Mental Health Outcomes in Emerging Adults Exposed to Childhood Maltreatment: The Moderating Role of Stress Reactivity

Melissa J. Hagan, Danielle S. Roubinov, Amy Kraft Mistler and Linda J. Luecken

Child Maltreat published online 11 June 2014
DOI: 10.1177/1077559514539753

Child Maltreatment
1-12

DOI: 10.1177/1077559514539753
cmx.sagepub.com
SAGE

Mental Health Outcomes in Emerging Adults Exposed to Childhood Maltreatment: The Moderating Role of Stress Reactivity

Melissa J. Hagan[1,2], Danielle S. Roubinov[1], Amy Kraft Mistler[1], and Linda J. Luecken[1]

Abstract

Childhood maltreatment is an established risk factor for varying configurations of psychological problems in emerging adulthood. The current study tested associations between childhood maltreatment, cortisol reactivity, and current mental health symptoms in emerging adulthood. Eighty-eight participants (aged 18–22) completed measures of childhood maltreatment and current internalizing and externalizing symptoms and participated in a 10-min conflict role-play task. Salivary cortisol was sampled throughout the task, and a residualized change score between baseline and peak time points was computed to capture reactivity. Results from robust regression analyses indicated that cortisol reactivity moderated the association between childhood maltreatment and mental health symptoms as hypothesized. Childhood maltreatment was related to greater internalizing problems among participants with higher cortisol reactivity, whereas maltreatment was associated with greater externalizing problems among participants who exhibited lower cortisol reactivity. Results suggest that patterns of cortisol reactivity in emerging adulthood may help elucidate mental health outcomes associated with childhood maltreatment.

Keywords

childhood maltreatment, cortisol, externalizing, internalizing, emerging adulthood

Emerging adulthood is a high-risk period for the development of mental health issues, and psychological problems evident during this period increase the risk for future psychopathology (Arnett & Tanner, 2006; Gutman & Sameroff, 2004). As a transitional stage of life characterized by challenging role changes, increased autonomy, and greater responsibility, emerging adulthood is a period in which the consequences of childhood adversity may be particularly impactful with regard to mental health. Physical and sexual abuse and neglect, in particular, are strong predictors of psychological maladjustment in youth, with emotional and physical health problems that persist across the life span (Benjet, Borges, & Medina-Mora, 2010; Clark, Caldwell, Power, & Stansfeld, 2010). Prospective and retrospective studies have found strong associations between childhood maltreatment and greater internalizing (e.g., depression, anxiety) and externalizing (e.g., aggression, antisociality) symptoms in emerging adulthood (Mersky & Topitzes, 2010) and later adulthood (Herrenkohl, Hong, Kilka, Herrenkohl, & Russo, 2013).

Despite the increased risk for maladjustment, there is substantial variability in adult mental health outcomes among those who experienced child maltreatment (e.g., Cicchetti, 2013; Luthar, Cicchetti, & Becker, 2000). At least one third of the adult survivors of abuse or neglect demonstrate remarkable resilience (McGloin & Widom, 2001; Mersky & Topitzes, 2010). Emerging adulthood is an optimal developmental period in which to examine mechanisms that may elucidate why some adults develop mental health problems secondary to childhood maltreatment and others do not. Yet, studies of childhood maltreatment conducted within a developmental psychopathology perspective have largely neglected this stage of development (Toth & Cicchetti, 2013). Emerging adults who experienced child maltreatment may maintain high levels of mental well-being as a result of ''adaptive internal systems'' that provide ''compensatory advantages'' (Mersky & Topitzes, 2010, p. 1087). Activity in the neuroendocrine stress response system is one such individual-level mechanism that may influence relations between early adversity and later mental health.

[1] Department of Psychology, Arizona State University, Tempe, AZ, USA
[2] Department of Psychiatry, University of California, San Francisco, CA, USA

Corresponding Author:
Linda J. Luecken, Department of Psychology, Arizona State University, Tempe, AZ 85287, USA.
Email: linda.luecken@asu.edu

The Hypothalamic–Pituitary–Adrenal Axis

The neuroendocrine system assumes an integral role in allostasis, the process through which physiological and behavioral change is enacted to maintain an individual's stability in response to environmental challenges (McEwen & Wingfield, 2003). During psychological stress, ascending (from the brain stem) and descending (from limbic structures) inputs activate the hypothalamic–pituitary–adrenal (HPA) axis, setting off a cascade of hormonal processes that result in the release of cortisol (Herman, Mueller, Figueiredo, & Cullinan, 2005). After the onset of a stressor, cortisol levels increase to the extent needed to facilitate responses to the threat, typically peaking between 20 and 40 min later. During stress, cortisol supports increased cardiovascular activity, stress-induced analgesia, and suppression of nonessential functions, such as growth, digestion, and reproduction (Gold & Chrousos, 2002). Cortisol also plays a key role in the consolidation of emotion-laden memory and the coordination of information processing, providing a foundation for the modulation of situation-relevant emotion and motivation (Cahill & McGaugh, 1998; de Kloet, 2010).

Individual Differences in the Cortisol Stress Response

Considerable individual differences exist in the development and functioning of the HPA axis. Essex et al. (2011) proposed that there are "episodic" components of the HPA axis that reflect responsivity to context and "stable" components that contribute to longer term patterns of physiological functioning. Responsiveness to context is illustrated by environmental influences on the calibration of the HPA axis, with both protective and adverse early experiences resulting in a wide range of alterations to the cortisol stress response (reviewed by Gunnar & Quevedo, 2007). Both exaggerated and blunted patterns of cortisol activity have been linked to family environments characterized by exposure to negative life events, conflict, abuse, and neglect (e.g., Carpenter, Shattuck, Tyrka, Geracioti, & Price, 2011; Cicchetti & Rogosch, 2001; Hagan, Luecken, Sandler, & Tein, 2010; Luecken, Kraft, & Hagan, 2009). On the other hand, over time and across development, the stress response system exhibits decreased plasticity and increased resistance to environmental influences (Boyce & Ellis, 2005). Laboratory tests with adult participants have shown consistency across time in cortisol reactivity (Cohen et al., 2000; Kirschbaum et al., 1995; van Eck, Nicolson, Berkhof, & Sulon, 1996), indicating stability in individual differences in the magnitude of the cortisol stress response (Cohen & Hamrick, 2003). Stability is also evident in the heritable components identified in diurnal cortisol patterns (Shirtcliff et al., 2012) and cortisol responses to stress (Kudielka, Hellhamer, & Wüst, 2009).

Child Maltreatment and Emerging Adult Mental Health: The Role of Stress Reactivity

The implications of cortisol dysregulation for mental health have been the focus of numerous studies. Most investigations examine direct relations between cortisol activity and mental health problems, with little regard to the life history of the individual. For example, elevated basal cortisol and greater cortisol reactivity have been associated with internalizing problems in youth (see Lopez-Duran, Kovacs, & George, 2009, for a review), while low levels of basal cortisol have been documented in the development of externalizing problems (for reviews see Alink et al., 2008; van Goozen, Fairchild, Snoek, & Harold, 2007). However, the relation between cortisol reactivity and mental health is often small in magnitude or not observed at all (Alink et al., 2008; Dienes, Hazel, & Hammen, 2013), suggesting that cortisol may play a more indirect role in mental health outcomes. For example, particular patterns of cortisol reactivity may interact with individual histories of childhood adversity to contribute to "susceptibility" (succumbing to negative consequences of exposure to adversity) or "resilience" (functioning positively in spite of adverse childhood experiences) in the long term.

