Brief Report

Prospective Effects of Post-Bereavement Negative Events on Cortisol Activity in Parentally Bereaved Youth

Melissa J. Hagan
Linda J. Luecken
Irwin N. Sandler
Jenn-Yun Tein
Program for Prevention Research
Department of Psychology
Arizona State University
Tempe, AZ 85287-1104
E-mail: melissa.hagan@asu.edu

ABSTRACT: *Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis has been implicated in the association between adverse childhood experiences, such as parental death, and mental and physical health problems. Recent research indicates that children who experience the death of a parent exhibit HPA axis dysfunction; however, the mechanisms underlying this association have not been explored. It is theorized that physiological dysregulation may result from exposure to stressful life events subsequent to parental death. The current study examined the prospective relations between negative events following parental death and cortisol activity in parentally bereaved youth. A greater number of post-bereavement negative events predicted significantly lower levels of cortisol activity 6 years later; this association remained significant after controlling for current externalizing symptoms and recent negative events. Results suggest that higher exposure to stressful events following childhood parental loss may result in long-term attenuated cortisol activity.* © *2010 Wiley Periodicals, Inc.* Dev Psychobiol 52: 394–400, 2010.

Keywords: *cortisol; parental loss; stress; children; adolescents*

INTRODUCTION

It is well documented that parental death during childhood and adolescence is associated with a higher risk of mental health problems throughout the life span (Kendler, Sheth, Gardner, & Prescott, 2002; Melhem, Walker, Moritz, & Brent, 2008). Emerging evidence suggests that hypothalamic–pituitary–adrenal (HPA) axis dysfunction, evidenced by hyper- or hypo-secretion of the glucocorticoid cortisol, may play a key role in the association between adverse childhood experiences, such as parental death, and psychopathology (Debellis et al., 1999; Gunnar & Quevedo, 2007). More specifically, recent research has demonstrated an association between the death of a parent during childhood and HPA axis dysregulation in the short- and long-term (e.g., Pfeffer, Altemus, Heo, & Jian, 2007; Tyrka et al., 2008). However, not all bereaved children experience HPA axis dysregulation, and the processes by which parental death may affect the HPA stress response systems of children and adolescents remain largely unexplored.

Although research has shown an association between parental loss and cortisol dysregulation, the direction of the association has been inconsistent across studies. Pfeffer et al. (2007) reported that children who lost a caregiver in the September 11, 2001 New York City terrorist attack exhibited significantly higher afternoon and evening basal cortisol levels compared to non-bereaved children. Conversely, other studies have found that children who experienced long-term separation from a parent exhibited lower levels of basal cortisol (Carlson & Earls, 1997; Flinn, Quinlan, Decker, Turner, & England, 1996). Studies conducted with adults who experienced parental death during childhood also show mixed results. Higher levels of basal cortisol (Nicolson, 2004) and increased cortisol responses to a dexamethasone/

Received 17 September 2009; Accepted 22 December 2009
Correspondence to: M. J. Hagan
Published online 19 February 2010 in Wiley InterScience (www.interscience.wiley.com). DOI 10.1002/dev.20433

corticotropin-releasing hormone (Tyrka et al., 2008) have been documented in adults who experienced early parental loss; whereas others found effects of parental loss on cortisol only in interaction with other factors such as abuse or concurrent psychological disorder (Breier et al., 1988; Luecken, 2000; Luecken & Appelhans, 2006).

According to cumulative risk theory, it is a confluence of risk factors rather than exposure to any one risk factor, such as parental death, that overwhelms the adaptive capacity of an individual and leads to dysfunction (Flouri & Kallis, 2007). In addition to the pain and stress associated with the loss itself, parentally bereaved youth face a series of stressors after the death, such as relocation of the family, economic hardship, and increased familial responsibilities (Lin, Sandler, Ayers, Wolchik, & Luecken, 2004). Although a number of studies have shown that recurrent exposure to negative life events can result in physiological dysregulation (Bevans, Cerbone, & Overstreet, 2008; Elzinga et al., 2008; Flinn, 2006), the research on the neurobiological effects of life stressors following a major traumatic event, such as parental loss, is very limited. Indeed, to our knowledge, no studies have explicitly examined the prospective relation between negative events subsequent to a major traumatic event and later physiological dysregulation in a sample of youth. The current study examined the association between negative life events (measured an average of 13 months, 24 months, and 7 years following the death) and an indicator of HPA functioning, cortisol response to conflict discussion task, in a sample of parentally bereaved adolescents and young adults 7 years following the death of a caregiver.

