Role of cortisol in mood and memory in patients with intractable temporal lobe epilepsy

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ABSTRACT

Objective: This study prospectively examined the relationships among late night salivary cortisol (NSC) levels and depressive symptoms, memory performance, and hippocampal volumes in patients with medically intractable temporal lobe epilepsy (TLE) and the potential mediating effects of cortisol in the relationships between these variables.

Methods: Participants included 24 adults with well-characterized medically refractory TLE (right = 11; left = 12; bitemporal = 1). All patients provided saliva samples and completed measures of mood, anxiety, and memory (objective and subjective). MRI-based volumetric analyses of the hippocampi were also conducted.

Results: As hypothesized, cortisol was found to be negatively related to several memory measures such that patients with higher cortisol levels demonstrated lower memory performance. However, unexpectedly, cortisol was not related to current symptoms of depression or anxiety, subjective memory ratings, or hippocampal volumes. Consistent with previous findings in the literature, a number of other relationships among the study variables were observed (objective memory and hippocampal volume; subjective memory and mood/anxiety). Results of mediator analyses suggested that cortisol does not mediate the relationship between depression and memory dysfunction or the relationship between depression and hippocampal atrophy.

Conclusions: While cortisol may play a role in memory performance in patients with TLE, it does not fully explain the relationship between depression and mesial temporal dysfunction, likely reflecting the complex and multifactorial relationships among these variables. Results confirm the relationship between memory performance and structural brain integrity and provide further support for a role of depression in subjective memory complaints. *Neurology*® 2012;78:1064-1068

GLOSSARY

CES-D = Center for Epidemiologic Studies Depression Scale; **MAC-S** = Memory Assessment Clinics Self-Rating Scale; **MPRAGE** = magnetization-prepared rapid gradient echo; **NSC** = night salivary cortisol; **RAVLT** = Rey Auditory Verbal Learning Test; **STAI** = State-Trait Anxiety Inventory; **TLE** = temporal lobe epilepsy; **WMS-III** = Wechsler Memory Scale, third edition.

Temporal lobe epilepsy (TLE) is the most prevalent type of seizure disorder in adults, and patients whose seizures arise from temporal regions are often at high risk for depression, memory dysfunction, and hippocampal atrophy. However, the relationships among these factors within patients with TLE have not been well-delineated. Research on patients with major depressive disorder has demonstrated that elevated glucocorticoids (GCs), such as cortisol, are associated with memory impairment and hippocampal atrophy often observed in patients with this mood disorder.^{1,2} Recent research has suggested that many epilepsy patients have increased cortisol levels related to seizure frequency³; however, no known studies have examined the potential role of GCs in mood and mesial temporal dysfunction in TLE. This study aimed to 1) prospectively examine the relationships among late night salivary cortisol (NSC) levels (an index of hypercortisolism) and depressive symptoms, memory performance, and hippocampal volumes in patients with medically intractable TLE, and 2) investigate the potential mediating

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Supplemental Data

From the Neurological Institute (R.M.B., J.S.C., B.D., A.A., K.U., R.I.N., C.S.K., G.E.T., I.M.N.), Center for Pediatric Behavioral Health (T.F.), and Endocrinology, Diabetes, and Metabolism (A.H.H.), Cleveland Clinic, OH.

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role of cortisol in the relationship between depression and memory dysfunction and hippocampal atrophy. We hypothesized that patients with elevated cortisol levels would evidence significantly more depressive symptoms, lower memory scores, and lower hippocampal volumes than patients with lower levels of cortisol. It was further hypothesized that cortisol would mediate the relationship between negative affect and mesial temporal integrity.

METHODS **Participants.** This study involved the prospective collection of data from 26 patients with well-characterized medically refractory epilepsy who were evaluated at Cleveland Clinic for surgical treatment of their seizures. A record review was conducted for patients scheduled for preoperative neuropsychological testing between January 2007 and July 2009 ($n = 199$) to determine if they met the following inclusion criteria: 1) aged 18 or older, 2) EEG studies consistent with temporal lobe epilepsy (i.e., all interictal epileptiform discharges arise from temporal lobe regions and ictal onset is consistent with temporal lobe epilepsy) and seizure semiology consistent with temporal lobe epileptogenic focus, as determined by review of an experienced epileptologist (B.D., A.A.), 3) no MRI abnormalities outside of temporal lobe regions, 4) fluent in English, 5) no history of mental retardation, developmental disorder, psychotic disorder, or neurosurgical intervention, 6) no thyroid dysfunction, and 7) not taking any glucocorticoids (e.g., hydrocortisone, prednisone). A total of 42 patients were eligible for study participation, and 38 of those presented for preoperative neuropsychological testing as scheduled. Three patients were not approached regarding the study, because they were unable to stay and meet with the investigator following their neuropsychology appointment. The remaining 34 patients were approached: 27 consented to participate and 7 declined. One of the consented patients completed the cognitive and mood measures, but did not return her saliva sample for cortisol testing. Of the remaining 26 patients enrolled in the study, 2 patients were excluded because their cortisol values were outliers, resulting in a final sample size of 24 (right = 11; left = 12; bitemporal = 1). Participants ranged in age from 20 to 61 years (mean = 39.71, SD = 11.56) and in education from 8 to 18 years (mean = 14.04, SD = 2.48). The mean age at seizure onset for the group was 26.33 (SD = 12.58) and the mean duration of epilepsy was 13.29 years (SD = 9.92).