The potential moderating influence of physiological functioning has been primarily explored in studies of autonomic and immune systems. Boyce et al. (1995) reported that children with heightened cardiovascular and immune reactivity raised in risky family environments exhibited the highest rate of respiratory illness, whereas history of adversity had no relation to health outcomes among those with low reactivity. Similarly, Obradovic, Bush, Stamperdahl, Adler, and Boyce (2010) found that high respiratory sinus arrhythmia (RSA) reactivity was associated with fewer externalizing symptoms in the context of low childhood adversity, but both high and low RSA reactivity was associated with more externalizing symptoms in conditions of high adversity. When skin conductance level reactivity was high, El-Sheikh, Keller, and Erath (2007) observed greater increases in internalizing symptoms among girls exposed to high marital conflict as opposed to those reared in low marital conflict environments.

Developmental psychopathology frameworks highlight dynamic interactions among the biological, cognitive, social, and environmental factors that influence psychological outcomes (Toth & Cicchetti, 2013). Stress reactivity may represent a moderating factor such that relations between childhood adversity and mental health symptoms vary with particular stress response patterns. It is also plausible that relations between cortisol reactivity and psychological functioning represent the end of an iterative cascade that begins with early adversity. That is, childhood maltreatment may be associated with a particular pattern of cortisol reactivity in emerging adulthood but also interact with cortisol reactivity to predict mental health problems.

The Current Study

The current study addresses limitations of existing research by examining the influence of cortisol reactivity on the relation between exposure to childhood maltreatment and mental health in emerging adulthood. The study uses an ecologically valid interpersonal conflict interaction task to evoke cortisol

reactivity, given that this type of stress may be particularly salient to emerging adults. We examined two models of childhood maltreatment, cortisol reactivity, and mental health problems. First, we tested our primary hypothesis that cortisol reactivity would interact with level of childhood abuse and neglect to explain the presence of mental health problems in emerging adulthood. Specifically, we expected that higher exposure to childhood maltreatment would be associated with greater internalizing problems in emerging adults who exhibited *elevated* cortisol reactivity to an interpersonal conflict situation. We also hypothesized that childhood maltreatment would be associated with greater externalizing problems but only among those who exhibited *attenuated* cortisol reactivity to the stressor task. Second, we examined the potential influence of childhood maltreatment on relations between cortisol levels and mental health. In this expanded model, it was postulated that childhood maltreatment would be associated with cortisol dysregulation and would also interact with cortisol reactivity to explain mental health symptoms in emerging adulthood.

Method

Participants

Participants included 88 emerging adults (aged 18–22; M_{age} = 18.67 years, standard deviation [*SD*] = .97) recruited from undergraduate psychology courses at a state university in the southwestern United States for a larger study of emotional and biological correlates of childhood exposure to family conflict. Participants were selected after completing a large screening survey that included the Conflict subscale of the Family Environment Scale (FES; Moos & Moos, 1994); they were invited to participate in the study if they scored in the highest or lowest quartiles on the Conflict Scale and had been raised by two continuously married biological parents who were both still living. One to three months after completing the screening survey, eligible participants were readministered the FES Conflict Scale. Those who did not score within the same high or low quartile on both administrations were removed from analyses (n = 13). Of the remaining 93 individuals, 2 did not complete the role-play task and 3 did not complete measures of child abuse and mental health, resulting in a final sample of 88 emerging adults (see Table 1).

Table 1. Sample Demographics (N = 88) and Study Variable Descriptives.

Sex, *n* (%)	
Female	44 (50)
Male	44 (50)
Participant age, *M* (*SD*); range = 18–22	18.7 (0.97)
Ethnicity, *n* (%)	
White, non-Hispanic	52 (59.1)
Hispanic	23 (26.1)
African American	6 (6.8)
Asian	4 (4.5)
Other	3 (3.4)
Family income, *n* (%)	
US$0–US$29,999	4 (4.5)
US$30,000–US$44,999	8 (9.1)
US$45,000US$59,999	11 (12.5)
US$60,000US$79,999	15 (17.1)
US$80,000–US$99,999	18 (20.5)
US$100,000+	28 (31.8)
No answer	4 (4.5)
CTQ-SF Physical/Sexual Abuse/Neglect, *M* (*SD*)	22 (5); range 18–42
ASR Internalizing, *M* (*SD*)	15 (10); range 1–42
ASR Externalizing, *M* (*SD*)	12 (7); range 1–30
Baseline cortisol, *M* (*SD*)	7.31 (4.05)
0 min posttask cortisol, *M* (*SD*)	8.73 (4.38)
20 min posttask cortisol, *M* (*SD*)	8.71 (5.13)
40 min posttask cortisol, *M* (*SD*)	7.82 (6.10)

Procedures

All procedures were approved by the university institutional review board. Data collection occurred between 2 p.m. and 6.30 p.m. on a weekday. Participants were asked to avoid alcohol for 24 hours before the lab visit and to abstain from exercising, eating, smoking, and drinking caffeinated beverages for two hours before the visit. They were queried about compliance and rescheduled if they did not comply. Following the informed consent process and a 20-min resting period, the first saliva sample (T1) was collected. Participants were then instructed on and completed the 10-min interpersonal conflict task described subsequently. Immediately after the task, participants provided a second saliva sample (T2). Twenty minutes after the end of task, a third saliva sample (T3) was collected. Following collection of the third saliva sample, participants responded to self-report questionnaires. Participants provided the fourth saliva sample (T4) 40 min after the task. Participants were compensated for their time with credits for research participation and were debriefed prior to leaving.

Role-play task. The stressful task exposed participants to a conflictual interpersonal situation chosen for its ecological relevance for emerging adults. Participants were asked to role-play a situation in which they are trying to study for an important exam, but their neighbor is playing his or her music too loud. During the video-recorded 10-min role-play, the participant was directed to stand in a designated location and at a fixed distance from his or her neighbor and ask the neighbor to turn down the music. A gender-matched research assistant played the part of the neighbor and maintained a neutral facial expression while following a scripted series of responses indicating refusal to cooperate. A separate research assistant monitored the role-play via video recording to ensure fidelity. This role-play task has been demonstrated in previous studies to result in significant cortisol and cardiovascular reactivity in young adults (Hernandez, Larkin, & Whited, 2009; Luecken et al., 2009; Semenchuk & Larkin, 1993) as well as significant emotional reactivity (Hagan, Roubinov, Purdom, & Luecken, 2014).