METHODS

Participants

Participants in the current study consisted of youth assigned to the no-treatment control condition in a randomized trial of a preventive intervention for parentally bereaved children. Families with at least one child in the age range of 8–16 years who experienced parental death between 3 and 30 months prior to the start of the study were recruited from the Phoenix, Arizona metropolitan area (for complete recruitment and eligibility details, see Sandler et al., 2003). Of the 109 youth assigned to the control condition, 102 (93.5%) participated in the 6-year follow-up, and 88 of these youth were eligible to participate in the conflict discussion task (youth were ineligible if they reported less than three verbal contacts with the caregiver in the prior month; for details see Luecken et al., in press). Out of the 88 eligible youth, 61 (69.3%) participated in a conflict discussion task and provided cortisol samples [the remainder either refused ($n = 12$) or had scheduling conflicts ($n = 15$)]. Table 1 includes demographic information on the sample.

Measures

Negative Life Events. Child experiences of negative events were assessed by parent- and child-report using the 51-item Negative Life Events Scale (NLES; Sandler et al., 2003). Negative events were assessed at four time points corresponding to the waves of the experimental trial. Past month negative events were assessed immediately prior to randomization (mean = 10 months following the death; range = 3–27 months post-death), postintervention (3 months after randomization; mean = 13 months post-death), and 11 months postintervention (mean = 24 months post-death). Negative events over the prior year were assessed 6 years post-intervention (mean = 7 years following the death). In the current study, we sought to examine negative life event exposure that a child more characteristically experiences following the bereavement period, rather than the stress associated with the period of disruption immediately following the death. At baseline, some children had experienced the death of a parent as early as 3 months prior. To allow at least 6 months following the death, the current study excluded the baseline assessment. This decision was supported by the high correlation between negative life events assessed at the second and third waves, $r(61) = .67$, $p < .0001$, compared to the correlation between baseline negative events and the composite of the two subsequent assessments of negative events, $r(61) = .36$, $p = .004$. Thus, in the current study, the NLES was administered twice during the post-bereavement period: 13 months (on average) following the death of a caregiver and again 11 months later. The NLES assesses different types of stressful events including interpersonal conflicts, changes in the child's environment, caregiver distress, and loss events (subsequent to parental death). Items were scored "1" if either parent or child reported that the event occurred and "0" if neither parent nor child endorsed the item as occurring. Scores at each of the two time points reflect the total number of events endorsed by parent or child as occurring during the prior month. A composite was computed by taking the average of the standardized scores at the two assessments (13 and 24 months post-death) to reflect the number of negative life

Table 1. Sample Characteristics

Demographic variable	*n*	% of total
Gender ($N = 61$)		
Male	39	64
Female	22	36
Ethnicity ($N = 61$)		
Hispanic	7	12
Anglo/Caucasian	38	63
African American	6	10
Native American	4	7
Other	5	8
Cause of death ($N = 60$)		
Illness	45	75
Accident	11	18
Homicide/suicide	4	7
Mean age at the time of parent's death (*SD*)	10.4 (2.5)	

events experienced in an average month during the post-bereavement period. This composite, hereafter referred to as *post-bereavement negative life events*, was used as the prospective predictor variable in the current study. Youth experiences of negative events were assessed again at the time of discussion task and cortisol sampling (an average of 7 years post-death) using an expanded version of the NLES, which included the original 51 items as well as a subset of 25 items from the Life Experiences Survey (Sarason, Johnson, & Siegel, 1978) to reflect life events that may occur in young adulthood (e.g., ''Your boy/girlfriend had a serious illness or injury''). Scores on the expanded version of the NLES reflect the total number of negative life events experienced over the prior year by youth report only, hereafter referred to as *recent negative life events*.

Current Mental Health Problems. Given prior evidence that psychopathology in parentally bereaved youth may be associated with negative life events (Li, Lutzke, Sandler, & Ayers, 1995; Lin et al., 2004) and cortisol activity (Pfeffer et al., 2007), the present study considered measures of current mental health problems as potential covariates. Current externalizing and internalizing symptoms were assessed during the 6-year follow-up to the experimental trial by self-report on the Youth Self-Report form if the participant was 14–18 years old or the Young Adult Self-Report form if the participant was over age 18 (Achenbach and Rescorla, 2001). Because adolescents and young adults completed different scales, item response theory was used with a large dataset obtained from Achenbach ($n = 800$) on the YSR/YASR to conduct an equating transformation that put the scores on a common metric (Kolen & Brennan, 1995) for the two scales. The internalizing and externalizing symptoms for this sample were then scored accordingly. Higher scores reflect a greater number of symptoms.

