Hormones and Behavior 55 (2009) 412–417

Contents lists available at ScienceDirect

Hormones and Behavior

journal homepage: www.elsevier.com/locate/yhbeh

Negative relationships in the family-of-origin predict attenuated cortisol in emerging adults

Linda J. Luecken *, Amy Kraft, Melissa J. Hagan

Department of Psychology, Arizona State University, Tempe, AZ 85287, USA

ARTICLE INFO

Article history:
Received 15 June 2008
Revised 23 December 2008
Accepted 31 December 2008
Available online 20 January 2009

Keyword:
Childhood family cortisol stress

ABSTRACT

Negative childhood family environments have been associated with stress-related physical and psychological health consequences across the lifespan. The present study examined the relation between adverse relationships in the family of origin and physiological stress response, as measured by salivary cortisol, in emerging adulthood. Seventy-six university students (age range = 18–22) selected from intact married families-of-origin characterized by either negative (n = 39) or positive (n = 37) relationship quality engaged in a challenging role play task. Results from multilevel models indicated that those from negative families exhibited significantly lower salivary cortisol across the task than those from positive families. This relation did not change in strength or direction after controlling for experiences with abuse or recent anxiety or depressive symptoms. These findings suggest the significance of early family relationships on the long-term activity of the HPA axis.

A considerable body of research supports an association between high-risk childhood family characteristics and negative physical and mental health outcomes throughout the life span. Family discord has been associated with the development of internalizing and externalizing symptoms in children and depression, anxiety, and poorer self-image in adolescents as well as adults (Burns and Dunlop, 2002; Kot and Shoemaker, 1999; Margolin et al., 2001). In addition, several studies have shown that individuals raised in high-risk family environments are at increased life-time risk for a wide range of behavioral and physical health related outcomes, including sleep disturbances, obesity, alcoholism, smoking initiation and prevalence, sexual disorders, somatic symptoms, chronic pain disorders, asthma, autoimmune disorders, chronic obstructive pulmonary disease, chronic bronchitis and emphysema, high blood pressure, and heart disease (Anda et al., 2006, 2008; Dong et al., 2004; Dube et al., 2002; Felitti et al., 1998; Lundberg, 1993; Margolin et al., 2001; Mechanic and Hansell, 1989; Repetti et al., 2002; Weidner et al., 1992).

It has been theorized that the physical and mental health consequences of exposure to family-of-origin adversity are co-morbid outcomes of common underlying biological processes, including dysregulation of the hypothalamic–pituitary–adrenal (HPA) stress system (Luecken and Lemery, 2004; Repetti et al., 2002; Troxel and Matthews, 2004). One of the most commonly studied components of the HPA axis is the glucocorticoid hormone cortisol. Quick and responsive cortisol secretion is necessary to adaptively respond to challenge. Exposure to chronic and/or acute stress can interfere with HPA axis activity, resulting in cortisol levels that are either too high or too low to adequately prepare the individual to meet situational demands (McEwen and Wingfield, 2003). Exposure to psychosocial stress can be especially harmful during childhood when physiological regulatory mechanisms are still developing (Gunnar and Quevedo, 2007). Anda et al. (2006) outline a strong case for the causal nature of childhood family adversity in the long-term development of psychological and physical disorder, articulating neuroendocrine dysregulation as a plausible pathway for the relation.

Exposure to chronic family adversity during childhood has been linked to both exaggerated cortisol activity (increased and prolonged cortisol secretion) and attenuated cortisol activity (evidenced by lower basal levels, flattened diurnal slopes, or blunted responses during challenge; DeBellis, 2002). For example, Pendry and Adam (2007) found that poorer marital functioning was associated with elevated basal cortisol in kindergarten-aged children and adolescents. In parentally-bereaved young adults, lower caring from the surviving parent was associated with exaggerated cortisol response to a challenge task (Luecken, 2000). In contrast, Granger et al. (1998) reported an association between higher levels of family conflict and lower levels of cortisol (measured prior to a conflict task) in 5–11 year old children. Attenuated cortisol reactivity has also been documented in kindergarten children exposed to high levels of interparental conflict (Davies et al., 2007). In young men age 18–37, a lifetime history of adverse life events (emotional abuse and neglect, physical abuse, sexual abuse, psychopathology of parents, parental divorce, and threat by disease), at least one of which occurred before the participant was 18, was associated with a blunted cortisol stress response (Elzinga et al., 2008).

* Corresponding author. Fax: +1 480 965 8544.
E-mail address: Luecken@asu.edu (L.J. Luecken).