Measures

Child maltreatment. Participants completed the 25-item Child Trauma Questionnaire–Short Form (CTQ-SF; Bernstein et al., 2003), a retrospective measure of experiences of childhood physical, emotional, and sexual abuse as well as emotional and physical neglect. Items on the CTQ-SF are rated on a Likert-type scale with responses ranging from 1 (*never true*) to 5 (*very often true*). The CTQ-SF has been validated in clinical and nonclinical populations and has demonstrated excellent criterion-related validity among adolescents for whom therapist's ratings were available (Bernstein et al., 2003). To increase the likelihood that retrospective report of maltreatment captured abuse and neglect rather than current depressive thought patterns, five items that assessed feeling unsupported or unloved (the emotional neglect subscale) were excluded. A confirmatory factor analysis was conducted to test the hypothesis that the remaining four subscales could be combined into a single composite of maltreatment. The model fit was excellent, $\chi^2 = 2.3$, $p = .32$, RMSEA = .04, CFI = .99, and SRMSR = .04. The 20 items were summed, with higher scores indicating greater abuse and neglect. The reduced measure had adequate internal consistency ($\alpha = .81$).

Current mental health problems. Internalizing and externalizing symptoms were assessed by the Adult Self-Report (ASR; Achenbach & Rescorla, 2003). Items were rated on a 3-point scale from 0 (*not true*) to 2 (*very true or often true*); subscale items were summed to create raw scores of internalizing and externalizing problems, respectively. Higher scores correspond to a greater number of symptoms. Both scales demonstrated good internal consistency (Internalizing, $\alpha = .91$; Externalizing, $\alpha = .83$), and previous research has shown the ASR to be a valid measure of mental health problems in this age-group (Rescorla & Achenbach, 2004).

Cortisol reactivity. Saliva samples were collected using the Salivette device (Sarstedt, Rommelsdorf, Germany). Four samples were collected, pretask (T1; baseline), immediately posttask (T2), 20-min posttask (T3), and 40-min posttask (T4). At each sampling period, participants were provided with a Salivette device, which contains a roll-shaped synthetic absorbent swab in a plastic tube. Participants were instructed to transfer the absorbent swab directly from the tube into their mouth and chew for 2 min to ensure that a sufficient volume of saliva was absorbed. Once complete, the swab was spit directly back into the tube to avoid contamination. Saliva samples were frozen at 0°F and shipped to Dresden Lab Services (Dresden, Germany) for analysis. High-sensitive enzyme immunoassays were performed in duplicate to analyze free cortisol. The range of sensitivity of the assay is .4–1.0 nmol/l. The intraassay coefficient of variation was 2.5%, and the average interassay coefficient of variation for Dresden Lab Services is 6.0%.

For the evaluation of cortisol reactivity to the task, raw cortisol values were log-transformed, and a residualized change score was computed by regressing the 20-min posttask cortisol sample (T3) on the baseline cortisol sample (T1), and time since waking to account for the diurnal variation; the residuals were then used as the measure of cortisol of reactivity. The residualized change score is used as an alternative to calculating difference scores because it adjusts for the baseline level but avoids some of the reliability concerns with difference scores (MacKinnon, 2008). For the current analyses, the residualized change score is preferable to analysis of covariance because it converts two variables (T1 and T3 samples) into one variable (residualized change), simplifying the analysis of the interaction of cortisol reactivity and childhood maltreatment on mental health. Raw cortisol values were inspected for outliers and none were above normal physiological range (45 nmol/L). However, the cortisol residualized change score for one participant was >3 *SD* above the mean. When the primary analyses were repeated removing this participant, the significance and pattern of effects did not change, therefore this participant's data were retained for analyses.

Covariates. Several variables were examined for potential inclusion as covariates in the final models, including participant's age and sex, smoking status, alcohol use, medication use (including birth control), body mass index (BMI), family income, current family conflict, current perceived stress, and comorbid internalizing/externalizing problems. Smoking status was assessed with one item ("Do you currently smoke cigarettes or cigars?"). To assess medication, participants were asked to list any medications, including birth control, that they were currently taking regularly; given that only two participants reported taking more than one medication, this item was coded as 0 (*no*) or 1 (*yes*). Participants reported on their alcohol intake (average number of alcohol servings consumed in a week) using the following scale: 0 (*none*), 1 (*1–2 servings*), 2 (*3–6 servings*), 3 (*7–12 servings*), 4, (*13–20 servings*), and 5 (*>20 servings*).

Current family conflict was measured using self-report on the 9-item Conflict subscale of the Moos FES (FES-C; Moos & Moos, 1994; $\alpha = .80$); items were rated 0 (*false*) or 1 (*true*), and the scores were summed. Higher values reflect greater conflict. Perceived stress over the past month was assessed by the 10-item Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983; $\alpha = .62$). Responses ranged from 0 (*never*) to 5 (*very often*) and were summed to create a total score. Acceptable psychometric properties, including validity, have been documented for the FES (Moos & Moos, 1994) and PSS (Cohen & Janicki-Deverts, 2012).

Data Analysis

First, variables were examined for univariate normality and outliers. Inspection of normality plots as well as skewness and kurtosis values revealed nonnormal distribution of childhood maltreatment and mental health scores. Given this, correlations were estimated using Kendall's τ coefficients that

provide a more robust measure of association than Pearson's correlations (Wilcox, 2012). Correlation analyses and *t*-tests were conducted to identify covariates (i.e., variables that contribute significant variance to the outcome *or* potentially explain relations between the independent and dependent variables) to be included in the final models. Next, regression diagnostics were conducted to identify any potential influential outliers in the multiple regression equations. Although all DFFITS and DFBETAS values fell within acceptable range (<1 in small or moderate samples; Cohen, Cohen, West, & Aiken, 2003), several cases showed high externally studentized residuals (>2, as recommended by Neter, Wasserman, & Kutner, 1989) in both the internalizing and the externalizing models.

To address potential undue influence from multivariate outliers, the use of robust regression methods is recommended, as such methods can provide stable estimates in the presence of outliers (Tabachnick & Fidell, 2006). All models in the current study were tested using the SAS ROBUSTREG procedure with least trimmed squares (LTS) estimation. This method minimizes the sum of the trimmed squared residuals. The ROBUSTREG procedure computes LTS estimates that are used to detect multivariate outliers, which are then downweighted in the final weighted least square regression. The method yields a test statistic that follows a chi-square distribution.

Prior to analyses, predictors and covariates were mean centered for the purpose of reducing error related to multicollinearity, which is often introduced by the inclusion of higher order terms (Aiken & West, 1991). The first model tested the hypothesis that childhood maltreatment would interact with current cortisol reactivity to explain the number of mental health symptoms in emerging adulthood. For this model, the outcome (internalizing or externalizing problems) was regressed on cortisol reactivity (the residualized change score), childhood maltreatment, and cortisol × childhood maltreatment. Models were then retested with the inclusion of covariates. In each model, the significance of the interaction term, cortisol × maltreatment, was examined. The α level was set at .05. Significant interactions were probed following procedures outlined by Aiken and West (1991), with simple slopes computed by regressing childhood maltreatment on internalizing or externalizing problems at high (+ 1 *SD*) and low (−1 *SD*) levels of cortisol reactivity, the moderator. The statistical significance of the estimate for the relation between childhood maltreatment and internalizing or externalizing problems was then examined.

