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Dex/CRH test cortisol response in outpatients with major depression and matched healthy controls

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Summary

Background—The dexamethasone/corticotropin releasing hormone (Dex/CRH) test has been proposed as a potential tool for identifying endophenotypes relevant to mood disorders. An exaggerated cortisol response to the test during major depressive episodes has been demonstrated for inpatients with melancholic or psychotic features. A diminished hormone response has been observed in chronically depressed outpatients.

Methods—Following a battery of self-report and interview assessments, 68 adults completed the Dex/CRH test. Thirty-four met structured interview criteria for current major depressive disorder and 34 age- and sex-matched control subjects had no current *or* lifetime DSM-IV depressive disorder. Effect of diagnosis on cortisol response to the Dex/CRH test was examined in a repeated measures general linear model.

Results—The matched groups were equivalent with regard to childhood adversity. Cortisol response to the Dex/CRH test among subjects with current MDD was not significantly different from that seen in matched healthy controls. Independent of diagnosis, an exploratory analysis showed a trend-level association between maltreatment history and diminished cortisol response; no interactive effects with depression diagnosis were detected.

Conclusions—The results do not support the hypothesis that elevated cortisol response to the Dex/CRH test represents a marker for major depressive episodes.

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Conflict of Interest

The authors report no disclosures relevant to this work. No therapeutic pharmaceutical or device products were utilized in this research protocol. Acthrel™ was provided at a discounted price by Ferring Pharmaceuticals. The authors disclose the following biomedical financial interests over the past two years and the foreseeable future. Drs. Tyrka, Price, and Carpenter have received grant/research support from the National Institutes of Health, the US Department of the Interior, the US Department of Defense, Sepracor, Pfizer, Cyberonics UCB Pharma, and Medtronic. Dr. Price has received speakers bureau honoraria from Jazz Pharmaceuticals and has served as a consultant for Gerson Lehrman, Wiley, Springer, and Lundbeck. Dr. Carpenter has served as a consultant or on the advisory board for Cyberonics, Neuronetics, Novartis and Wyeth, and has received honoraria for continuing medical education from AstraZeneca and speakers' bureau honoraria for Cyberonics. Ms. Ross, Dr. Kelly, and Dr. Anderson report no biomedical financial interests.

Contributors

Dr. Carpenter participated in all stages of research design, implementation of all procedures, analysis of data, interpretation of results and manuscript preparation. Drs. Tyrka and Price contributed to implementation of the study procedures, interpretation of results, and manuscript preparation; Ms. Ross and Dr. Kelly performed clinical assessments on subjects, assisted on data analysis, and contributed to interpretation of results and manuscript preparation; Dr. Anderson performed biochemical assays and contributed to interpretation of data and manuscript preparation.

Keywords

Childhood abuse; Mood disorders; Depression; Cortisol; HPA axis; Early life stress

1. Introduction

During the 1980s, the dexamethasone suppression test (DST) became a popular research tool for assessment of hypothalamic—pituitary—adrenal system function in depression (Carroll et al., 1981). Despite its utility in the prediction of clinical outcome in some patients (Greden et al., 1983; Ribeiro et al., 1993), especially those with psychotic features (Nelson and Davis, 1997), widespread use of the DST as a diagnostic tool was limited by the test's low sensitivity, ranging between 20% and 50% (Arana et al., 1985).

The DST was extended and refined by combining it with the corticotropin releasing hormone (CRH) stimulation test, which involves placement of an intravenous line for serial withdrawal of blood samples before and after an injected bolus of CRH. Holsboer-Trachsler and colleagues (Holsboer-Trachsler et al., 1987) developed the combined dexamethasone/CRH test to enhance the magnitude of observed differences between healthy controls and depressed patients. The test involves pre-treatment with oral dexamethasone (Dex) 1.5 mg at 11:00 p.m. followed by administration of CRH 100 µg via intravenous bolus at 3:00 p.m. the next day. The Dex/CRH test cortisol response curve incorporates several components, each of which may be uniquely relevant to vulnerability for mood disorders and response to past and current stressors. The post-dex (pre-CRH) baseline cortisol value(s) represent an index of glucocorticoid feedback suppression sensitivity at the level of the pituitary, while the subsequent, post-CRH infusion time-course curve reflects capacity to escape from relative suppression and mount a pituitary—adrenal hormone response to the CRH bolus. In essence, the Dex/CRH test allows examination of an individual's physiological HPA response to a “chemical” stress stimulus (i.e., intravenous CRH bolus) in a psychologically neutral setting, largely bypassing limbic and higher cortical inputs engaged by a physically or emotionally threatening stressor. It provides both baseline and dynamic stress-response system data, and can be easily administered in a standard laboratory setting. As such, the Dex/CRH test may prove to be a useful assessment tool for identification of endophenotypes relevant to risk for mood disorders.