Procedures and Analytic Strategy

During the 6-year follow-up (an average of 7 years following parental death), youth and their caregivers participated in a conflict discussion task conducted at the participants' homes between 3:45 and 8:15 p.m. (a priori time range was 3:00–9:00 p.m.) on a weekday. The videotaped task was a 12-min standardized behavioral observation interaction in which the youth and caregiver discuss current problems common to late adolescence/young adulthood. Three issues for discussion were selected based on caregiver and youth responses to the Parent Issues Checklist (Prinz, Foster, Kent, & O'Leary, 1979), which was completed just before the discussion task. Cortisol was sampled from youth at four time points across the discussion task: prediscussion, immediately after the discussion, 15 min after the discussion, and 30 min after the discussion. Samples were obtained with the Salivette device (''plain'' variety; 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 mean intra- and inter-assay coefficients of variation 4.13% and 8.89%.

Although we were interested in both reactivity and total cortisol across participation, preliminary analyses found no evidence of cortisol reactivity to the task (i.e., an increase from baseline), therefore area under the curve with respect to ground (AUC_G) was calculated using the trapezoidal method (Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003) and served as the primary-dependent variable. Cortisol AUC_G was computed using the raw values and then log-transformed to correct for deviations from normality. Multiple linear regression was used to examine two models: (1) the prospective effect of post-bereavement negative life events (assessed as a composite of negative events reported 13 and 24 months following parental death and representing exposure to negative events in an average month during this period) on cortisol AUC_G (measured an average of 7 years following parental death) and (2) the effect of recent negative life events (the total number of events experienced over the prior year) on cortisol AUC_G.

RESULTS

Descriptive Statistics and Preliminary Analyses

Descriptive statistics of the predictors, outcome, and final covariates are included in Table 2. At the 13-month post-death assessment, children and caregivers reported an average of 12.85 ($SD = 4.82$) negative events in the past month. Twenty-four months following parental death, families reported child exposure to an average of 10.66 negative events ($SD = 5.83$) over the past month. Six years later (at the time of cortisol sampling), youth (mean age = 17.18) reported an average of 12.77 recent negative events ($SD = 7.37$) over the prior year. Raw

Table 2. Descriptive Statistics

	M	*SD*	Range
Participant age	17.18	2.15	14–22
Externalizing symptoms	3.87	.89	1.98–5.67
Internalizing symptoms	3.55	1.36	0–6.23
Negative life events 13 months post-death	12.85	4.82	0–24
Negative life events 24 months post-death	10.66	5.83	0–28
Negative life events 7 years post-death	12.77	7.37	1–35
Total cortisol output (AUC_G) 7 years post-death	.23	.15	.063–.847

values of cortisol AUC_G ranged from .063 to .846 μg/dl ($M = .23$; $SD = .15$).

As noted above, cortisol AUC_G was log-transformed and these values were used in all analyses. There was a marginally significant correlation between cortisol AUC_G and post-bereavement negative events, $r(57) = .24$, $p = .06$, however there was no correlation between cortisol and recent negative events, $r(56) = -.05$, *ns*. Zero-order correlations between the studied variables and potential covariates (hormonal contraception, smoking status, caffeine intake, yearly income, current age, gender, ethnicity, time since parental death, cause of parental death, and time of day at which cortisol was sampled) were also examined. Only participant age correlated significantly with cortisol output, $r(57) = .25$, $p = .05$. In addition, given the growing literature on the association between basal cortisol and mental health problems (Alink et al., 2008; Shirtcliff & Essex, 2008), the correlations between negative events, cortisol output, and current internalizing and externalizing symptoms were examined. Current mental health symptoms did not correlate with post-bereavement negative events; however, recent negative life events correlated significantly with both current externalizing, $r(60) = .46$, $p < .001$, and internalizing symptoms, $r(60) = .43$, $p < .001$. Although cortisol AUC_G was not correlated with current internalizing symptoms, $r(56) = -.11$, *ns*., there was a significant negative correlation between cortisol and current externalizing symptoms, $r(56) = -.27$, $p = .04$. Thus, participant age and current externalizing symptoms were included as covariates in all models, and current internalizing symptoms were included as an additional covariate in the model examining the effect of recent negative events on cortisol activity.