0018-506X/$ – see front matter © 2009 Elsevier Inc. All rights reserved.
doi:10.1016/j.yhbeh.2008.12.007

The patterns of cortisol dysregulations associated with childhood adversity are diverse and research has yet to identify when hypercortisolism versus hypocortisolism is likely to develop. A number of factors have been theorized to influence how the experience of early adversity may contribute towards cortisol dysregulation. Chronicity of exposure to stress during childhood may be an important factor contributing to the pattern of cortisol dysregulation. Exaggerated cortisol secretion may be apparent shortly following trauma, but over time, attenuated cortisol secretion is theorized to develop as a protective mechanism against over-exposure to cortisol (DeBellis, 2002; Tarullo and Gunnar, 2006). The type of adversity experienced may also be relevant in predicting the pattern of dysregulation (Tyrka et al., 2008). For example, Carpenter et al. (2007) reported that sexual abuse during childhood was associated with elevated cortisol stress response in adulthood, whereas emotional neglect was associated with attenuated cortisol. The presence of psychopathology (typically PTSD) may also be important to consider when examining the complicated neurobiological sequelae of adverse childhood experiences (Pfeffer et al., 2007; Gunnar and Donzella, 2002; Heim et al., 2001). For example, adult women with PTSD related to childhood sexual abuse had afternoon hypocortisolemia relative to nonabused women and abused women without PTSD (Bremner et al., 2007). However, several studies have reported that the presence of less severe psychological distress does not explain the relation between early adversity and later life cortisol dysregulations (Elzinga et al., 2008; Carpenter et al., 2007; Luecken and Appelhans, 2006; Tyrka et al., 2008).

Although the literature has tended to focus on the health risks of prolonged elevated cortisol levels, suppressed cortisol activity has also been associated with a number of mental and physical health problems, including depression, post-traumatic stress disorder, internalizing and externalizing disorders, fibromyalgia, chronic fatigue syndrome, rheumatoid arthritis, asthma, and somatoform disorders (Heim et al., 2000; Raison and Miller, 2003). Thus, further understanding contextual predictors of the diverse patterns of cortisol dysregulation associated with adverse early life experiences is needed in the field.

Although it is clear that cortisol dysregulation has negative consequences for physical and mental health over the lifespan, research examining the effects of a negative family environment on HPA activity has primarily focused on children and adolescents, or has considered a very wide age range of adults, with no attention paid to developmental periods across the lifespan. The current study focuses attention on the effects of childhood relationship adversity on neuroendocrine activity during the developmental stage between adolescence and adulthood, when individuals are typically transitioning from dependence on parents to full autonomy. The term "emerging adulthood" has appeared relatively recently in the literature to describe this developmental period, roughly 18–25 years of age (Arnett, 2000). Although most navigate it well, emerging adulthood can be a highly stressful time, with increasing risk for stress-related disorders and the emergence of psychopathology (Arnett, 2007; Masten et al., 2004; Romer and Walker, 2007). Emerging adulthood is conceptualized as a time when individual trajectories of health become more firmly established. Thus, it is particularly important to understand the processes by which early life experiences can influence hormonal regulation in this transitional period. Little is known about how cortisol activity in this age group relates to negative family relationships experienced earlier in life, however a recent study that included adult children of divorce (ages 21–25), found that higher interparental conflict in the family-of-origin predicted attenuated cortisol response to the CRH stimulation test (Bloch et al., 2007).

While most studies have focused on neuroendocrine dysregulation associated with significant childhood maltreatment (e.g., sexual abuse), the current study evaluates cortisol levels and reactivity in emerging adults as a function of their exposure to childhood relationship adversity in the form of high conflict, low cohesion, and low expressiveness in their families-of-origin. Although little has been studied regarding the influence of expressiveness in the childhood family, maternal responsiveness has been related to secure attachment and lower baseline cortisol in infants (Gunnar et al., 1996), and verbal and nonverbal displays of affection have been shown to have a stress-buffering effect on cortisol in adults (Floyd et al., 2007). It is increasingly recognized that caregivers play a critical role in the development of children's biological stress regulation. It is theorized that warm and affectionate parenting during childhood helps children develop self-soothing and self-regulatory skills associated with well-regulated biological stress response systems, and children who lack caring and responsive relationships are at risk of developing lasting neurobiological dysregulation (Gunnar and Quevedo, 2007; Luecken and Lemery, 2004; Repetti et al., 2002).

For the current study, a role-play task was used to investigate how past family relationship experiences influence cortisol responses to current socially challenging interactions. It was predicted that compared to participants reporting more positive relationships, participants reporting negative family relationships would exhibit attenuated cortisol levels and diminished reactivity during the challenging role-play task, and that this relation would be independent of reports of sexual or physical abuse. Current theories suggest that psychological distress may partially explain the effects of reported childhood adversity on HPA function (Repetti et al., 2002; Troxel and Matthews, 2004); therefore current symptoms of depression and anxiety were evaluated as mediators of cortisol dysregulation.