Finally, given the evidence that childhood adversity can impact the stress response system in young adulthood, we explored a more complex model in which childhood maltreatment might be associated with a particular pattern of cortisol reactivity in emerging adulthood and also interact with cortisol reactivity to explain the presence of mental health symptoms. Examination of this model followed guidelines outlined by MacKinnon (2008), in which path "a" quantifies the regression of cortisol reactivity on childhood maltreatment, and path "b" quantifies the relation of cortisol reactivity to mental health, adjusted for the effects of maltreatment. The significance of path "a" would justify testing a moderated mediation model in which childhood maltreatment is associated with cortisol reactivity and also interacts with cortisol reactivity to explain internalizing and externalizing symptoms (i.e., moderation of the "b" pathway by the independent variable; Preacher, Rucker, & Hayes, 2007).

Results

Preliminary Analyses

Demographics and descriptive statistics are included in Table 1. Correlations or *t*-tests were used to determine which variables should be controlled for in models testing whether the interaction between maltreatment and cortisol explained the presence of mental health symptoms. As shown in Table 2, externalizing and internalizing problems were significantly positively correlated. In addition, age and current family conflict were positively correlated with internalizing symptoms, and current family conflict and average weekly alcohol use was positively correlated with externalizing problems. Current family conflict was also positively correlated with childhood maltreatment. Although externalizing problems did not differ by sex, $t(86) = 1.14$, $p = .26$, females reported more internalizing problems than males ($M_F = 18.02$ vs. $M_M = 11.61$), $t(78) = 3.10$, $p = .003$. Participants who endorsed regularly smoking cigarettes or cigars reported significantly greater externalizing problems compared to nonsmokers ($M_S = 19.89$ vs. $M_{NS} = 11.15$), $t(9) = 3.14$, $p = .012$. None of the covariates examined were related to both cortisol reactivity and mental health symptoms. Although not statistically significant, there was a marginal positive correlation between BMI and cortisol reactivity, $p = .06$ (see Table 2), and females demonstrated marginally lower cortisol reactivity compared to males, $t(86) = 1.78$, $p = .08$. Therefore, we elected to be conservative by including BMI and sex as covariates in analyses exploring the association between childhood maltreatment and cortisol reactivity.

Primary Analyses

First, we tested the hypothesis that the relation between childhood maltreatment and internalizing symptoms in emerging adulthood would be moderated by cortisol reactivity. Results of these analyses are presented in Table 3. There was a significant interaction between childhood maltreatment and cortisol reactivity, which remained significant after controlling for sex, age, current externalizing problems, and current family conflict. Simple slopes were estimated at high (+1 *SD*) and low (−1 *SD*) levels of cortisol reactivity, controlling for the covariates noted earlier. As shown in Figure 1, there was a significant positive association between childhood maltreatment and internalizing problems among those who exhibited high reactivity to the task, $b = .72$, standard error (*SE*) = .16, $\chi^2 = 19.63$, $p < .01$. In contrast, among those who showed lower cortisol reactivity to the task, there was no association between child

Table 2. Kendall's τ Correlations Among Study Variables.

	1	2	3	4	5	6	7	8	9
1. CTQ-SF	1.0								
2. CORT	.01	1.0							
3. ASR-I	.27**	−.07	1.0						
4. ASR-E	.42**	−.07	.32**	1.0					
5. AGE	.06	.03	.17*	.07	1.0				
6. INCOME	−.16†	−.01	−.08	.04	−.14	1.0			
7. BMI	.10	.13†	.01	.08	.08	−.12	1.0		
8. Alcohol	.02	−.08	−.05	.21*	−.01	.06	.08	1.0	
9. FES-C	.41**	.01	.26**	.32**	.004	−.14	.13†	.09	1.0
10. PSS	.07	.07	.10	.06	.02	−.07	−.04	.10	.02

Note: CTQ = Child Trauma Questionnaire–Short Form; CORT = cortisol reactivity; ASR-I = Adult Self-Report Internalizing Scale; ASR-E = Adult Self-Report Externalizing Scale; BMI = body mass index; FES-C = Conflict Subscale of the Family Environment Scale; PSS = Perceived Stress Scale.

†$p < .10$.

**$p < .01$.

*$p < .05$.

Table 3. Robust regression Analysis With Least Trimmed Squares Estimation Predicting Internalizing Problems.

	Basic Model			Full Model		
Variable	β	χ^2	*p*	β	χ^2	*p*
Constant	12.24	297.80	<.01	7.11	6.13	.01
ASR-E				0.27	6.13	.01
Sex				3.71	8.22	.004
Age				1.91	9.71	.002
FES-C				0.17	0.28	.59
CORT	0.11	0.02	.88	0.95	2.11	.15
CTQ-SF	0.62	17.23	<.01	0.39	5.35	.02
CORT × CTQ	0.35	4.90	.03	0.34	5.98	.01

Note. ASR-E = Adult Self-Report Externalizing Scale; Sex = male (coded 0) and female (coded 1); FES-C = Conflict Subscale of the Family Environment Scale; CORT = cortisol reactivity; CTQ-SF = Child Trauma Questionnaire–Short Form.

Table 4. Robust Regression Analysis With Least Trimmed Squares Estimation Predicting Externalizing Problems.

	Basic Model			Full Model		
Variable	β	χ^2	*p*	β	χ^2	*p*
Constant	12.09	355.59	<.01	7.80	41.96	<.01
ASR-I	—	—	—	0.17	7.46	.006
Alcohol use	—	—	—	1.27	7.11	.008
Smoker status	—	—	—	3.99	3.33	.07
FES-C	—	—	—	0.09	0.09	.77
CORT	−1.22	3.31	.07	−0.82	1.93	.16
CTQ-SF	0.74	29.48	<.01	0.57	14.68	<.01
CORT × CTQ	−0.22	2.29	.13	−0.27	3.96	.047

Note. ASR-I = Adult Self-Report Internalizing Scale; Smoker status = nonsmoker (coded 0) or smoker (coded 1); FES-C = Conflict Subscale of the Family Environment Scale; CORT = cortisol reactivity; CTQ-SF = Child Trauma Questionnaire–Short Form.

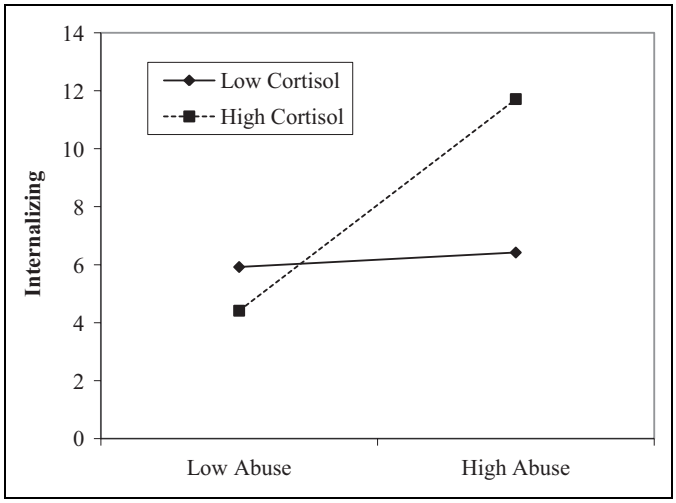

Figure 1. Relation between the childhood maltreatment composite score and the current internalizing symptoms moderated by cortisol reactivity to a psychosocial stress task.
Note. Slopes are plotted at 1 *SD* above and below the moderator mean.