Standard protocol approvals, registrations, and patient consents. This study was approved by the Cleveland Clinic Institutional Review Board, and written informed consent was obtained from all patients participating in the study.

Neuropsychological assessment. In addition to video-EEG monitoring and MRI studies, all patients undergoing presurgical evaluations for treatment of epilepsy at Cleveland Clinic undergo routine neuropsychological evaluations that include the following measures of objective and subjective memory: Rey Auditory Verbal Learning Test (RAVLT),⁴ select subscales of the Wechsler Memory Scale, third edition (WMS-III)⁵ (i.e., logical memory, verbal paired associates, faces, and family pictures), and the Memory Assessment Clinics Self-Rating Scale (MAC-S)⁶ ability and frequency subscales. As part of their study participation,

Table 1 Patient scores on cognitive and behavioral measures^a

Measure	Mean (SD)
RAVLT total: trials 1-5	90.17 (14.06)
RAVLT long delay free recall	89.61 (15.06)
WMS-III auditory immediate memory	94.92 (13.53)
WMS-III auditory delayed memory	95.46 (12.85)
WMS-III visual immediate memory	89.79 (14.70)
WMS-III visual delayed memory	91.50 (13.36)
STAI state anxiety subscale	54.00 (15.01)
STAI trait anxiety subscale	59.09 (12.04)
CES-D	19.54 (14.10)
MAC-S ability subscale	59.87 (11.99)
MAC-S frequency subscale	71.20 (17.58)

Abbreviations: CES-D = Center for Epidemiologic Studies Depression Scale; MAC-S = Memory Assessment Clinics Self-Rating Scale; RAVLT = Rey Auditory Verbal Learning Test; STAI = State-Trait Anxiety Inventory; WMS-III = Wechsler Memory Scale, third edition.

^a Memory scores are reported as standard scores (mean = 100, SD = 15), STAI scores are reported as t scores (mean = 50; SD = 10), and CES-D and MAC-S scores are reported as raw scores.

participants also completed the following additional measures: Center for Epidemiologic Studies Depression Scale (CES-D)⁷ and State-Trait Anxiety Inventory (STAI).⁸ A description of the cognitive and behavioral measures is provided in appendix e-1 on the *Neurology*[®] Web site at www.neurology.org, and a summary of patient scores is provided in table 1. One patient did not complete the STAI, and one patient did not complete the RAVLT. Therefore, correlations with those measures reflect sample sizes of 22 or 23 rather than 24.

Night salivary cortisol. All participants provided a midnight saliva sample, which was returned to the principal investigator the following morning. In order to reduce the chance of obtaining stress-induced elevated cortisol levels, patients were instructed to provide the samples on a rather low stress day after having been seizure-free for at least 24 hours. Participants were instructed to collect the saliva sample as close as possible to bedtime. All subjects were provided with written instructions for collecting NSC samples, which advised them to rinse their mouths thoroughly with water 5 minutes prior to collection to eliminate food remnants. They were also instructed to avoid brushing teeth, engaging in strenuous physical activity, and smoking or drinking coffee, alcohol, or juice prior to collection.

MRI-based volumetric analyses. All participants underwent brain MRI as part of a standard presurgical epilepsy evaluation, including sagittal T1, coronal fluid-attenuated inversion recovery and T2, and high-resolution coronal magnetization-prepared rapid gradient echo (MPRAGE) images on a 1.5-T scanner (Siemens, Erlangen, Germany). Coronal 3D MPRAGE sections (slice thickness 2 mm, no interslice gap) were used to outline the mesial temporal lobe structures. Sequence details were repetition time 11.08 msec, echo time 4.3 msec, 10° flip angle, inversion time 360 msec, in-plane field of view 230 mm², 256 × 256 matrix. Operator-based volumetry of the mesial temporal structures was performed on contiguous coronal MPRAGE slices as described previously.⁹ All image processing

Table 2 Pearson correlations between study measures with 95% confidence intervals