Methods

Participants

Recruitment and selection criteria

Participants included 76 students recruited from Introductory Psychology classes after completing a large screening survey that included the Family Relationships subscales (FR; conflict, cohesion, expressiveness) of the *Moos Family Environment Scale* (FES; Moos and Moos, 1994). Respondents were asked to complete the FES in reference to their family environment prior to age 16. The scale was scored such that higher scores reflect more positive relationships (Cronbach's α=.90). Emerging adults (age range=18–22) raised in continuously married families by both biological parents and who scored in the highest or lowest quartiles of FR on the screening survey were invited to participate. Respondents who experienced early parental death or separation, or whose parents had ever divorced were not eligible. Eighty-one potential participants were invited to a lab session one to three months after completing the screening survey, at which time they again completed the FES. Only participants who scored within the same high or low quartile on both administrations were included in analyses (n=76; 39 from the lowest quartile and 37 from the highest quartile). Test–retest reliability was high, R=.91. Sample characteristics are displayed in Table 1.

Procedure

Participation occurred in the afternoon, between 1 and 5 PM, Monday–Friday. Nicolson (2008) recommends afternoon sampling for reactivity tasks because the cortisol response is easier to provoke and is more easily distinguished from background "noise" (e.g., spontaneous pulsatile episodes, normal morning declines in basal levels). Participants were asked to refrain from use of alcohol the night before participation, cold medication the day of

Table 1
Sample demographics

	Full sample	Negative relationships	Positive relationships
Age (M, SD)	18.9 (.97)	18.8 (.90)	19.1 (1.1)
Gender (*n*)			
Male	39	19	20
Female	37	20	17
Ethnicity (*n*)			
Hispanic	13	7	6
Anglo/Caucasian	57	28	29
African American	2	2	0
Asian/Pacific Islander	2	1	1
Other	2	1	1
Parental education level (*n*)*			
High school diploma	5	2	3
Some college	19	9	10
Jr College/Technical	4	0	4
College degree	29	15	14
Postgraduate degree	17	13	4
Not reported	2	0	2
Family relationships (M, SD)***	7.3 (6.7)	1.64 (3.7)	13.28 (2.7)
Sexual abuse (M, SD)	0.55 (2.1)	0.64 (2.5)	0.53 (1.9)
Physical abuse (M, SD)**	6.67 (3.2)	7.46 (3.7)	5.64 (1.5)
Anxiety (M, SD)*	8.3 (7.9)	9.4 (8.2)	6.3 (5.6)
Depression (M, SD)**	8.5 (6.0)	10.1 (6.0)	6.1 (4.2)

* $p<.10$.
** $p<.01$.
*** $p<.001$.

participation, and caffeine, energy drinks, eating, smoking, or exercise for at least 2 h prior to participation. Compliance was queried prior to participation, and those who did not comply were rescheduled. After providing informed consent and measuring height and weight, participants rested for 15 min, after which time the first saliva sample was taken for determination of cortisol. Participants then were instructed on and completed a 10-minute role-play task, after which 3 more saliva samples were collected: immediately after the task, 20 min after the task, and 40 min after the task. Participants completed questionnaires after the role-play, including current psychological distress assessed with the *Beck Anxiety Inventory* (Beck, 1990; α=.91) and the *Beck Depression Inventory II* (Beck, 1996; α=.84). Self-reported experiences of sexual or physical abuse during childhood were assessed with the *Childhood Trauma Questionnaire* (CTQ; Bernstein et al., 1994; sexual abuse α=.83; physical abuse α=.80), a self-report retrospective measure of abusive treatment during childhood that has shown strong evidence of reliability and validity (Bernstein et al., 2003).

Role-play task

The role-play task was designed to reflect a "real-life" interpersonal stressor, resulting in more diverse emotional and behavioral responses than traditional laboratory stressors such as mental arithmetic (e.g., Waldstein et al., 1998). For 10 min, participants role-played a challenging interpersonal situation (requesting a neighbor to turn down loud music so he/she can study for an important exam) with a same-sex research assistant. The interaction was videotaped, and the research assistant maintained a neutral expression and posture while following an ordered series of scripted responses indicating a refusal to cooperate.

Cortisol sampling

Four saliva samples were collected immediately before and at 0, 20, and 40 min after the role-play task. Samples were obtained with the Salivette device (Sarstedt, Rommelsdorf, Germany), and were stored frozen at 0 °F for 1–3 months before being shipped on dry ice to Salimetrics (State College, PA) for analysis of free cortisol using high-sensitive enzyme immunoassay. The test has a range of sensitivity from .007 to 1.8 μg/dl, and average intra- and inter-assay coefficients of variation 4.13% and 8.89%. Cortisol values were log-transformed to correct for deviations from normality. However, graphical display of the data uses non-transformed values for ease of interpretation.