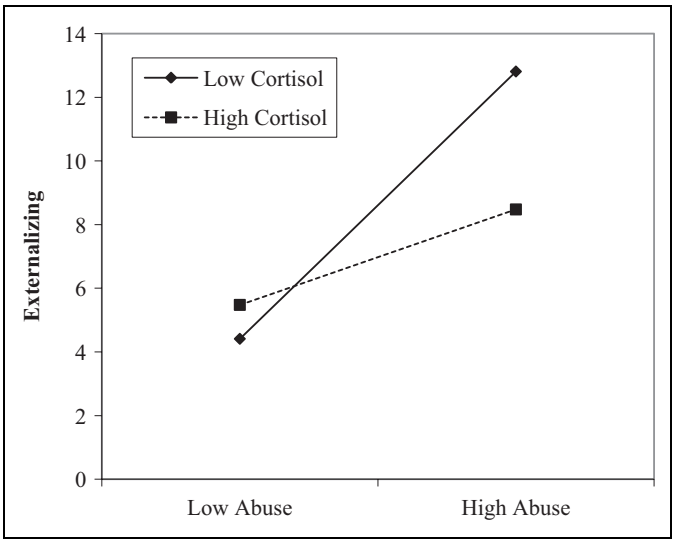

Figure 2. Relation between the childhood maltreatment composite score and the current externalizing symptoms moderated by cortisol reactivity to a psychosocial stress task.
Note. Slopes are plotted at 1 *SD* above and below the moderator mean.

maltreatment and current internalizing problems, $b = .05$, $SE = .26$, $\chi^2 = .04$, $p > .80$.

Next, we tested the hypothesis that the relation between childhood maltreatment and externalizing symptoms in emerging adulthood would be moderated by cortisol reactivity. Results of these analyses are presented in Table 4. In the basic model, there was a significant positive relation between childhood maltreatment and externalizing problems, but the interaction between childhood maltreatment and cortisol reactivity did not reach significance. After controlling for average weekly alcohol use, smoking status, current internalizing problems, and current family conflict, however, there was a significant interaction between childhood maltreatment and cortisol reactivity. Simple slopes were estimated at high (+1 *SD*) and low (−1 *SD*) levels of cortisol reactivity, controlling for the covariates noted earlier. As shown in Figure 2, there was a significant positive relation between child maltreatment and externalizing problems among those who exhibited lower cortisol reactivity, $b = .83$, $SE = .23$, $\chi^2 = 13.03$, $p < .01$. At higher levels of cortisol reactivity, the positive relation between child maltreatment and externalizing problems was not significant, $b = .30$, $SE = .16$, $\chi^2 = 3.54$, $p = .06$.

Finally, we explored the possibility that cortisol reactivity mediated the relation between childhood maltreatment and mental health in addition to interacting with childhood maltreatment. Consistent with the correlations (Table 2), there was no relation between childhood maltreatment and cortisol reactivity, controlling for sex and BMI, $b = -.004$, $SE = .02$, $\chi^2 = .04$, $p = .84$. This result did not change after controlling for the other covariates described earlier or if all covariates were removed. Further, exploratory analyses of moderation by sex

revealed no association between childhood maltreatment and cortisol reactivity by sex ($p = .38$). Given the lack of an association between childhood maltreatment and cortisol reactivity, analyses to investigate the proposed moderated mediation model were not pursued.

Discussion

Childhood maltreatment is an established risk factor for varying configurations of psychological problems in emerging adulthood. Although adversity-induced alterations to cortisol responsivity to stress have been implicated in psychopathology, evidence is far from unequivocal, especially with regard to this critical transitional life period. Little has been written about the mental health implications of high cortisol reactivity for emerging adults with childhood exposure to maltreatment. The current study found that relations between childhood maltreatment and externalizing and internalizing symptoms were moderated by cortisol reactivity to an interpersonal conflict task. These findings represent a significant contribution to a body of literature that has predominantly focused upon other developmental stages or presumed direct relations between cortisol reactivity and mental health problems.

Exaggerated Cortisol Reactivity, Childhood Maltreatment, and Internalizing Problems

In the current study, higher levels of childhood maltreatment were associated with more internalizing problems only among those who evidenced higher (i.e., 1 *SD* above the mean) cortisol reactivity to an interpersonal conflict task. Interestingly, those who reported low exposure to maltreatment but evidenced high cortisol reactivity reported the fewest internalizing problems. These findings are in line with the evolutionary-developmental model of differential susceptibility (Belsky, 1997), which posits that highly reactive individuals may have increased sensitivity to the negative effects of early adversity and the positive effects of nurturing environments. Interpretation of the current finding within the context of a differential susceptibility framework is further strengthened by the absence of an association between childhood maltreatment and internalizing problems among those exhibiting lower cortisol reactivity (Roisman et al., 2012). However, the presence of support, warmth, or other positive family qualities was not assessed in the current study and should not be inferred from a lack of maltreatment. Future research that evaluates a wider range of family characteristics will be well poised to examine evolutionary-developmental models of stress physiology and mental health.

Attenuated Cortisol Reactivity, Childhood Maltreatment, and Externalizing Problems

An extensive body of literature documents associations between childhood maltreatment and externalizing behaviors and between externalizing problems and attenuated cortisol activity, but few studies have examined these factors simultaneously (for exceptions, see Cook, Chaplin, Sinha, Tebes, & Mayes, 2012; Murray-Close, Han, Cicchetti, Crick, & Rogosch, 2008). The present investigation found that childhood maltreatment was associated with more externalizing symptoms among emerging adults who demonstrated lower cortisol reactivity (i.e., 1 *SD* below the mean). This result is consistent with research documenting increased aggression in physically abused children who exhibit attenuated cortisol activity (Scarpa, 1997) and investigations showing that psychophysiological functioning and violence exposure have additive effects on externalizing problems (e.g., Raine, 2002; Scarpa, Fikretoglu, & Luscher, 2000). Rather than reflecting differential susceptibility, the aforementioned relations are consistent with developmental dual-risk and clinical diathesis-stress models (Monroe & Simons, 1991; Sameroff, 1983). Conceptualized within these frameworks, attenuated cortisol may reflect a "vulnerability" factor that heightens the risk of maladaptive behavioral outcomes in the context of adversity. Researchers have suggested that attenuated cortisol reactivity may reflect a phenomenon in which physiological underarousal undermines inhibition, resulting in ineffective suppression of externalizing-type behaviors (Klimes-Dougan, Hastins, Granger, Usher, & Zahn-Waxler, 2001; van Goozen et al., 2007). Exposure to childhood abuse may bear a particularly strong relation to later aggression and other disruptive behavior patterns among physiologically underaroused emerging adults who are less able to inhibit or regulate their behavior.

Childhood Maltreatment and Cortisol in Emerging Adulthood

Research has demonstrated the potential for early adverse experiences to contribute to HPA axis dysregulation. Contrary to some prior studies of adults exposed to abuse earlier in life (e.g., Carpenter et al., 2007, 2009), childhood maltreatment was not directly related to cortisol reactivity in the present investigation. One possible explanation is the importance of the time period at which the maltreatment occurred. Illustratively, a large study of over 450 adolescents found that major stressful life events did not predict cortisol reactivity at age 16; however, events that occurred between ages 6 and 11 predicted higher cortisol, whereas events that occurred between ages 12 and 15 predicted lower cortisol activity (Bosch et al., 2012). Depending on the timing of the maltreatment, it is likely that some emerging adults would exhibit exaggerated cortisol response, some would exhibit a blunted cortisol response, and some may show neither pattern.

