The Association Between PTSD Symptoms and Salivary Cortisol in Youth: The Role of Time Since the Trauma

Carl F. Weems
Department of Psychology, University of New Orleans, New Orleans, LA

Victor G. Carrion
Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, Stanford, CA

This study examined the direction of association between symptoms of posttraumatic stress disorder (PTSD) and cortisol levels among youth with recent and distal traumas (N = 50; mean age = 10.7 years). Each had a clinical interview for PTSD symptoms, a cortisol assessment, and the time since the child’s most recent trauma was assessed. Results indicated that the time since the most recent trauma moderated the association between cortisol and PTSD symptoms and comparisons indicated that there were significant differences in the size of the correlations across the recent and distal trauma groups. The results point to a potentially important role of the time since trauma in understanding the relationship between PTSD symptoms and cortisol.

The activity of the hypothalamic–pituitary–adrenal (HPA) axis is part of the fight–flight reaction in response to stress and fear. In particular, fear reactions are associated with elevations in the secretion of cortisol, a corticosteroid hormone produced by the adrenal cortex (See Gunnar, 2001, for a review). Such findings have suggested an important role of cortisol in stress-related problems such as posttraumatic stress disorder (PTSD). However, findings have been inconsistent. Studies have indicated, somewhat paradoxically considering the normative cortisol response, decreased basal cortisol levels in combat veterans, Holocaust survivors, and sexual abuse victims with PTSD (Boscarino, 1996; Yehuda et al., 1995). Other studies, however, have reported increased levels of cortisol in adults with PTSD (Lemieux & Coe, 1995; Maes et al., 1998). Clarifying the association between symptoms of PTSD and cortisol may help in translating basic research on the function of the HPA axis to patient populations.

Data on the function of the HPA axis in youth who experience trauma and develop PTSD symptoms is also emerging (De Bellis, 2001); however, this literature has also shown inconsistencies. For example, maltreated children diagnosed with PTSD have shown increased cortisol levels after 24-hour urinary collection compared to matched controls (De Bellis et al., 1999). Goenjian et al. (1996) evaluated adolescents 5 years after their experience of the Armenian earthquake and found that adolescents living close to the epicenter and who still had PTSD symptoms had lower basal salivary cortisol levels. De Bellis (2001) in a review of the literature on cortisol response in PTSD theorized that there may be developmental (both age related and time related) differences in the association (see also,
Weems and Carrion (2005). This view postulates that after an initial normal increased cortisol reaction, HPA axis functioning may be altered to a condition of relatively lowered basal cortisol as time passes since the traumatic event. Indeed, recent research on youth assessed for cortisol levels on admission to a Level 1 trauma center point to a positive correlation ($r = .31$) between cortisol levels and PTSD symptoms assessed at 6 weeks (Delahanty, Nugent, Christopher, & Walsh, 2005).

In this study, we sought to test the De Bellis (2001) hypothesis by examining cortisol's correlation with PTSD in two sets of youth (i.e., children who have had a recent traumatic event and those who trauma was experienced over a year ago). Drawing from De Bellis (2001) we reasoned that differences in previous findings might be due to cortisol being differentially associated with PTSD symptoms depending on the time since the traumatic event (i.e., among youth with recent traumatic events cortisol would be positively related to PTSD symptoms, but in those with distal events the association would be negative). The data for this study are from Carrion et al. (2002). In that study youth with a history of exposure to trauma and PTSD symptoms were compared to age- and gender-matched healthy control subjects. Results indicated that the clinical group demonstrated significantly elevated diurnal cortisol levels when compared to the control group (particularly for prebed levels). However, PTSD symptoms were not found to be linearly associated with cortisol levels in the trauma group. In this study, we focused on the trauma group and examined the associations in light of the De Bellis (2001) hypothesis reasoning that the time since the trauma would moderate the association between cortisol levels and PTSD symptoms.

**METHOD**

Details of the participants, measures, and data collection procedures can be found in Carrion et al. (2002). Briefly, the sample was recruited from local social service departments and mental health clinics. All of the children in this sample ($N = 50$) were referred to the project due to exposure to interpersonal trauma, all reported a traumatic event and PTSD symptoms. Each had a cortisol assessment and the time since the child’s most recent trauma was assessed. Fifty-one children were given cortisol assessments; however, one child did not have a reliable time since the trauma assessment and was not included in this study. The mean age of the children in this sample was 10.7 years with a range of 7 to 14 years. The sample was composed of 20 girls and 30 boys, Euro American ($n = 23$), African American ($n = 19$), Hispanic ($n = 5$), Asian ($n = 2$), and other ($n = 1$).

To assess PTSD symptoms and the time since the most recent traumatic event we used the Clinician-Administered PTSD Scale for Children and Adolescents (CAPS-CA; Nader et al., 1996). The time since the most recent trauma was measured in months (actual range = 1–108). No participants reported experiencing ongoing traumatic events. Potential comorbid anxiety and depressive disorders were assessed with the Schedule for Affective Disorders and Schizophrenia for School-Age Children–Present and Lifetime Version (K-SADS-PL; Kaufman et al., 1997). Cortisol samples were obtained from the participant during home-based measurements. It was collected 4 times a day (pre-breakfast, prelunch, predinner, and prebed) over the course of 3 consecutive days producing 12 samples. Samples were processed using the Magic Cortisol radioimmunoassay kit produced by Ciba-Corning (Giessen, Germany) as adapted for salivary cortisol analysis. Cortisol is reported in micrograms per deciliter. As recommended for increased reliability (see Gunnar, 2001) an aggregate score (mean) from the 3 days was created for each time period and to reduce the number of statistical tests. We used the single overall mean cortisol level (mean across times of day) for analyses in this investigation. Exploratory analyses were conducted on each of the times of day. Participants’ pubertal development was determined by self-report (Marshal & Tanner, 1970).