Data analysis

Multilevel Linear Modeling was used to evaluate group differences in cortisol response to the role-play task. The data were modeled using the SPSS MIXED procedure, with the repeated cortisol measures forming the within-person dimension. Within-person cortisol sample order (1, 2, 3, or 4) and the squared sample order term were included to model the pattern of responses over time. Early family group served as the between-persons dimension, coded with the negative relationship group assigned a value of '0', and the positive relationship group coded as '1'. Covariates included gender, parental education level, and the time of day of sampling.

Mediation analyses were conducted following the methods of MacKinnon (2008). Briefly, evidence of mediation requires a significant relation between the independent variable (family relationship quality) and the proposed mediator (anxiety and depressive symptoms), as well as a significant relation between the mediator and the dependent variable (cortisol) after adjusting for family relationship quality. Methods for testing the significance of the mediated effect are outlined in MacKinnon (2008).

Table 2
Zero-order correlations

	1.	2.	3.	4.	5.	6.	7.	8.
1. Family relationships	–							
2. Sexual abuse	.041	–						
3. Physical abuse	−.40**	−.02	–					
4. Anxiety symptoms	−.29*	−.07	.33**	–				
5. Depressive symptoms	−.46**	−.01	.45**	.58**	–			
6. Pre-task cortisol	.13	−.27*	−.29*	−.08	−.11	–		
7. 0-minute post cortisol	.14	−.19	−.15	−.12	−.16	.71**	–	
8. 20-minute post cortisol	.20	−.18	−.08	−.13	−.09	.58**	.86**	–
9. 40-minute post cortisol	.21	−.19	−.11	−.12	−.07	.50**	.67**	.88**

Cortisol values are non-transformed and unadjusted for covariates.
* $p<.05$.
** $p<.01$.

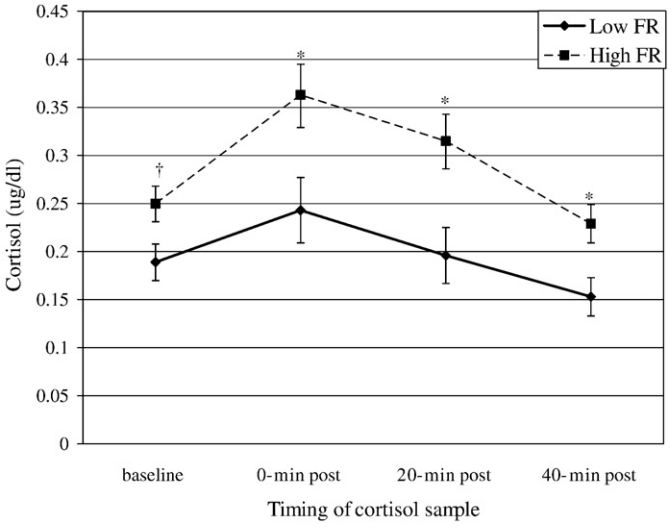

Fig. 1. Family relationship group difference in cortisol. Error bars represent standard errors of the means; cortisol values are non-transformed and adjusted for time of day, gender, and parental education; "FR" = family relationship quality. *$p<.05$; †$p=.06$.

Results

Preliminary group comparisons

Zero-order correlations between study variables of substantive interest are provided in Table 2. The family groups were compared for equivalence on demographic variables and covariates potentially associated with cortisol levels. Chi-square and *t*-tests found no group differences in gender (p=.73), ethnicity (p=.72), family income (p=.36), age (p=.29), body mass index (BMI; p=.30), waist/hip ratio (p=.60), hormonal contraceptive use (p=.42), use of medications (p=.38), or smoking status (p=.21). There was a trend towards higher parental education in the negative family group, $t(72)=1.9$, p=.067. On the day of testing, the groups did not differ on the time that they awoke (p=.35), the time of day of testing (p=.89), the time of their last meal (p=.63), the number of cigarettes smoked (p=.46), or caffeine/energy drink consumption (p=.39).

Group differences in cortisol response to the task

The hypothesis was evaluated that the negative family group would exhibit an attenuated cortisol response to the role-play task relative to the positive family group. The main effect of family group was significant, β=0.134; 95% Confidence Interval [CI], 0.032–0.236; $F(1,62)=6.95$, p=.011; Cohen's d=.67, a medium-sized effect according to Cohen's (1988) criteria. The negative family group showed significantly lower cortisol across the task (see Fig. 1). Although a significant curvilinear component was evident across the sample, the magnitude of cortisol reactivity (baseline to first post-task, p=.44) or recovery (baseline to final sample, p=.62) did not differ by family group.

The main effect of family group remained significant after controlling for potential covariates including BMI, age, waist-hip ratio, family income, smoking status, caffeine or energy drinks, the time they awoke that day, the time of their last meal, medication use, and hormonal contraceptive use. Univariate analyses predicting cortisol at each sampling time separately found that the groups significantly differed in cortisol at all time points except for baseline (baseline p=.06; immediately post-task p=.02; 20 min post-task p=.006; 40 min post-task p=.007).