Some of the initial literature related to the Dex/CRH test and depression described *elevated* cortisol and ACTH responses seen among severely ill depressed inpatients, relative to healthy controls (Heuser et al., 1994; Holsboer et al., 1987; Modell et al., 1997), with a tendency for the post-CRH infusion cortisol response curves to diminish or “normalize” with antidepressant treatment and resolution of depressive symptoms (Nikisch et al., 2005). High cortisol Dex/CRH response has been attributed to a change in the adrenal cortex that causes ACTH-independent, disorderly basal cortisol release (Carroll et al., 2007), to amplification of the CRH signal at the level of the pituitary by arginine vasopressin (Scott et al., 1999), and to altered capacity or function of the glucocorticoid receptors in the feedback loop (Holsboer, 2000; Modell et al., 1997). Perhaps most consistently reported in the literature are associations of exaggerated cortisol Dex/CRH response with (1) shortened time to relapse and greater recurrence of depressive episodes (Appelhof et al., 2006; Aubry et al., 2007; Hatzinger et al., 2002) and (2) psychotic and melancholic features (Carroll et al., 2007). However, the finding of cortisol hyper-reactivity to this and other neuroendocrine probe measures, once considered a hallmark of major depressive disorder (MDD), has not been consistently borne out in research examining HPA axis reactivity in a variety of depressive subpopulations.

For example, research in this area has demonstrated that a pattern of relatively “excessive” cortisol release, as measured by the increased response to the Dex/CRH test, is typically not associated with depression in outpatients or in those with chronic course or atypical features (Brouwer et al., 2006; Gervasoni et al., 2004; Oshima et al., 2000; Rydmark et al., 2006; Watson et al., 2002). Suicidal behavior has also been shown to be more characteristic of depressed inpatients with *attenuated*, rather than exaggerated, Dex/CRH responses (Pfennig et al., 2005), and significantly *lower* cortisol and ACTH responses to the Dex/CRH were reported in a study of depressed women with chronic social stressors (Rydmark et al., 2006), a pattern interpreted as reflecting hypoactive central stress-response circuitry.

This apparent inconsistency may suggest two distinct endophenotypes of major depression that manifest clinically with overlapping symptoms (Bremner et al., 2007; Penninx et al., 2007). Alternatively or additionally, other independent variables associated with HPA axis response, such as exposure to childhood adversity, may be the relevant determinants of the direction of abnormal Dex/CRH findings seen in some patient groups. One investigation that evaluated depressed patients with the Dex/CRH test while considering the effects of early life trauma in men (Heim et al., 2008) found hormone responses associated with MDD in men differed as a function of childhood trauma history. Another study comparing depressed patients with healthy controls on the Dex/CRH test found that patients reporting high levels of childhood emotional neglect had cortisol response patterns similar to those seen among healthy controls, while depressed patients without such histories had relatively elevated levels (Watson et al., 2007). To further investigate the relative effects of depression diagnosis and self-reported early life stress on Dex/CRH response, we compared a group with current MDD and a matched group without any history of depressive disorder. The potential confounding effect of childhood maltreatment was examined in post-hoc analyses.

2. Methods and materials

Sixty-eight adults participated in this study. Control subjects were recruited from the community via flyers as well as through Internet and newspaper advertisements for healthy adults. Outpatients with MDD seeking treatment in clinical trials and individuals in the community responding to news-paper advertisements describing research on depressive symptoms were recruited. Depressed subjects were matched by age and sex to a control group of subjects who reported no current or lifetime mood disorders, as confirmed by diagnostic interview. Subjects underwent physical and neurological examinations. Electrocardiograms and laboratory studies for complete blood count, serum electrolytes, thyroid stimulating hormone, urine toxicology, and urinalysis were conducted. Subjects were excluded if they worked night shifts, or if they had one or more of the following conditions: acute or unstable medical illness, a history of brain injury, seizure disorder, endocrine disease, or current substance abuse. Also excluded were individuals undergoing treatment with drugs which might influence HPA axis function, including psychotropic medications, beta blockers, angiotensin-converting enzyme inhibitors, ketoconazole, metyrapone, and corticosteroids. Subjects were free of these medications for at least two weeks (6 weeks for fluoxetine) prior to participation. Oral contraceptives were allowed. All subjects gave voluntary written informed consent to participate in this study, which was approved by the Butler Hospital Institutional Review Board, and were paid for their time and travel to the research clinic.

Current and lifetime history of Axis I psychiatric diagnoses were assessed using the Structured Clinical Interview for DSM-IV (SCID (First et al., 1996)). The 28-item version of the Childhood Trauma Questionnaire (CTQ total and 5 subscale scores (Bernstein et al., 2003)) was used to assess childhood maltreatment. The following additional self-report measures were included to further characterize subjects: (1) Inventory of Depressive

Symptomatology-Self Report (IDS-SR; (Rush et al., 1996)), (2) State-Trait Anxiety Questionnaire (STAI; (Spielberger, 1983)), (3) Perceived Stress Scale (PSS; (Cohen et al., 1983)), and the Quality of Life Enjoyment and Satisfaction Questionnaire (Q-LES-Q; (Endicott et al., 1993)).