Finally, although time of day did not correlate with cortisol, $r(57) = -.18$, $p = .18$, cortisol secretion exhibits a normative diurnal pattern, and time of day of sampling could be a confound. Accordingly, all models were examined with time of day as a covariate (in addition to age and mental health symptoms). Inclusion of time of day did not affect the strength of the relations between negative life events and cortisol in the prospective or concurrent models, nor did it predict cortisol or account for significant model variance; therefore, it was dropped from the final models.

Primary Analyses

Table 3 includes the results of both the prospective and concurrent models, controlling for the relevant covariates. The prospective model was examined first by regressing cortisol AUC_G on post-bereavement negative events, controlling for participant age and current externalizing symptoms. There was a significant association such that a greater number of post-bereavement negative events predicted lower cortisol 6 years later, $B = -.17$, $SE = .08$, $t = -2.03$, $p = .048$. The model accounted for 19% of the variance in cortisol AUC_G, $F = 4.15$, $p = .01$; post-bereavement negative events accounted for 6% of the variance above and beyond the covariates, $F = 4.11$, $p = .048$.

Next, the effect of post-bereavement events on cortisol activity above and beyond recent negative events was examined by entering recent negative events into the model. Post-bereavement negative events remained a significant predictor of cortisol 6 years later, $B = -.17$, $SE = .09$, $t = -2.04$, $p = .046$, controlling for exposure to events over the previous year, age, and current

Table 3. Regression Models of Post-bereavement Negative Events (Prospective Model) and Recent Negative Events (Concurrent Model) Predicting Total Cortisol Output (AUC_G), Controlling for Current Externalizing Problems

	Prospective model (post-bereavement events)			Concurrent model (recent negative events)		
	B	*SE*	*t*	*B*	*SE*	*t*
Intercept	−1.98*	.68	−2.89	−1.96*	.73	−2.68
Participant age	.06	.03	1.88	.06	.04	1.67
Current externalizing	−.17*	.08	−2.07	−.19	.12	−1.67
Current internalizing	—	—	—	.006	.08	.07
R^2 (*F*)[a]	*.13 (3.93)**			*.13 (2.98)*		
Negative life events	−.17*	.08	−2.03	.003	.01	.33
R^2 (*F*)[b]	*.19 (4.15)***			*.13 (1.92)*		
R^2 change (*F*)	*.06 (4.11)**			—		

Italics are used to differentiate these R-squared statistics from the betas in the rest of the table.

*$p \leq .05$.

**$p \leq .01$.

[a]R^2 and *F* statistics for all the covariates in the model.

[b]R^2 and *F* statistics for all the covariates and negative life events in the model.

externalizing symptoms; the proportion of variance accounted for by post-bereavement negative events did not change.

The total number of recent negative events was then examined as an independent predictor of cortisol activity. As expected, given the nonsignificant correlation between recent negative events and cortisol, there was no association between cortisol AUC_G and recent negative life events, $B = .004$, $SE = .01$, $t = .29$, *ns*., controlling for participant age, externalizing symptoms, and internalizing symptoms.

DISCUSSION

The goal of the current study was to evaluate the theory that negative life events experienced subsequent to the death of a parent predict cortisol activity in a longitudinal sample of parentally bereaved youth. A significant prospective effect of post-bereavement negative life events on total cortisol output was found, such that a greater number of negative life events measured between 13 and 24 months following parental death was associated with lower cortisol secretion 7 years after the death. The prospective relation between negative events and cortisol remained significant after controlling for participant age, current externalizing symptoms, and recent negative life events. This study makes a significant contribution to the literature by demonstrating a prospective relation of exposure to negative life events and cortisol activity several years later in a population of youth exposed to a major stressful event, the death of a parent.

The inverse relation between the number of negative life events that occurred 6 years earlier and current cortisol output is consistent with the attenuated pattern of cortisol found in many children, adolescents, and adults exposed to chronic adversity earlier in life (Elzinga et al., 2008; Heim, Newport, Bonsall, Miller, & Nemeroff, 2001; Tarullo & Gunnar, 2006). Although other studies have found a positive relation between stressful experiences and cortisol activity (Bevans et al., 2008; Marin, Martin, Blackwell, Stetler, & Miller, 2007; Rao, Hammen, Ortiz, Chen, & Poland, 2008), the time between exposure to adversity and cortisol assessment in many of these studies was much shorter (e.g., a period of months). A recent meta-analysis of stress and HPA axis functioning found that chronically low levels of cortisol can appear after a substantial amount of time has passed since exposure to adversity (Miller, Chen, & Zhou, 2007). Indeed, the current study found that exposure to negative events at one developmental stage (middle childhood/early adolescence) predicted lower cortisol activity several years later (during late adolescence/young adulthood), whereas no association was found between recent negative life events and cortisol.