Physical and sexual abuse

The family groups did not differ on reports of sexual abuse (p=.83), but the negative family group reported significantly higher physical abuse, $t(71)=2.7$, p=.009, than the positive family group. Across the sample, higher reports of sexual abuse were associated with lower cortisol, $\beta=-0.023$, $F(1,67)=4.09$, p=.047. Similarly, physical abuse was associated with lower cortisol across the sample, $\beta=-0.017$, $F(1,67)=4.10$, p=.047. However, when sexual and physical abuse were included in the model, the relation between family group and cortisol remained significant, β=0.122; 95% Confidence Interval [CI], 0.014–0.229, $F(1,60)=5.10$, p=.028, Cohen's d=.58. As a final test, participants who scored more than 2 SD above the mean on either physical or sexual abuse (n=9; 5 from the negative relationship group) were removed from analysis. Family group remained a significant predictor of cortisol, β=0.130; 95% Confidence Interval [CI], 0.020–0.241, $F(1,54)=5.57$, p=.022, Cohen's d=.64.

Mediation by current emotional distress

Next, analyses were conducted to evaluate if current anxiety or depressive symptoms explained the family group difference in cortisol levels. The negative family relationship group reported significantly more symptoms of depression, $t(72)=3.35$, p=.001, d=.79, and showed a trend towards higher anxiety, $t(74)=1.92$, p=.059, d=.45. However, multilevel modeling revealed that neither depressive (p=.23) nor anxiety symptoms (p=.45) were associated with cortisol levels. Further, family group remained significantly associated with cortisol after controlling for anxiety and depressive symptoms, β=0.139; 95% Confidence Interval [CI], 0.026–0.251; p=.017; Cohen's d=.63. Therefore current anxiety and depressive symptoms were not further evaluated as mediators of the group difference in cortisol.

Discussion

The current findings extend previous research with children and adolescents by demonstrating that neuroendocrine dysregulation in emerging adults is associated with negative relationships in the family-of-origin, potentially increasing the risk of physical and mental health disorders across the lifespan. As predicted, results suggested attenuated cortisol in those exposed to childhood families characterized as high in conflict, low in cohesion, and low in expressiveness relative to participants reporting more positive early family relationships. The family group difference remained significant after controlling for reports of sexual and physical abuse in the family of origin, as well as current anxiety and depressive symptoms. Further, the family group difference remained after removing participants reporting higher levels of abuse. The family groups did not differ on the magnitude of reactivity to the task, suggesting that results should be interpreted as reflecting group differences in overall levels of afternoon cortisol rather than stress reactivity.

While it has been clearly established that children raised in adverse family environments are at increased risk of developing various forms of psychological and medical problems, the present study examined an important pathway by which this process may occur: dysregulation of physiological stress response systems. The current findings highlight the potential long-term physiological impact of growing up in a negative family environment. Consistent with the literature on the development of cortisol dysregulation, the current results can be interpreted as supporting the theory that chronic and exaggerated physiological stress responses associated with stressful childhood experiences can attenuate over time (DeBellis, 2002). More specifically, adaptation to chronic stress and potential overexposure to glucocorticoids may result in lower cortisol levels, as were found in the present study.

Unlike previous studies focused on childhood sexual abuse or other forms of severe maltreatment, it is notable that the current findings suggest cortisol dysregulation associated with less severe childhood family adversity. In fact, the impact of negative relationships within the family of origin on cortisol remained after statistically controlling for self-reported sexual or physical abuse, and after removing from analysis any participants reporting above average experiences of abuse. These exploratory analyses suggest that exposure to childhood family adversity does not have to be severe in magnitude to affect long-term cortisol regulation.

Symptoms of anxiety or depression did not predict cortisol levels or the magnitude of reactivity to the role-play task, and they did not explain the relation between difficult family-of-origin relationships and cortisol levels in emerging adulthood. Although researchers have theorized that current psychological distress might partially explain neuroendocrine dysregulations associated with childhood adversity (e.g., Repetti et al., 2002; Luecken and Lemery, 2004), and research suggests that psychopathology such as PTSD may moderate the neuroendocrine impact of childhood maltreatment (e.g., Bremner et al., 2007), there is little empirical evidence to support this assumption for non-clinical levels of distress. Previous studies have also found that recent nonclinical levels of distress did not explain cortisol differences associated with early family disruptions (Elzinga et al., 2008; Carpenter et al., 2007; Luecken and Appelhans, 2006; Tyrka et al., 2008). Clinical diagnoses of current and lifetime psychopathology were not obtained for the present study,

therefore affective disorder cannot be ruled out as a mediator or moderator.

