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Linking Plasma Cortisol Levels to Phenotypic Heterogeneity of Posttraumatic Stress Symptomatology

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Abstract

Introduction—Recent confirmatory factor analytic studies of the dimensional structure of posttraumatic stress disorder (PTSD) suggest that this disorder may be best characterized by five symptom dimensions—re-experiencing, avoidance, numbing, dysphoric arousal, and anxious arousal. Hypothalamic-pituitary-adrenal (HPA) axis dysregulation in PTSD and has been attributed to enhanced glucocorticoid responsiveness. However, little is known about how altered HPA-axis function is related to this contemporary phenotypic model of PTSD.

Methods—We compared morning plasma cortisol levels of drug-free civilian adults with PTSD (N=29) to trauma-exposed (TC; N=12) and non trauma-exposed healthy controls (HC; N=23). We then examined the relation between cortisol levels and a contemporary 5-factor ‘dysphoric arousal’ model of PTSD symptoms among individuals with PTSD.

Results—After adjustment for white race/ethnicity, education, lifetime alcohol use disorder, and current smoking status, the PTSD (Cohen’s $d=1.1$) and TC (Cohen’s $d=1.3$) groups had significantly lower cortisol levels than the HC group; cortisol levels did not differ between the TC and PTSD groups. Except for age ($r=-.46$), none of the other demographic, trauma-related, or clinical variables, including lifetime mood/anxiety disorder and severity of current depressive and anxiety symptoms, were associated with cortisol levels. In a stepwise linear regression analysis, age ($\beta = -.44$) and severity of emotional numbing symptoms ($\beta = -.35$) were independently associated with cortisol levels in the PTSD group; none of the other PTSD symptom clusters or depression symptoms were significant. Post-hoc analyses revealed that severity of the emotional numbing symptom of restricted range of affect (i.e., unable to have loving feelings) was independently related to cortisol levels ($\beta = -.35$).

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Conclusion—These results suggest that trauma-exposed civilian adults with and without PTSD have significantly lower cortisol levels compared to healthy, non-trauma-exposed adults. They further suggest that low cortisol levels among adults with PTSD may be specifically linked to emotional numbing symptomatology that is unique to the PTSD phenotype and unrelated to depressive symptoms.

Keywords

PTSD; cortisol; 5-factor PTSD model; numbing; hypothalamic-adrenal-pituitary axis

Introduction

Exposure to traumatic events increases the risk for developing posttraumatic stress disorder (PTSD). Over the past two decades, a growing body of emerging confirmatory factor analytic (CFA) studies has begun to examine more refined models of PTSD symptom dimensionality that extend the 3-factor *Diagnostic and Statistical Manual of Mental Disorders-Fourth Edition* (DSM-IV) model to more refined, theory-based 4- or 5-factor models (Yufik and Simms 2010). The most recent development in this literature is a novel 5-factor ‘dysphoric arousal’ model, which builds on theoretical work by Watson (Watson 2005) to suggest that PTSD symptomatology is comprised of separate re-experiencing, avoidance, numbing, dysphoric arousal (e.g., sleep difficulties), and anxious arousal (e.g., exaggerated startle) symptom clusters (Elhai, Biehn et al. 2011). To date, more than a dozen CFA studies conducted in a broad range of trauma-exposed samples, including nationally representative samples, have found that this model provides a significantly better representation of PTSD symptom dimensionality than the DSM-IV or alternative 4-factor models (Elhai, Biehn et al. 2011; Pietrzak, Tsai et al. 2012; Armour, Carragher et al. 2013); Table 1 shows how PTSD symptoms are mapped in each of the models. Emerging work from our group has found preliminary evidence of potential neurobiological correlates for the 5-factor model in relation to serotonin 1b receptor (Pietrzak, Henry et al. 2013) and norepinephrine transporter (Pietrzak, Gallezot et al. 2013) systems in PTSD. However, to date, no study of which we are aware has examined how cortisol levels may relate to this newly proposed and empirically supported phenotypic model of PTSD symptomatology.