Limitations

The results should be considered in the context of the study's limitations. First, the lack of a previous measurement of cortisol (prior to or immediately after parental death) limits our interpretation of a cause–effect relationship between negative life events subsequent to the death and cortisol activity 6 years later. It is plausible that other variables may have influenced cortisol activity, negative events, or the relation between them. In addition, the absence of an earlier measure of cortisol precludes an evaluation of the impact of negative life events on the pattern of HPA activity across the years following the death. For example, it is not known whether stress exposure initially promoted exaggerated cortisol, which resulted in chronic attenuation over time, or if attenuated cortisol was exhibited immediately following the negative events. While the current prospective analysis demonstrates an association between negative events subsequent to parental death and neuroendocrine activity several years later, alternative explanations for this association cannot be ruled out.

Second, it was expected that the conflict discussion task would allow evaluation of cortisol reactivity as well as total cortisol output; however, reactivity to the task was not evident. Although conflict discussion tasks are commonly used to evaluate cortisol reactivity, recent studies have found that parent–child discussions may elicit a unique pattern characterized by declining cortisol (e.g., Adam, Klimes-Dougan, & Gunnar, 2007). Alternatively, anticipatory cortisol increases may have occurred or the task may not have been stressful enough to elicit reactivity. Thus, cortisol activity in the current study should be interpreted within the context of a parent–child discussion conducted in late afternoon/early evening. The data cannot provide information about the long-term impact of negative life events on basal cortisol, diurnal cortisol secretion, or cortisol activity at other times of the day. Future examinations should include additional methods of cortisol measurement (e.g., basal levels of cortisol, diurnal slope, etc.). Third, stressful events were assessed at a limited number of time points; yearly measures of stressful events over a prolonged period of time would provide a more stable estimate of the level of chronic stress experienced following parental death. Despite this potential limitation, the use of the NLES is a strength of the current study. Based on a decade of research, the NLES was constructed in collaboration with service professionals and bereaved youth, and it encompasses negative events specific to a bereavement context as well as general negative life events that can be verified

by outside observers (Haine, Ayers, Sandler, Wolchik, & Weyer, 2003). Moreover, prior research with the measure demonstrated significant relations with measures of psychological symptomatology (Li et al., 1995).

Finally, limited statistical power associated with the sample size ($N = 61$) prevented the examination of potential moderators. For example, cumulative exposure to negative events may have a stronger effect in certain populations, such as younger children or youth with a history of mental health problems. Although post hoc analyses to examine potential moderation of the association between negative events and cortisol output by factors such as age, sex, and mental health symptoms did not find significant moderation, given the small sample size, these analyses had very low power to detect moderation effects. In addition to examining these characteristics as potential moderators, future studies may want to consider how the effects of past negative life events on cortisol activity are influenced by available resources (e.g., high quality parent–child relationship, active coping) or other risk factors (e.g., high appraisal of threat associated with the negative life events). Despite these limitations, the current study has important strengths including the prospective longitudinal study design and the multi-rater assessment of negative life events. Whereas the majority of past research assessed exposure to stress retrospectively and by self-report only, assessment of exposure to negative life events in the current study included parent and child report and took place at multiple time points over a period of 6 years.

CONCLUSIONS

Previous research indicates that the psychological consequences of parental loss are associated with stressors which occur in the post-death environment (e.g., stressful life events characterized by interpersonal conflict, additional losses, environmental changes, family stress, etc.). Although a few studies have examined neurobiological functioning in youth who experienced the death of a parent (e.g., Pfeffer et al., 2007), the present study is the first to identify a significant negative association between exposure to negative life events subsequent to the death of a parent and cortisol activity several years later. This pattern is consistent with evidence from other studies indicating that high levels of exposure to stressful events can result in attenuated cortisol activity long after stress exposure has occurred. Given the association between low levels of cortisol and externalizing symptoms, the findings from the present study have important implications for future research on the physiological mechanisms underlying the development of psychopathology among parentally bereaved youth.

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