The current findings suggest that mechanisms other than recent emotional distress may play a significant role in the impact of early family relationships on cortisol in emerging adulthood. For example, those from negative family environments may have used a coping style of emotional disengagement from the overall lab experience, potentially resulting in lower overall cortisol levels. However, there was not a group difference in the magnitude of reactivity to the task, suggesting a similar neurobiological impact of the task for both groups. It would be valuable for future research to evaluate other potential mediators or moderators of the relation between family relationships and long-term physiological or physical health outcomes. Factors that have received theoretical attention include health behaviors, emotion regulation skills, cognitive appraisals, coping strategies, and genetic influences (Luecken et al., 2006; Repetti et al., 2002; Troxel and Matthews, 2004). It would also be valuable to consider the impact of childhood family experiences on other measures of neurobiological activity (e.g., serotonin, norepinephrine) and how effects on these biological systems might interact to predict long-term health outcomes. In short, the current findings suggest that some aspect of perceived childhood relationship quality influences current cortisol activity, an intriguing finding worthy of future studies that delve further into the potential underlying biological, behavioral, or psychological mechanisms of action.

The implications of the current findings are subject to several study limitations. The sample included students at a large public university. Although we controlled for family income and parental education level, findings may underestimate the potential HPA impact for individuals with fewer socioeconomic resources. The interpretation of the results is consistent with current empirical and theoretical literature on cortisol dysregulation associated with childhood adversity, however it is possible that individuals from more positive families were exhibiting exaggerated cortisol levels for reasons that were not measured (e.g., stress of separation from their parents). This possibility seems unlikely given that those from more negative families reported increased depressive symptoms and a near-significant increase in anxiety levels. Further, the relation between family group and cortisol was not explained by current anxiety or depressive symptoms.

The family relationship measure used to categorize participants into groups assessed overall experiences in the family, and successfully distinguished levels of cortisol between the groups. Assessments of specific family relationships, including interparental, parent–child, and siblings may reveal which relationships generate the most long-term risk. Participants in the current sample were from intact married families. Future research may wish to consider divorced families as well as assessing for changes over time in conflict or the quality of family relationships. Further examining the specific qualities of negative family environments that are the most stressful and also the most amenable to change will be valuable for the development of prevention or intervention efforts with high-risk children.

Participants were asked to retrospectively recall experiences of family relationships. A common concern with the use of retrospective reports is that current emotional state may bias recall. It is notable that high test–retest reliability was found (R=.91), and that current depressive and anxiety symptoms did not significantly explain or alter the relation between family experiences and cortisol. Experiences of abuse were also retrospectively self-reported. However, the CTQ is a well-validated retrospective measure of childhood abuse, shown to have measurement invariance of its factor structure across clinical and community samples, and criterion-related validity established through corroboration with therapist ratings and information from sources such as clinical records, child welfare investigations, and other informants (Bernstein et al., 2003). Self-reports of current anxiety and depressive symptoms were obtained at a single assessment time. Although the BDI and BAI are well-validated measures, they only assess symptoms in the last 2 weeks. A measure of longer-lasting or more severe distress was not available in this study but may provide further insight into the relation between family relationships and cortisol levels, particularly in clinical samples.

In conclusion, the current study indicates that emerging adults who perceive negative family-of-origin relationships exhibit attenuated afternoon cortisol levels, across a challenging interpersonal task, and over and above experiences of abuse and current symptoms of depression or anxiety. Dysregulation of physiological stress response systems, including the HPA axis, has been associated with numerous short- and long-term health risks. Thus, the current study's results identify a potentially important risk factor for lifespan health problems. Findings suggest that uncovering the most stressful components of growing up in an adverse family environment and their lasting physiological impact can provide options for intervention that have long-term benefits for the psychological and physical health of at-risk children.

Acknowledgment

This research was supported by NIMH R03 MH069804-1 Luecken (PI).

References

Anda, R.F., Felitti, V.J., Bremner, J.D., Walker, J.D., Whitfield, C., Perry, B.D., Dube, S.R., Giles, W.H., 2006. The enduring effects of abuse and related adverse experiences in childhood: a convergence of evidence from neurobiology and epidemiology. Eur. Arch. Psychiatry Clin. Neurosci. 256, 174–186.

Anda, R.F., Brown, D.W., Dube, S.R., Bremner, J.D., Felitti, V.J., Giles, W.H. (2008). Adverse childhood experiences and chronic obstructive pulmonary disease in adults. Am. J. Prev. Med. 34(5), 396–403.

Arnett, J.J., 2000. Emerging adulthood: a theory of development from the late teens through the twenties. Am. Psychol. 55 (5), 469–480.

Arnett, J.J., 2007. Suffering, selfish, slackers? Myths and reality about emerging adults. J. Youth Adol. 36, 23–29.

