	.056 (.006)[e]	.056 (.006)[e]	.056 (.006)[e]
−2 × log likelihood	625.034	606.388	595.695

The analysis used the natural logarithm of cortisol.

MDD, major depressive disorder; PTSD, posttraumatic stress disorder; SE, standard error.

[a] Measured in 1-hour increments.

[b] Measured in 10-minute increments.

[c] $p < .05$.

[d] $p < .01$.

$e_p < .001$.