PTSD has been linked to altered glucocorticoid signaling based on the idea of enhanced glucocorticoid responsiveness on the one hand (Yehuda, Southwick et al. 1993; Yehuda, Golier et al. 2004) and lower ambient cortisol on the other (Yehuda, Boissoneau et al. 1995). However, there is also increasing recognition of the complex interactive effects of the molecular mechanisms underlying PTSD risk after trauma; the neuroendocrine consequences of early life adversity; as well as findings of gene-by-environment interactions that explain, at least in part, how early in life trauma may increase risk for adult PTSD (Yehuda, Flory et al. 2010). A limitation of extant research, however, is that few studies have examined the relation between basal cortisol levels and heterogeneous symptom clusters that characterize the phenotypic expression of PTSD. Understanding how cortisol relates to the phenotypic expression of PTSD can provide greater specificity regarding the role of cortisol in mediating component aspects of this complex phenotype and may help guide the development of more targeted intervention strategies. Available studies in Holocaust survivors (Yehuda, Kahana et al. 1995) and combat veterans (Mason, Wang et al. 2001; Wabbeh and Oken 2013) have observed that low cortisol levels are linked to increased severity of avoidance/numbing symptoms, which are characterized by persistent avoidance of trauma-related stimuli and numbing of general responsiveness. Other studies have observed associations with other PTSD symptom clusters, including re-experiencing (Steudte, Kirschbaum et al. 2013) and hyperarousal (Wessa, Rohleder et al. 2006) symptoms. While these studies support the idea of a specific association between abnormal

HPA-axis function and the phenotypic expression of PTSD, they are based on dimensional models of PTSD that have received limited empirical support, such as the 3-factor DSM model.

We had two aims in the current study. First, we compared basal cortisol levels in healthy, non-trauma-exposed individuals to trauma-exposed individuals with and without PTSD. Second, we evaluated the differential association between basal cortisol levels and the 5-factor dysphoric arousal model of PTSD among individuals with PTSD. The sample for the current study is the same as that in which we recently examined cannabinoid type 1 receptor availability and endocannabinoid levels in trauma-exposed individuals with and without PTSD (Neumeister, Normandin et al. 2013), and we have since recruited four additional participants with PTSD.

Methods

Participants

Data for this study were drawn from a larger neuroimaging study (Neumeister, Normandin et al. 2013). Participants were recruited through public advertisements searching for individuals with and without non-combat lifetime trauma histories. No participants were currently being treated with psychotropic medication or psychotherapy during the study. The study was approved by the New York University Institutional Review Board and the Yale University School of Medicine Human Investigation Committee. As previously described (Neumeister, Normandin et al. 2013), participants who provided written informed consent were diagnosed using DSM, Fourth Edition – Text Revision (DSM-IV-TR) criteria and the Structured Clinical Interview for DSM-IV (SCID) (First, Spitzer et al. 1995) – modules for major depression, social anxiety disorder, panic disorder, schizophrenia and psychotic disorders, and alcohol and drug use disorders. PTSD symptom severity was assessed using the Clinician-Administered PTSD Scale for DSM-IV (CAPS) (Blake, Weathers et al. 1995), trauma history using the Traumatic Life Events Questionnaire (TLEQ) (Kubany, Haynes et al. 2000); and current depression and anxiety severity using the Montgomery-Åsberg Depression Rating Scale (MADRS) (Montgomery and Asberg 1979) and the Hamilton Anxiety Rating Scale (HAM-A) (Hamilton 1959), respectively. To meet TC inclusion criteria, individuals must have been exposed to at least one potentially traumatic event that met the DSM-IV-TR PTSD criteria A1 and A2, but have no lifetime PTSD or other Axis I diagnosis. PTSD was diagnosed according to standardized CAPS procedures. All participants self-reported their wake-up time on the day of the study as 5:30am and all participants rested for about 60 minutes in a supine position before cortisol samples were collected at 8:30am.