Beck, A.T., 1990. The Beck Anxiety Inventory. Harcourt Brace & Co, San Antonio.

Beck, A.T., 1996. The Beck Depression Inventory II. Harcourt Brace & Co., San Antonio.

Bernstein, D.P., Fink, L., Handelsman, L., Foote, J., Lovejoy, M., Wenzel, K., Sapareto, E., Ruggiero, J., 1994. Initial reliability and validity of a new retrospective measure of child abuse and neglect. Am. J. Psychiatry 151, 1132–1136.

Bernstein, D.P., Stein, J.A., Newcomb, M.D., Walker, E., Pogge, D., Ahluvalia, T., Stokes, J., Handelsman, L., Medrano, M., Desmond, D., Zule, W., 2003. Development and validation of a brief screening version of the Childhood Trauma Questionnaire. Child Abuse Negl. 27, 169–190.

Bloch, M., Peleg, I., Koren, D., Aner, H., Klein, E., 2007. Long-term effects of early parental loss due to divorce on the HPA axis. Horm. Behav. 51, 516–523.

Bremner, D., Vermetten, E., Kelley, M.E., 2007. Cortisol, dehydroepiandrosterone, and estradiol measured over 24 hours in women with childhood sexual abuse-related posttraumatic stress disorder. J. Nerv. Ment. Dis. 195 (11), 919–927.

Burns, A., Dunlop, R., 2002. Parental marital quality and family conflict: longitudinal effects on adolescents from divorcing and non-divorcing families. J. Divorce Remarriage 37, 57–74.

Carpenter, L.L., Carbalho, J.P., Tyrka, A.R., Wier, L.M., Mello, A.F., Mello, M.F., Anderson, G.M., Wilkinson, C.W., Price, L.W., 2007. Decreased adrenocorticotropic hormone and cortisol responses to stress in healthy adults reporting significant childhood maltreatment. Biol. Psychiatry 62, 1080–1087.

Cohen, J., 1988. Statistical Power Analysis for the Behavioral Sciences, 2nd Edition. Academic Press, New York.

Davies, P.T., Sturge-Apple, M.L., Cicchetti, D., Cummings, E.M., 2007. The role of child adrenocortical functioning in pathways between interparental conflict and child maladjustment. Dev. Psychol. 43, 918–930.

DeBellis, M.D., 2002. Developmental traumatology: a contributory mechanism for alcohol and substance use disorders. Psychoneuroendocrinol. 27, 155–170.

Dong, M., Giles, W.H., Felitti, V.J., Dube, S.R., Williams, J.E., Chapman, D.P., Anda, R.F., 2004. Insights into causal pathways for ischemic heart disease: adverse childhood experiences study. Circulation 110 (13), 1761–1766.

Dube, S.R., Anda, R.F., Felitti, V.J., Edwards, V.J., Croft, J.B., 2002. Adverse childhood experiences and personal alcohol abuse as an adult. Addictive Behav. 27, 713–725.

Elzinga, B.M., Roelofs, K., Tollenaar, M.S., Bakvis, P., van Pelt, J., Spinhoven, P., 2008. Diminished cortisol responses to psychosocial stress associated with lifetime adverse events: a study among healthy young subjects. Psychoneuroendocrinology 33, 227–237.

Felitti, V.J., Anda, R.F., Nordenberg, D., Williamson, D.F., Spitz, A.M., Edwards, V., Koss, M.P., Marks, J.S., 1998. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. Am. J. Prev. Med. 14, 245–258.

Floyd, K., Mikkelson, A.C., Tafoya, M.A., Farinelli, L., La Valley, A.G., Judd, J., Davis, K.L., Haynes, M.T., Wilson, J., 2007. Human affection exchange: XIV. Relational affection predicts resting heart rate and free cortisol secretion during acute stress. Behav. Med. 32 (4), 151–156.

Granger, D.A., Serbin, L.A., Schwartzman, A., Lehoux, P., Cooperman, J., Ikeda, S., 1998. Children's salivary cortisol, internalizing behavior problems, and family environment: results from the Concordia Longitudinal Risk Project. Int. J. Behav. Dev. 22, 707–728.

Gunnar, M.R., Donzella, B., 2002. Social regulation of the cortisol levels in early human development. Psychoneuroendocrinol. 27 (1–2), 199–220.

Gunnar, M.R., Quevedo, K., 2007. The neurobiology of stress and development. Ann. Rev. Psychol. 58, 145–173.

Gunnar, M.R., Brodersen, L., Nachmias, M., Buss, K., Rigatuso, J., 1996. Stress reactivity and attachment security. Dev. Psychobiol. 29, 191–204.

Heim, C., Ehlert, U., Hellhammer, D.H., 2000. The potential role of hypocortisolism in the pathophysiology of stress-related bodily disorders. Psychoneuroendocrinol. 25 (1), 1–35.