On a subsequent visit, subjects completed the Dex/CRH test as follows. The night before the test, a single oral dose of dexamethasone 1.5 mg was self-administered at 11:00 p.m. The following day, participants arrived at 12:00 p.m. and were given lunch. Topical anesthetic cream containing lidocaine 2.5% and prilocaine 2.5% was applied to the subject's forearm between 12:30 and 12:45 p.m. At 1:00 p.m., an indwelling intravenous (IV) catheter was inserted in the forearm by a research nurse with extensive experience with IV catheter placement. Subjects then remained in a semi-recumbent position throughout the procedure except to use the bathroom. They were permitted to read or watch pre-selected movies that did not contain emotionally charged material. Vital signs were monitored throughout the test. At 3:00 p.m., CRH (corticotropin ovine triflutate, Acthrel®, Ferring Pharmaceuticals, Inc.) 100 µg reconstituted in 2 ml 0.9% sodium chloride was infused intravenously over 30 s. Blood samples were drawn at 2:59 p.m. (baseline) and every 15 min thereafter until 5:00 p.m. Samples were immediately stored on ice, centrifuged within 45 min, and then stored at -80 °C for assay of cortisol.

Cortisol assays were performed on samples from 2:59 p.m., 3:30 p.m., 3:45 p.m., 4:00 p.m., and 4:15 p.m. The GammaCoat cortisol I-125 coated-tube radioimmunoassay (RIA) kit (INCSTAR Corp., Stillwater, Minn.) was used to measure cortisol at each time point. The intra-assay and inter-assay CVs observed for quality assessment samples (5 and 20 µg/dl) were less than 5% and 10%, respectively.

Statistical analyses were conducted using SPSS 14.0 for Windows. All analyses were two-tailed with alpha set to $p < .05$. Independent *t*-tests and chi-square tests were performed to compare the groups regarding baseline characteristics, including history of childhood maltreatment as measured by the CTQ total score, five subscale scores, and the dichotomized overall CTQ classification (reflecting whether or not the subject reached “moderate to severe” level of maltreatment on any of the subscales). Cortisol values were positively skewed and were therefore log-transformed to meet normal distribution requirements for parametric tests; raw data are displayed in graphs for greater transparency. Repeated measures general linear models (GLMs) were used to determine whether there were differences in cortisol response to the Dex/CRH test between groups. Post-hoc testing of the model was done with the inclusion of age, sex, diagnosis, and maltreatment history and to explore possible interactions between depression and adverse childhood environment.

3. Results

Clinical characteristics for the MDD subjects and matched controls are displayed in Table 1. While the MDD group reported more depressive symptoms, more perceived stress, greater levels of state and trait anxiety, and poorer overall quality of life, the two groups were equivalent with regard to several CTQ indices of childhood maltreatment (total, 5 subscale scores, and dichotomized overall score). As expected, significant multivariate and within-groups effects of time were observed, reflecting overall change in hormone concentrations during the test. There was not a significant effect of group or of group-by-time interaction in the repeated measures GLM analysis of cortisol (Fig. 1). A subgroup analysis could not be performed to fully examine the interactive effects of childhood adversity with depression because the sample size was too small. However, neither CTQ score (continuous) nor CTQ dichotomized score emerged as a significant covariate when added to the model. The main finding was not changed when only those subjects without childhood maltreatment ($n = 32$)

were included in the model. Examination of men and women in separate analyses did not produce significant group effects for either sex. When we explored the effect of oral contraceptive (OC) use among female subjects by entering it as a covariate, the results were unchanged and OC use did not emerge as a significant effect. The exploratory model (repeated measures GLM with age, sex, diagnosis, maltreatment history, and diagnosis x maltreatment history) revealed a trend-level ($p = .08$) group effect of maltreatment history, such that lower cortisol responses to the Dex/CRH test were seen for those reporting early life adversity on the CTQ across both depressed and healthy control groups. Significant independent effects of diagnosis or interaction between depression and childhood maltreatment were not seen in the analyses we conducted.

4. Discussion

The main finding of this investigation was that relative to age- and sex-matched healthy controls, cortisol responses to the Dex/CRH test were no different for a sample of outpatients with current MDD. We used a self-report scale rather than a clinician-rated interview for assessment of symptom severity, but comparison of the IDS-SR scores to others in the literature suggests our sample of 34 depressed subjects had symptom scores comparable to those currently required for most antidepressant clinical trials (Rush et al., 2006). Twelve of these subjects (35.3%) met the SCID criteria for melancholic subtype; none of the subjects had psychotic features. While we did not have complete history regarding number of prior depressive episodes for all MDD subjects, all but a few MDD subjects were seeking treatment for their symptoms at the time they were studied.