Scores on the re-experiencing, avoidance, numbing, dysphoric arousal, and anxious arousal subscales were derived by summing the corresponding CAPS items (i.e., sum of Frequency + Intensity scores for each symptom that comprises the subscale). Table 1 shows individual symptoms that comprise each of the five symptom subscales.

Data Analysis

Shapiro-Wilk tests were conducted to assess data distributions of all study variables for normality. Non-normally distributed variables (e.g., cortisol levels) were transformed using logarithmic-base-10 transformations prior to analysis. Analyses of variance (ANOVA) were then used to compare continuously-distributed demographic and clinical variables of the HC, TC, and PTSD groups, as well as cortisol levels between these groups; ² tests were used to compare categorical variables. An analysis of covariance (ANCOVA) was conducted to test for group differences in cortisol levels. White race/ethnicity, education,

lifetime alcohol use disorder, and current smoking status were entered as covariates in this analysis. In these analyses, group (HC, TC, and PTSD) and demographic, trauma-related, and clinical variables that differed by group were entered as independent variables, and cortisol levels as the dependent variable. Pairwise comparisons—least-squares difference tests—were computed to compare outcome measures in each of the three groups, with $p < .01$ used to indicate significant group differences. Effect sizes of differences in cortisol levels in the TC and PTSD groups relative to the HC group were expressed using Cohen's d $((M_{\text{group1}} - M_{\text{group2}}) / SD_{\text{pooled}})$ and 95% confidence intervals (CIs). To examine the relation between the 5-factor dysphoric arousal model of PTSD symptoms and cortisol levels in the PTSD group, we conducted zero-order Pearson product-moment correlations and a stepwise linear regression analysis. In the regression analysis, demographic and trauma-related variables associated with cortisol levels at the $p < .05$ level in bivariate analyses, total severity of PTSD symptoms (i.e., CAPS scores), as well as scores on PTSD symptom clusters that comprise the 5-factor dysphoric arousal model of PTSD symptomatology were entered as independent variables, and cortisol levels were entered as the dependent variable. When scores on a symptom cluster emerged as significantly related to cortisol levels, post-hoc analyses were conducted to examine which individual symptom(s) that comprises that cluster was related to these levels.

RESULTS

Demographic, trauma, and clinical characteristics

Table 2 shows demographic, trauma, and clinical characteristics of the HC, TC, and PTSD groups. The groups did not differ with respect to age, sex, BMI, lifetime or past-month alcohol use, drug use disorder, and nicotine dependence; the HC group was more highly educated than the TC and PTSD groups; and was comprised of more White individuals than the TC group. Among current drinkers, number of drinking days in the past month did not differ by group. The PTSD group was more likely than the HC and TC groups to be current smokers and to have a lifetime mood or other anxiety disorder (16 (55.2%) had MDD; 1 (3.4%) had social anxiety disorder; and 1 (3.4%) had panic attacks), and they reported greater severity of current depression and anxiety symptoms. Compared to the TC group, the PTSD group reported greater severity of overall PTSD symptoms, as well as all symptom clusters; age of first trauma, number of traumas, and index traumatic event did not differ.

Group differences in cortisol levels

Results of an ANOVA with group (HC, TC, PTSD) entered as the independent variable and cortisol entered as the dependent variable revealed a significant effect of group ($F = 17.25$, $p < .001$). Compared to the healthy control group ($M = 12.45$, $SD = 3.78$), the Trauma Control ($M = 6.81$, $SD = 2.68$; $p < .001$, $d = 1.63$, 95% CI = .84–2.43) and PTSD ($M = 8.30$, $SD = 3.16$; $p < .001$, $d = 1.20$, 95% CI = .61–1.80) groups had significantly lower cortisol levels; the Trauma Control and PTSD groups did not differ ($p = .40$). Results of an ANCOVA that incorporated White race/ethnicity, education, lifetime alcohol use disorder, and current smoking status into this analysis revealed that group ($F(2,51) = 10.71$, $p < .001$) was the only variable significantly related to cortisol levels. None of the other variables were significant (all F 's $< .96$, all p 's $> .33$). Compared to the healthy control group ($M = 11.78$, $SD = 3.42$), the Trauma Control ($M = 7.13$, $SD = 4.19$; $p = .002$, $d = 1.30$, 95% CI = .54–2.06) and PTSD ($M = 7.58$, $SD = 4.00$; $p < .001$, $d = 1.14$, 95% CI = .55–1.73) groups had significantly lower cortisol levels; the Trauma Control and PTSD groups did not differ ($p = .75$).