Heim, C., Newport, D.J., Bonsall, R., Miller, A.H., Nemeroff, C.B., 2001. Altered pituitary–adrenal axis responses to provocative challenge tests in adult survivors of childhood abuse. Am. J. Psychiatry 158 (4), 575–581.

Kot, L., Shoemaker, H.M., 1999. Children of divorce: an investigation of the developmental effects from infancy through adulthood. J. Divorce Remarriage 31, 161–178.

Luecken, L.J., 2000. Parental caring and loss during childhood and adult cortisol responses to stress. Psychol. Health 15, 841–851.

Luecken, L.J., Appelhans, B.M., 2006. Early parental loss and cortisol stress responses in young adulthood: the moderating role of family environment. Dev. Psychopathol. 18, 295–308.

Luecken, L.J., Lemery, K., 2004. Early caregiving and adult physiological stress responses. Clin. Psychol. Rev. 24, 171–191.

Luecken, L.J., Appelhans, B.M., Kraft, A.J., Brown, A., 2006. Never far from home: impact of early-life family relationships on health in adulthood. J. Soc. Pers. Relat. 23, 189–203.

Lundberg, O., 1993. The impact of childhood living conditions on illness and mortality in adulthood. Soc. Sci. Med. 36, 1047–1052.

MacKinnon, D., 2008. Introduction to Statistical Mediation Analysis. Lawrence Erlbaum Associates, New York.

Margolin, G., Oliver, P.H., Medina, A.M., 2001. Conceptual issues in understanding the relations between interparental conflict and child adjustment: integrating developmental psychopathology risk/resilience perspectives. In: Grych, J.H., Fincham, F.D. (Eds.), Interparental Conflict and Child Development: Theory, Research, and Application. Cambridge University Press, Cambridge, United Kingdom, pp. 9–38.

Masten, A.S., Burt, K.B., Roisman, G.I., Obradovic, J., Long, J.D., Tellegen, A., 2004. Resources and resilience in the transition to adulthood: continuity and change. Dev. Psychopath. 16, 1071–1094.

McEwen, B.S., Wingfield, J.C., 2003. The concept of allostasis in biology and biomedicine. Horm. Beh., 42, 2–15.

Mechanic, D., Hansell, S., 1989. Divorce, family conflict, and adolescents' well-being. J. Health Soc. Behav. 30, 105–116.

Moos, R.H., Moos, B.S., 1994. Family Environment Scale Manual. Consulting Psychologists Press, Palo Alto, CA.

Nicolson, N.C., 2008. Measurement of cortisol. In: Luecken, L.J., Gallo, L.C. (Eds.), Handbook of Physiological Research Methods in Health Psychology. Sage Publications, Thousand Oaks, CA.

Pendry, P., Adam, E.K., 2007. Associations between parents' marital functioning, maternal parenting quality, maternal emotion and child cortisol levels. Int. J. Behav. Dev. 31, 218–231.

Pfeffer, C.R., Altemus, M., Heo, M., Jian, H., 2007. Salivary cortisol and psychopathology in children bereaved by the September 11, 2001 terror attacks. Biol. Psychiatry 61 (8), 957–965.

Raison, C.L., Miller, A.H., 2003. When not enough is too much: the role of insufficient glucocorticoid signaling in the pathophysiology of stress-related disorder. Am. J. Psychiatry 160, 1554–1565.

Repetti, R.L., Taylor, S.E., Seeman, T.E., 2002. Risky families: family social environments and the mental and physical health of offspring. Psychol. Bull. 128, 330–366.

Romer, D., Walker, E.F., 2007. Adolescent Psychopathology and the Developing Brain. Oxford University Press, New York, NY.

Tarullo, A., Gunnar, M.R., 2006. Child Maltreatment and the Developing HPA Axis. In: McCarthy, M.M., deVries, G. (Eds.), Horm. Behav., 5, pp. 632–639.

Troxel, W.M., Matthews, K.A., 2004. What are the costs of marital conflict and dissolution to children's physical health? Clin. Child Fam. Psychol. Rev. 7, 29–57.

Tyrka, A.R., Wier, L., Price, L.H., Ross, N., Anderson, G.M., Wilkinson, C.W., Carpenter, L.L., 2008. Childhood parental loss and adult hypothalamic–pituitary–adrenal function. Biol. Psychiatry 63(12), 1147–1154.

Waldstein, S.R., Neumann, S.A., Burns, H.O., Maier, K.J., 1998. Role-played interpersonal interaction: ecological validity and cardiovascular reactivity. Ann. Behav. Med. 20, 302–309.

Weidner, G., Hutt, J., Connor, S.L., Mendell, N.R., 1992. Family stress and coronary risk in children. Psychosom. Med. 54, 471–479.