**RESULTS**

The sample was divided into children who experienced trauma within the last year (recent trauma; $n = 26$) and...
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Figure 1. Slopes of cortisol on posttraumatic stress disorder symptoms as a function of the time since the most recent trauma.

Children who had not experienced a trauma within the last year (distal trauma; n = 24). These groups were compared using t tests and chi-square tests on demographic variables (age, gender, family income, or parent’s education level), pubertal status, number of traumatic events reported, and comorbid anxiety and depressive disorders. The results indicated that the groups did not differ with regard to any of these variables. Results further indicated that children who experienced trauma within the last year and children who had not experienced a trauma within the last year did not differ with regards to number of PTSD symptoms (M = 36.9, SD = 2.3 versus M = 38.64, SD = 21.1, t < 1) or cortisol levels (M = 0.22, SD = 0.1 versus M = 0.24, SD = 0.1, t < 1). Cortisol was not correlated with number of traumatic events reported (r = .02).

To test the main hypothesis, hierarchical multiple linear regression analyses were conducted to examine if time since the trauma moderated the association between PTSD symptoms and cortisol levels. Cortisol levels and the duration variable (0 = recent trauma, 1 = distal trauma) were entered in Step 1, change in $R^2 = .007$, $F$ change < 1, and interaction term of cortisol level by duration in the second step. The interaction term was significant, change in $R^2 = .12$, $F$ change (1, 46) = 6.38, $p < .05$. The interaction is presented in Figure 1. The association between PTSD symptoms and cortisol levels for each of the times of day (prebreakfast, prelunch, predinner, and prebed) was tested in a similar manner as overall cortisol levels for exploratory purposes. Prebed cortisol levels significantly interacted with time since the trauma, change in $R^2 = .13$, $F$ change (1, 46) = 6.91, $p < .05$. No other interactions (prebreakfast, prelunch, and predinner) were significant.

Because Carrion et al. (2002) found that girls had higher cortisol levels than boys, we examined whether sex influenced the association between PTSD symptoms and cortisol using a similar regression approach as that used for the time since the trauma. Results indicated that sex did not moderate the association, change in $R^2 = .04$, $F$ change (1, 46) = 2.06, ns.

Differences in the size and direction of correlations (using both Pearson and Spearman rank correlations) between the two groups of youth were also examined.
Results indicated that cortisol and PTSD symptoms were significantly positively correlated in the recent trauma group \((r = .41, \rho = .43)\) beyond the .05 level, but not in the distal trauma group where correlations were negative \((r = -.29, \rho = -.25)\), but did not reach statistical significance. Fisher’s \(r\) to \(z\) tests indicated that the differences in the correlation between cortisol and PTSD were statistically significant \((z\ for r = 2.43, z\ for \rho = 2.37, both ps < .05)\) across the two groups.

**DISCUSSION**

Results point to an important role of the time since the traumatic event in understanding the relationship between PTSD symptoms and cortisol levels and support the theorizing of De Bellis (2001). These findings may help to explain inconsistencies in the literature (e.g., De Bellis et al. 1999; Goenjian et al., 1996). Specifically, the relation (i.e., slope of the regression lines) between cortisol and PTSD symptoms were different in the recent trauma group and the distal trauma group, and comparisons indicated that there were significant differences in the size of the correlations across the recent and distal trauma groups. This finding is consistent with the idea that cortisol is positively associated with PTSD symptoms among those with recent traumas, but that this association is different as the time since the most recent trauma elapses. However, mean cortisol levels were not lower in the distal trauma group. This finding might seem inconsistent with a view of altered cortisol levels as time elapses (i.e., differences in the association between cortisol and PTSD symptoms due to mean differences in cortisol levels among those with recent and distal traumatic events). However, there are wide individual differences in normative cortisol levels in youth (e.g., what is elevated for one person may not be so for another and optimal levels of cortisol have not been established; see Gunnar, 2001, for further discussion). Moreover, our study was cross sectional in nature and so precludes conclusions about change over time.

Although this study may help to clarify the complex relation between PTSD and cortisol response the study has several limitations. Conclusions about the progression of the PTSD symptom association with cortisol are limited because our data are from concurrent assessment of symptoms and cortisol and retrospective assessment of time since the trauma. Moreover, the sample size was relatively small when broken down into the two groups. In particular, the small sample size may have influenced statistical conclusions in the distal trauma group where correlations were negative, but not statistically significant. It will thus be important to further explore our findings using prospective designs. However, research shows that after repeated measurement, PTSD symptoms tend to decline over time (Weems, Saltzman, Reiss, & Carrion, 2003) and so repeated measures designs may also be limited in showing time related differences in the direction of the association between PTSD symptoms and cortisol levels. Cross-sequential designs may be useful in this regard (i.e., one that compares individuals with differences in their most recent trauma and follows them over time). Finally, results of our exploratory analyses indicated that the prebed cortisol levels were moderated by the time since the most recent trauma. None of the other cortisol levels (prebreakfast, prelunch, predinner) showed any association with PTSD symptoms levels. Such results further point to research attention on the daily rhythms in cortisol levels to help to further clarify the complex relation between HPA axis functioning and traumatic stress.

**REFERENCES**


implications for research, treatment, and policy. Development and Psychopathology, 13, 539–564.