Relation between 5-factor model of PTSD symptoms and cortisol levels in PTSD group

Bivariate correlations in the PTSD group indicated that age was significantly negatively correlated with cortisol levels ($r = -.46$, $p = .011$); none of the other demographic, trauma-

related, or clinical variables—including life time mood or anxiety disorder, index trauma or total scores of HAM-A and MADRS—were associated with cortisol levels (all r 's $< .32$, all p 's $> .08$). Although total CAPS scores ($r = -.21$, $p = .29$) were not significantly associated with cortisol levels, scores on the emotional numbing subscale from the 5-factor dysphoric arousal model of PTSD were significantly associated with this outcome ($r = -.39$, $p = .039$); scores on the re-experiencing ($r = -.22$, $p = .26$), avoidance ($r = -.14$, $p = .46$), dysphoric arousal ($r = -.04$, $p = .85$), and anxious arousal ($r = .18$, $p = .35$) symptom subscales were not significantly related to cortisol levels.

In a stepwise linear regression analysis (adjusted $R^2 = .29$), age ($\beta = -.44$, $t = 2.74$, $p = .011$) and severity of emotional numbing symptoms ($\beta = -.35$, $t = 2.20$, $p = .037$) were significantly associated with cortisol levels; total severity of PTSD symptoms (CAPS scores) and the other symptom clusters were not significant (all β 's $< .06$, all p 's $> .73$). Figure 1 shows a scatterplot of cortisol levels and numbing symptoms. Post-hoc analyses revealed that scores on the numbing symptom C6—restricted range of affect (e.g., unable to have loving feelings)—were independently related to cortisol levels ($\beta = -.35$, $t = 2.20$, $p = .037$); the other numbing symptoms were not significant (all β 's $> -.14$, all p 's $> .39$).

Discussion

We found lower plasma cortisol levels in trauma-exposed individuals with and without PTSD relative to healthy, non trauma-exposed individuals. These results corroborate prior studies suggesting that exaggerated functioning of the HPA-axis in PTSD results in low basal cortisol levels (for review (Daskalakis, Lehrner et al. 2013)). Notably, we found lower cortisol levels in both the PTSD and Trauma Control groups, which suggests that low basal cortisol do not distinguish trauma survivors with and without PTSD, and therefore may not be an ideal disease biomarker. Nevertheless, in adult PTSD patients with a history of early life trauma, our results reveal a differential relation between low cortisol levels and a novel five-factor phenotypic model of PTSD, indicating that low cortisol is specifically linked to severity of emotional numbing symptoms in civilians with PTSD.

Dysregulation of the corticotropin releasing factor (CRF)-HPA axis in PTSD is well established (Yehuda 2009). However, there are inconsistencies across studies, which are likely attributable to differences in study methodologies and statistical adjustment for key moderating variables such as trauma histories, age, sex, and co-morbid conditions/symptoms such as depression. We measured morning cortisol under resting conditions and our results accord with those of a recent meta-analysis, which reported lower morning cortisol levels in trauma-exposed individuals with and without PTSD compared to non-traumatized controls (Morris, Compas et al. 2012). Notably, our finding of a negative association between age and cortisol levels in the PTSD group is inconsistent with the observation in a general population sample of men and women in which age was been found to be associated with higher cortisol levels (Larsson, Gullberg et al. 2009). Additional research in larger samples of men and women that represent the full age spectrum of individuals with PTSD will be useful in further evaluating the relation between age and cortisol levels across the lifespan.