Clinical Implications of Findings

Childhood abuse is a risk factor for a wide range of mental health problems in young adulthood, including depression, anxiety, substance abuse, and aggression (Hooven, Nurius, Logan-Greene, & Thompson, 2012; Horwitz, Widom, McLaughlin, & White, 2001). Many treatment models address the role of childhood trauma in the development of mental

health disorders (e.g., Chard, 2005; Steil, Dyer, Priebe, Kleindeinst, & Bohus, 2011) but often focus on depression and anxiety, with less attention to other salient outcomes such as aggression. Trauma treatment models also lack consideration of the potential moderating effects of HPA axis reactivity and instead focus only on experiential correlates of the autonomic nervous system (the "fight-flight-freeze response") when assisting therapy clients in making sense of traumatic experiences and posttraumatic reactions (e.g., Resick, Monson, & Chard, 2008). This framework anticipates a reactive profile of physiological stress responsivity to the detriment of integrating experiential correlates of cortisol response profiles into treatment. The clinical implications of individual differences in stress physiology represent a valuable direction for future research, particularly given the current finding that childhood maltreatment may result in greater externalizing symptomatology for those with lower cortisol reactivity.

Limitations

Limitations of the study should be considered when interpreting the results. Participants were college students from intact married families. Parental divorce has been associated with mental health symptoms in adults (Afifi, Boman, Fleisher, & Sareen, 2013) and cortisol response in emerging adults (Kraft & Luecken, 2009); therefore, we chose to avoid potential confounding of divorce or parental separation. Caution should be taken in generalizing results to other populations. In addition, given the cross-sectional design, we cannot draw conclusions about when the observed patterns of physiological reactivity or mental health symptoms developed or their stability across development. As noted earlier, the lack of an association observed between childhood maltreatment and cortisol reactivity in emerging adulthood does not preclude the possibility that childhood abuse altered HPA axis functioning prior to adolescence.

Childhood maltreatment was assessed by retrospective report. Prior research suggests measurement error associated with retrospective reports does not significantly affect estimates in models that examine the long-term impact of early adversity (Fergusson, Horwood, & Boden, 2011). Nonetheless, multiple approaches were taken to reduce the likelihood that findings were a product of biased reporting. We assessed child maltreatment as a composite of specific, operationalized items capturing discrete experiences of abuse and neglect and did not include items that may have been subject to mood-congruent recall (e.g., "I felt loved"). In addition, only participants whose reports of family environment were consistent across two administrations of a measure of family environment spaced 1–3 months apart were invited to participate in the current study. Furthermore, we controlled for levels of current family conflict.

Directions for Future Research

The present investigation focused on only one aspect of the stress response. The autonomic nervous system (ANS) and neuroendocrine system are known to interact in complex ways (Sapolsky, Romero, & Munck, 2000). There is an increasing awareness in the field that assessment of these interacting systems may provide a more nuanced understanding of the relations between environmental stress and mental health (e.g., Bauer, Quas, & Boyce, 2002). ANS activity may moderate the relation between early adversity and cortisol activity as well as the relation between cortisol reactivity and mental health symptoms (Allwood, Handwerger, Kivlighan, Granger, & Stroud, 2011; El-Sheikh, Erath, Buckhalt, Granger, & Mize, 2008). Examination of ANS activity as a moderator of relations between childhood maltreatment, cortisol reactivity, and mental health was beyond the scope of this article. However, future research would benefit from incorporating cross-system measurements of stress physiology into investigations of childhood maltreatment and long-term mental health.

Conclusion

There is recognition that "the transition to adulthood in individuals with histories of child maltreatment emerges as a largely untapped area" of research, particularly in regard to studies that consider psychosocial and biological factors simultaneously rather than in isolation (Toth & Cicchetti, 2013, p. 137). Studies that attend to individual differences in neuroendocrine activity have the potential to advance the understanding of long-term mental health implications of exposure to childhood maltreatment. Identification of moderators that influence the mental health consequences of childhood abuse represents an encouraging line of research, suggesting that early experiences of adversity may not lead to poor psychological health outcomes in the absolute but under specific conditions of risk. Results support the importance of conceptualizing the role of cortisol reactivity more broadly, given that cortisol reactivity was shown to moderate rather than mediate the association between childhood maltreatment and psychological symptoms in emerging adulthood.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This research was supported by an American Heart Association award #0815544G.

References

Achenbach, T. M., & Rescorla, L. A. (2003). *Manual for ASEBA adult forms & profiles*. Burlington, VT: University of Vermont, Research Center for Children, Youth, & Families.

Afifi, T. O., Boman, J., Fleisher, W., & Sareen, J. (2009). The relationship between child abuse, parental divorce, and lifetime mental disorders and suicidality in a nationally representative adult sample. *Child Abuse & Neglect*, *33*, 139–147.

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Thousand Oaks, CA: Sage.

Alink, L. R. A., van IJzendoorn, M. H., Bakermans-Kranenburg, M., Mesman, J., Juffer, F., & Koot, H. M. (2008). Cortisol and externalizing behavior in children and adolescents: Mixed meta-analytic evidence for the inverse relation of basal cortisol and cortisol reactivity with externalizing behavior. *Developmental Psychobiology*, *50*, 427–450.

Allwood, M. A., Handwerger, K., Kivlighan, K. T., Granger, D. A., & Stroud, L. R. (2011). Direct and moderating links of salivary alpha-amylase and cortisol stress-reactivity to youth behavioral and emotional adjustment. *Biological Psychology*, *88*, 57–64.

Arnett, J. J., & Tanner, J. L. (Eds.). (2006). *Emerging adults in America: Coming of age in the 21st century*. Washington, DC: American Psychological Association.

Bauer, A. M., Quas, J. A., & Boyce, W. T. (2002). Associations between physiological reactivity and children's behavior: Advantages of a multisystem approach. *Journal of Developmental and Behavioral Pediatrics*, *23*, 102–113.

Belsky, J. (1997). Variation in susceptibility to rearing influences: An evolutionary argument. *Psychological Inquiry*, *8*, 182–186.

Benjet, C., Borges, G., & Medina-Mora, M. E. (2010). Chronic childhood adversity and onset of psychopathology during three life stages: childhood, adolescence and adulthood. *Journal of Psychiatric Research*, *44*, 732–740.

Bernstein, D. P., Stein, J. A., Newcomb, M. D., Walker, E., Pogge, D., Ahluvia, T., . . . Zule, W. (2003). Development and validation of a brief screening version of the childhood trauma questionnaire. *Child Abuse and Neglect*, *27*, 169–190.

Bosch, N. M., Riese, H., Reijneveld, S. A., Bakker, M. P., Verhulst, F. C., Ormel, J., & Oldehinkel, A. J. (2012). Timing matters: Long term effects of adversities from prenatal period up to adolescence on adolescents' cortisol stress response: The TRAILS study. *Psychoneuroendocrinology*, *37*, 1439–1447.