This finding differs from that reported by other investigators who have described mediating effects of childhood abuse on cortisol response to the Dex/CRH test in depressed subjects (Heim et al., 2008; Watson et al., 2007). We did not detect an interaction between depression and early environment, however the present study was not sufficiently powered to adequately address this question. The trend-level finding of diminished cortisol response among maltreated subjects is consistent with another study we conducted in a much larger sample of nondepressed adults (Carpenter et al., in press). Specifically, in that study, self-reported emotional abuse was found to be associated with relatively dampened cortisol response, and the effect appeared to amplify with advancing age. Different types of childhood trauma may be associated with differential outcomes with regard to HPA axis function in adulthood.

Potential biases introduced by estrogen use and menopausal status were controlled for by matching and statistical methods. The study had adequate power (0.8) to detect a moderately large effect size ($d = .69$ at $\alpha = 0.05$) such as that reported by other researchers comparing groups of patients with MDD and matched controls (Kunugi et al., 2006; Watson et al., 2002), but we did not replicate their findings. We cannot rule out the possibility of a smaller effect size that was not detectable in our study. We are not aware of any other published study that has evaluated the Dex/CRH response in depressed patients and age- and sex-matched healthy subjects with both groups equivalent with regard to early life adversity.

While there may be many important determinants of cortisol response to the Dex/CRH test, and the observed pattern of HPA response may reflect important underlying biology relevant to mood and other health disorders, our results do not support the hypothesis that elevated cortisol response to Dex/CRH test is a robust marker for MDD diagnosis in moderately symptomatic outpatients. Since we did not perform serial Dex/CRH tests in these subjects, we cannot determine whether the pattern of cortisol response we observed in our sample of depressed individuals would change or revert to some other “baseline” state upon remission of the episode. Revisiting databases from past studies with depressed patients to further

explore diagnostic subtypes or other subject characteristics associated with increased cortisol response to the Dex/CRH test may be one strategy to help identify the phenotypic variables associated with a distinct biological endophenotype. Additional studies that match large subject groups on these and other variables associated with Dex/CRH test response (e.g., temperament, candidate genes, specific types of childhood maltreatment) will be needed to determine how the test can best be used to predict, diagnose, or monitor mental health outcomes.

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Fig. 1. Plasma cortisol responses to Dex/CRH test in adults with current MDD ($n = 34$) and a control group without any current or lifetime Axis I disorders ($n = 34$). The subjects were matched on age and sex. Data in the figure are not transformed and not adjusted for any covariates.

Table 1

Clinical Characteristics of subjects with current major depressive disorder (MDD; $n = 34$) and matched healthy control subjects with no current/lifetime Axis I mood disorders.

	Current MDD ($n = 34$)	Control ($n = 34$)	<i>p</i> -value
Age (y), mean (SD)	40.50 (12.75)	39.74 (12.11)	n.s.
Range	19–60	19–58	
Sex, n (%)			
Male	14 (41.18)	14 (41.18)	n.s.
Female	20 (58.82)	20 (58.82)	
Perceived Stress Scale			
Mean (SD)	33.48 (9.18)	17.15 (5.37)	<0.001
Inventory of Depressive			
Symptomatology-Self Report, Total score, mean (SD)	36.0 (10.24)	8.85 (6.56)	<0.001
State-Trait Anxiety Inventory			
Mean (SD)			
State	47.38 (11.11)	30.38 (8.26)	<0.001
Trait	53.97 (8.84)	32.85 (10.15)	<0.001
Quality of Life Enjoyment And Satisfaction Questionnaire			
Total score, mean (SD)	39.21 (9.08)	58.65 (7.27)	<0.001
Childhood Trauma Questionnaire (CTQ)			
Total score, mean (SD)	9.99 (3.90)	8.54 (3.21)	n.s.
CTQ Subscales			
n (% meeting moderate-severe)			
Emotional abuse	13 (38%)	9 (26%)	n.s.
Physical abuse	10 (29%)	4 (12%)	0.074
Sexual abuse	9 (26%)	7 (21%)	n.s.
Physical neglect	8 (24%)	4 (12%)	n.s.
Emotional neglect	14 (41%)	9 (26%)	n.s.
CTQ Dichotomized			
n (% meeting threshold on any of 5 subscales)	20 (58.82)	16 (47.06)	n.s.
Oral contraceptive use n (%)	3 (8.82)	5 (14.71)	n.s.
Postmenopausal status* n (%)	4 (11.76)	4 (11.76)	n.s.
Melancholic Subtype n (%)	12 (35.29)	-	-

* None using hormone replacement therapy.