Linking abnormal glucocorticoid signaling with a contemporary 5-factor model provides a more refined understanding of the role of cortisol in relation to unique symptom dimensions that comprise the PTSD phenotype. Specifically, among individuals with PTSD, our analyses revealed a specific association between low cortisol levels and emotional numbing symptoms. Notably, total PTSD severity or any other symptoms in our sample were not related to cortisol levels. Given the correlational nature of this association, our data do not allow us to conclusively establish a causal relationship. However, these data do suggest specificity of abnormal glucocorticoid signaling in mediating a key aspect of the PTSD

phenotype—emotional numbing/restricted affect—which was not attributable to severity of other PTSD, depression, or anxiety symptoms, all of which were concomitantly assessed. These results are in line with a study in combat veterans, which reported an association between cortisol and shame/disengagement (Mason, Wang et al. 2001), and extend this finding to civilians with primarily assaultive trauma-related PTSD.

A limitation of the present study is the cross-sectional nature of the data and that only a single measurement of cortisol was collected. However, because all study participants reported that they woke up at the same time on the day of their study, possible effects of between-group variations in awakening on the cortisol awakening response do not explain our results. Clearly, we and others (Morris, Compas et al. 2012) underscore the importance of collecting longitudinal data in never-traumatized individuals, which could help elucidate whether changes in neuroendocrine function observed in adult PTSD is a pre-existing vulnerability, or alternatively, whether it emerges as a neurobiological consequence in response to trauma exposure. Taken together, results of the current study substantiate previous reports of low basal cortisol levels in adults with a history of early in life trauma and suggest that low cortisol may be uniquely linked to the severity of emotional numbing symptoms among individuals with PTSD.

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Figure 1.
Scatterplot of association between cortisol levels and emotional numbing symptoms among individuals with PTSD.

Table 1

Item mappings of DSM-IV, Dysphoria, Numbing, and Dysphoric Arousal structural models of PTSD symptom dimensionality

DSM-IV PTSD symptom	Item Mappings			
	DSM-IV	Dysphoria	Numbing	Dysphoric Arousal
B1. Intrusive thoughts of trauma	R	R	R	R
B2. Recurrent dreams of trauma	R	R	R	R
B3. Flashbacks	R	R	R	R
B4. Emotional reactivity to trauma cues	R	R	R	R
B5. Physiological reactivity to trauma cues	R	R	R	R
C1. Avoiding thoughts of trauma	A	A	A	A
C2. Avoiding reminders of trauma	A	A	A	A
C3. Inability to recall aspects of trauma	A	D	N	N
C4. Loss of interest	A	D	N	N
C5. Detachment	A	D	N	N
C6. Restricted affect	A	D	N	N
C7. Sense of foreshortened future	A	D	N	N
D1. Sleep disturbance	H	D	H	DA
D2. Irritability	H	D	H	DA
D3. Difficulty concentrating	H	D	H	DA
D4. Hypervigilance	H	H	H	AA
D5. Exaggerated startle response	H	H	H	AA

Note. DSM-IV=Diagnostic and Statistical Manual of Mental Disorders, 4th edition. R=Re-experiencing; A=Avoidance; H=Hyperarousal; D=Dysphoria; N=Numbing; DA=Dysphoric Arousal; AA=Anxious Arousal.

Table 2

Demographic and clinical characteristics of study groups.