Boyce, W. T., Chesney, M., Alkon-Leonard, A., Tschann, J. M., Adams, S., Chesterman, B., . . . Wara, D. (1995). Psychobiologic reactivity to stress and childhood respiratory illness: Results of two prospective studies. *Psychosomatic Medicine*, *57*, 411–422.

Boyce, W. T., & Ellis, B. J. (2005). Biological sensitivity to context: I. An evolutionary-developmental theory of the origins and functions of stress reactivity. *Development and Psychopathology*, *17*, 271–301.

Cahill, L., & McGaugh, J. L. (1998). Mechanisms of emotional arousal and lasting declarative memory. *Trends in Neurosciences*, *21*, 294–299.

Carpenter, L. L., Carvalho, J. P., Tyrka, A. R., Wier, L. M., Mello, A. F., Mello, M. F., . . . Price, L. H. (2007). Decreased adrenocorticotropic hormone and cortisol responses to stress in healthy adults reporting significant childhood maltreatment. *Biological Psychiatry*, *62*, 1080–1087.

Carpenter, L. L., Shattuck, T. T., Tyrka, A. R., Geracioti, T. D., & Price, L. H. (2011). Effect of childhood physical abuse on cortisol stress response. *Psychopharmacology*, *214*, 367–375.

Carpenter, L. L., Tyrka, A. R., Ross, N. S., Khoury, L., Anderson, G. M., & Price, L. H. (2009). Effect of childhood emotional abuse and age on cortisol responsivity in adulthood. *Biological Psychiatry*, *66*, 69–75.

Chard, K. M. (2005). An evaluation of cognitive processing therapy for the treatment of posttraumatic stress disorder related to childhood sexual abuse. *Journal of Consulting and Clinical Psychology*, *73*, 965–971.

Cicchetti, D. (2013). Resilient functioning in maltreated children: Past, present, and future perspectives. *Journal of Child Psychology and Psychiatry*, *54*, 402–422.

Cicchetti, D., & Rogosch, F. A. (2001). Diverse patterns of neuroendocrine activity in maltreated children. *Development and Psychopathology*, *13*, 677–693.

Clark, C., Caldwell, T., Power, C., & Stansfeld, S. A. (2010). Does the influence of childhood adversity on psychopathology persist across the lifecourse? A 45-year prospective epidemiologic study. *Annals of Epidemiology*, *20*, 385–394.

Cohen, J., Cohen, P., West, S. G., & Aiken, L. S. (2003). *Applied multiple regression/correlation analysis for the behavioral sciences*. Mahwah, NJ: Lawrence Erlbaum.

Cohen, S., & Hamrick, N. (2003). Stable individual differences in physiological response to stressors: Implications for stress-elicited changes in immune related health. *Brain, Behavior, and Immunity*, *17*, 407–414.

Cohen, S., Hamrick, N., Rodriguez, M. S., Feldman, P. J., Rabin, B. S., & Manuck, S. B. (2000). The stability of and intercorrelations among cardiovascular, immune, endocrine, and psychological reactivity. *Annals of Behavioral Medicine*, *22*, 171–179.

Cohen, S., & Janicki-Deverts, D. (2012). Who's stressed? Distributions of psychological stress in the United States in probability samples from 1983, 2006 and 2009. *Journal of Applied Social Psychology*, *42*, 1320–1334.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, *24*, 385–396.

Cook, E. C., Chaplin, T. M., Sinha, R., Tebes, J. K., & Mayes, L. C. (2012). The stress response and adolescents' adjustment: The impact of child maltreatment. *Journal of Youth and Adolescence*, *41*, 1067–1077.

de Kloet, E. R. (2010). From vasotocin to stress and cognition. *European Journal of Pharmacology*, *626*, 18–26.

Dienes, K. A., Hazel, N. A., & Hammen, C. L. (2013). Cortisol secrection in depressed and at-risk adults. *Psychoneuroendocrinology*, *38*, 927–940.

El-Sheikh, M., Erath, S. A., Buckhalt, J. A., Granger, D. A., & Mize, J. (2008). Cortisol and children's adjustment: The moderating role of sympathetic nervous system activity. *Journal of Abnormal Child Psychology*, *36*, 601–611.

El-Sheikh, M., Keller, P. S., & Erath, S. A. (2007). Marital conflict and risk for child maladjustment over time: Skin conductance level reactivity as a vulnerability factor. *Journal of Abnormal Child Psychology*, *35*, 715–727.

Essex, M. J., Shirtcliff, E. A., Burk, L. R., Ruttle, P. L., Klein, M. H., Slattery, M. J., . . . Armstrong, J. M. (2011). Influence of early life stress on later hypothalamic-pituitary adrenal axis functioning and its covariation with mental health symptoms: A study of the allostatic process from childhood to adolescence. *Development and Psychopathology*, *23*, 1039–1058.

Fergusson, D. M., Horwood, L. J., & Boden, J. M. (2011). Structural equation modeling of repeated retrospective reports of childhood maltreatment. *International Journal of Methods in Psychiatric Research*, *20*, 93–104.

Gold, P. W., & Chrousos, G. P. (2002). Organization of the stress system and its dysregulation in melancholic and atypical depression: High vs. low CRH/NE states. *Molecular Psychiatry*, *7*, 254–275.

Gunnar, M. R., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, *58*, 145–173.

Gutman, L. M., & Sameroff, A. J. (2004). Continuities in depression from adolescence to young adulthood: Contrasting ecological influences. *Development and Psychopathology*, *16*, 967–984.

Hagan, M. J., Luecken, L. J., Sandler, I. N., & Tein, J. Y. (2010). Prospective effects of post-bereavement negative events on cortisol activity in parentally bereaved youth. *Developmental Psychobiology*, *52*, 394–400.

Hagan, M. J., Roubinov, D. S., Purdom Marreiro, C. L., & Luecken, L. J. (2014). Childhood interparental conflict and HPA axis activity in young adulthood: Examining nonlinear relations. *Developmental Psychobiology*, *56*, 871–880.

Herman, J. P., Mueller, N. K., Figueiredo, H., & Cullinan, W. E. (2005). Neurocircuit regulation of the hypothalamo-pituitary-adrenocortical stress response - an overview. In T. Steckler, N. H. Kalin & J. M. H. M. Reul (Eds.), *Handbook of stress and the brain* (pp. 405–418). San Diego, CA: Elsevier.

Hernandez, D. H., Larkin, K. T., & Whited, M. C. (2009). Cardiovascular response to interpersonal provocation and mental arithmetic among high and low hostile young adult males. *Applied Psychophysiology and Feedback*, *34*, 27–35.

Herrenkohl, T. I., Hong, S., Klika, J. B., Herrenkohl, R. C., & Russo, M. J. (2013). Developmental impacts of child abuse and neglect related to adult mental health, substance use, and physical health. *Journal of Family Violence*, *28*, 191–199.

Hooven, C., Nurius, P. S., Logan-Greene, P., & Thompson, E. A. (2012). Childhood violence exposure: Cumulative and specific effects on adult mental health. *Journal of Family Violence*, *27*, 511–522.