	Healthy controls	Trauma controls	PTSD	Test of difference	Pairwise comparisons
N	23	12	29		
	M (SD) or n (%)	M (SD) or n (%)	M (SD) or n (%)		
Age	32.1 (8.5)	29.7 (7.9)	33.0 (10.5)	F(2,61)=.52, p=.59	-
Female sex	12 (52.2%)	5 (41.7%)	18 (62.1%)	$\chi^2(2)=1.52$, p=.47	-
White race/ethnicity	14 (60.9%)	2 (16.7%)	10 (34.5%)	$\chi^2(2)=7.22$, p=.027	HC>TC
Education (years)	16.0 (2.1)	14.1 (1.8)	14.4 (2.9)	F(2,61)=3.46, p=.038	HC>TC, PTSD
Body mass index	25.0 (3.4)	26.9 (4.5)	25.4 (4.1)	F(2,61)=.59, p=.56	-
Lifetime alcohol use	11 (73.3%)	3 (42.9%)	16 (66.7%)	$\chi^2(2)=2.00$, p=.37	-
Lifetime alcohol use disorder	0 (0%)	0 (0%)	8 (27.6%)	$\chi^2(2)=11.03$, p=.004	PTSD>HC, TC
Lifetime drug use disorder	0 (0%)	0 (0%)	3 (10.3%)	$\chi^2(2)=3.80$, p=.15	-
Alcohol use in past 30 days	9 (39.1%)	6 (50.0%)	11 (37.9%)	$\chi^2(2)=55$, p=.76	-
Number drinking days in past month	4.3 (3.4)	6.8 (5.0)	5.7 (2.9)	F(2,21)=.87, p=.43	-
Current smoker	0 (0%)	0 (0%)	10 (34.5%)	$\chi^2(2)=14.30$, p=.001	PTSD>HC,TC
Nicotine dependence	0 (0%)	0 (0%)	3 (10.3%)	$\chi^2(2)=3.80$, p=.15	-
Trauma characteristics					
Age at first trauma	-	13.0 (4.8)	12.6 (10.1)	F(1,39)=.02, p=.88	-
Number of traumas	-	3.0 (2.0)	3.4 (3.5)	F(1,39)=.15, p=.70	-
Index traumatic event				$\chi^2(2)=.78$, p=.38	-
Physical or sexual assault	-	7 (58.3%)	21 (72.4%)		
Other trauma	-	5 (41.7%)	8 (27.6%)		
CAPS score	-	5.6 (7.6)	69.9 (20.5)	F(1,39)=110.49, p<.001	PTSD>TC
Re-experiencing symptoms	-	1.1 (1.7)	20.2 (7.8)	F(1,39)=68.96, p<.001	PTSD>TC
Avoidance symptoms	-	1.2 (2.0)	9.9 (4.6)	F(1,39)=39.27, p<.001	PTSD>TC
Numbing symptoms	-	0.2 (0.6)	18.1 (8.1)	F(1,39)=57.92, p<.001	PTSD>TC
Dysphoric arousal symptoms	-	1.8 (3.5)	13.6 (4.2)	F(1,39)=73.65, p<.001	PTSD>TC
Anxious arousal symptoms	-	1.3 (3.0)	8.5 (3.7)	F(1,39)=36.05, p<.001	PTSD>TC

	Healthy controls	Trauma controls	PTSD	Test of difference	Pairwise comparisons
N	23	12	29		
	M (SD) or n (%)	M (SD) or n (%)	M (SD) or n (%)		
Lifetime mood or anxiety disorder	0 (0%)	0 (0%)	17 (58.6%)	$\chi^2(2)=27.94, p<.001$	PTSD>HC, TC
MADRS score	2.2 (3.0)	3.5 (6.5)	22.6 (10.2)	F(2,61)=54.19, p<.001	PTSD>HC, TC
HAM-A score	1.6 (2.5)	2.3 (3.3)	20.7 (10.1)	F(2,61)=54.76, p<.001	PTSD>HC, TC

Note. SD=standard deviation; PTSD=Posttraumatic stress disorder; SD=standard deviation; CAPS=Clinician-Administered PTSD Scale; MADRS= Montgomery-Åsberg Depression Rating Scale; HAM-A=Hamilton Anxiety Rating Scale. Physical assault includes sexual abuse, domestic violence, and other non-combat related physical violence.