Horwitz, A. V., Widom, C. S., McLaughlin, J., & White, H. R. (2001). The impact of childhood abuse and neglect on adult mental health: A prospective study. *Journal of Health and Social Behavior*, *42*, 184–201.

Kirschbaum, C., Pruessner, J. C., Stone, A. A., Federenko, I. S., Gaab, J., Lintz, D., . . . Hellhammer, D. H. (1995). Persistent high cortisol responses to repeated psychological stress in a sub-population of healthy men. *Psychosomatic Medicine*, *57*, 468–474.

Klimes-Dougan, B., Hastins, P. D., Granger, D. A., Usher, B. A., & Zahn-Waxler, C. (2001). Adrenocortical activity in at-risk and normally developing adolescents: Individual differences in salivary cortisol basal levels, diurnal variation, and responses to social challenges. *Development and Psychopathology*, *13*, 695–719.

Kraft, A. J., & Luecken, L. J. (2009). Childhood parental divorce and cortisol in young adulthood: Evidence for mediation by family income. *Psychoneuroendocrinology*, *34*, 1363–1369.

Kudielka, B. M., Hellhammer, D. H., & Wust, S. (2009). Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology*, *34*, 2–18.

Lopez-Duran, N. L., Kovacs, M., & George, C. J. (2009). Hypothalamic-pituitary-adrenal axis dysregulation in depressed children and adolescents: A meta-analysis. *Psychoneuroendocrinology*, *34*, 1272–1283.

Luecken, L. J., Kraft, A. J., & Hagan, M. (2009). Negative relationships in the family-of-origin predict attenuated cortisol in emerging adults. *Hormones and Behavior*, *55*, 412–417.

Luthar, S. S., Cicchetti, D., & Becker, B. (2000). The construct of resilience: A critical evaluation and guidelines for future work. *Child Development*, *71*, 543–562.

MacKinnon, D. P. (2008). *Introduction to statistical mediation analysis*. Mahwah, NJ: Erlbaum.

McEwen, B. S., & Wingfield, J. C. (2003). The concept of allostasis in biology and biomedicine. *Hormones & Behavior*, *43*, 2–15.

McGloin, J. M., & Widom, C. S. (2001). Resilience among abused and neglected children grown up. *Development and Psychopathology*, *13*, 1021–1038.

Mersky, J. P., & Topitzes, J. (2010). Comparing early adult outcomes of maltreated and non-maltreated children, A prospective longitudinal investigation. *Children and Youth Services Review*, *32*, 1086–1096.

Monroe, S. M., & Simons, A. D. (1991). Diathesis-stress theories in the context of life stress research: Implications for the depressive disorders. *Psychological Bulletin*, *110*, 406–425.

Moos, R. H., & Moos, B. S. (1994). *Family environment scale manual*. Palo Alto, CA: Consulting Psychologists Press.

Murray-Close, D., Han, G., Cicchetti, D., Crick, N. R., & Rogosch, F. A. (2008). Neuroendocrine regulation and physical and relational aggression: The moderating roles of child maltreatment and gender. *Developmental Psychology*, *44*, 1160–1176.

Neter, J., Wasserman, W., & Kutner, M. H. (1989). *Applied linear regression models*. Homewood, IL: Irwin.

Obradovic, J., Bush, N. R., Stamperdahl, J., Adler, N. E., & Boyce, W. T. (2010). Biological sensitivity to context: The interactive effects of stress reactivity and family adversity on socio-emotional behavior and school readiness. *Child Development*, *81*, 270–289.

Preacher, K. J., Rucker, D. D., & Hayes, A. F. (2007). Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. *Multivariate Behavioral Research*, *42*, 185–227.

Raine, A. (2002). Biosocial studies of antisocial and violent behavior in children and adults: A review. *Journal of Abnormal Child Psychology*, *30*, 311–326.

Rescorla, L. A., & Achenbach, T. M. (2004). The Achenbach System of Empirically Based Assessment (ASEBA) for ages 18 to 90 years. In L. A. Rescorla & T. M. Achenbach (Eds.), *The use of psychological testing for treatment planning and outcomes assessment: Vol. 1*. Instruments for adults (3rd ed, pp. 115–152). Mahwah, NJ: Laurence Erlbaum.

Resick, P. A., Monson, C. M., & Chard, K. M. (2008). *Cognitive processing therapy: Veteran/military version*. Washington, DC: Department of Veterans' Affairs.

Roisman, G. I., Newman, D. A., Fraley, R. C., Haltigan, J. D., Groh, A. M., & Haydon, K. C. (2012). Distinguishing differential susceptibility from diathesis-stress: Recommendations for evaluating interaction effects. *Development and Psychopathology*, *24*, 389–409.

Sameroff, A. J. (1983). Developmental systems: Contexts and evolution. In P. Mussen (Ed.), *Handbook of child psychology* (Vol. 1, pp. 237–294). New York, NY: Wiley.

Sapolsky, R. M., Romero, L. M., & Munck, A. U. (2000). How do glucocorticoids influence stress responses? Integrating permissive, sup- pressive, stimulatory, and preparative actions. *Endocrine Reviews*, *21*, 55–89.

Scarpa, A. (1997). Aggression in physically abused children: The interactive role of emotion regulation. In A. Raine, P. A. Brennan, D. P. Farrington, & S. A. Mednick (Eds.), *Biosocial bases of violence* (pp. 341–344). New York, NY: Plenum.

Scarpa, A., Fikretoglu, D., & Luscher, K. (2000). Community violence exposure in a young adult sample: Psychophysiology and aggressive behavior. *Journal of Community Psychology*, *28*, 417–425.

Semenchuk, E. M., & Larkin, K. T. (1993). Behavioral and cardiovascular responses to interpersonal challenges among male offspring of essential hypertensives. *Health Psychology*, *12*, 416–419.

Shirtcliff, E. A., Allison, A. L., Armstrong, J. M., Slattery, M. J., Kalin, N. H., & Essex, M. J. (2012). Longitudinal stability and developmental properties of salivary cortisol levels and circadian rhythms from childhood to adolescence. *Developmental Psychobiology*, *54*, 493–502.

Steil, R., Dyer, A., Priebe, K., Kleindienst, N., & Bohus, M. (2011). Dialectical behavior therapy for posttraumatic stress disorder related to childhood sexual abuse: A pilot study of an intensive residential treatment program. *Journal of Traumatic Stress*, *24*, 102–106.

Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics*. Boston, MA: Pearson/Allyn & Bacon.

Toth, S. L., & Cicchetti, D. (2013). A developmental psychopathology perspective on child maltreatment. *Child Maltreatment*, *18*, 135–139.

van Eck, M. M. M., Nicolson, N. A., Berkhof, H., & Sulon, J. (1996). Individual differences in cortisol responses to a laboratory speech task and their relationship to responses to stressful daily events. *Biological Psychology*, *43*, 69–84.

van Goozen, S. H. M., Fairchild, G., Snoek, H., & Harold, G. T. (2007). The evidence for a neurobiological model of childhood antisocial behavior. *Psychological Bulletin*, *133*, 149–182.

Wilcox, R. (2012). *Introduction to robust estimation and hypothesis testing*. San Diego, CA: Academic Press.