Measure	Right hippocampus	Left hippocampus	CES-D	STAI-state	STAI-trait	RAVLT-TTL	RAVLT-LDFR	Aud-Imm	Aud-Del	Vis-Imm	Vis-Del	MAC-S-ability	MAC-S-freq
NSC	0.096	-0.112	-0.272	-0.318	-0.427 ^a	-0.069	-0.425 ^a	-0.222	-0.222	-0.444 ^a	-0.461 ^a	-0.160	0.258
	-0.34 to 0.50	-0.51 to 0.32	-0.61 to 0.15	-0.65 to 0.11	-0.71 to -0.02	-0.47 to 0.35	-0.71 to -0.02	-0.57 to 0.20	-0.57 to 0.20	-0.72 to -0.05	-0.73 to -0.07	-0.53 to 0.26	-0.16 to 0.60
Right hippocampus	—	0.711 ^b	-0.108	0.030	-0.057	0.476 ^a	0.164	0.112	0.265	-0.076	-0.112	0.156	0.252
		0.41 to 0.87	-0.51 to 0.33	-0.41 to 0.46	-0.48 to 0.38	0.06 to 0.75	-0.29 to 0.56	-0.32 to 0.51	-0.18 to 0.62	-0.48 to 0.36	-0.51 to 0.32	-0.28 to 0.54	-0.19 to 0.61
Left hippocampus	—	—	-0.127	-0.122	-0.127	0.445 ^a	0.327	0.023	0.319	-0.268	-0.328	0.209	0.245
			-0.33 to 0.53	-0.53 to 0.32	-0.53 to 0.32	0.02 to 0.74	-0.12 to 0.66	-0.40 to 0.44	-0.12 to 0.65	-0.62 to 0.17	-0.66 to 0.11	-0.23 to 0.58	-0.20 to 0.60
CES-D	—	0.623 ^b	—	0.791 ^b	-0.326	-0.209	-0.103	-0.103	-0.047	0.147	0.181	-0.293	-0.464 ^a
		0.28 to 0.82	0.56 to 0.91	-0.65 to 0.10	-0.57 to 0.22	-0.49 to 0.31	-0.44 to 0.36	-0.27 to 0.52	-0.24 to 0.54	-0.27 to 0.52	-0.24 to 0.54	-0.62 to 0.13	-0.73 to -0.08
STAI-state	—	0.792 ^b	—	—	-0.192	-0.028	0.023	0.024	0.024	0.157	0.229	-0.079	-0.182
		0.56 to 0.91	-0.57 to 0.25	-0.44 to 0.40	-0.39 to 0.43	-0.39 to 0.43	-0.27 to 0.53	-0.20 to 0.59	-0.48 to 0.34	-0.20 to 0.59	-0.20 to 0.59	-0.48 to 0.34	-0.55 to 0.25
STAI-trait	—	—	—	—	-0.197	-0.010	0.078	0.149	0.149	0.109	0.160	-0.131	-0.444 ^a
			-0.57 to 0.24	-0.43 to 0.41	-0.35 to 0.47	-0.28 to 0.53	-0.32 to 0.50	-0.27 to 0.54	-0.52 to 0.30	-0.27 to 0.54	-0.27 to 0.54	-0.52 to 0.30	-0.72 to -0.04
RAVLT-TTL	—	0.666 ^b	—	—	—	—	0.613 ^b	0.638 ^b	0.638 ^b	0.210	0.218	0.308	0.358
		-0.85 to -0.35	0.27 to 0.82	0.31 to 0.83	-0.22 to 0.57	-0.22 to 0.57	-0.22 to 0.57	-0.21 to 0.58	-0.12 to 0.64	-0.21 to 0.58	-0.21 to 0.58	-0.12 to 0.64	-0.06 to 0.67
RAVLT-LDFR	—	—	—	—	—	—	0.297	0.309	0.309	0.352	0.357	0.425 ^a	0.197
		-0.13 to 0.63	-0.12 to 0.64	-0.07 to 0.67	-0.06 to 0.67	-0.06 to 0.67	-0.06 to 0.67	-0.06 to 0.67	-0.06 to 0.67	-0.06 to 0.67	-0.06 to 0.67	-0.06 to 0.67	-0.23 to 0.56
Aud-Imm	—	0.884 ^b	—	—	—	—	—	0.884 ^b	0.884 ^b	0.566 ^b	0.544 ^b	0.063	0.066
		0.75 to 0.95	0.21 to 0.79	0.21 to 0.79	0.21 to 0.79	0.21 to 0.79	0.21 to 0.79	0.21 to 0.79	0.21 to 0.79	0.21 to 0.79	0.21 to 0.79	0.21 to 0.79	0.21 to 0.79
Aud-Del	—	—	—	—	—	—	—	—	—	—	—	—	—
		-0.09 to 0.65	-0.10 to 0.64	-0.10 to 0.64	-0.10 to 0.64	-0.10 to 0.64	-0.10 to 0.64	-0.10 to 0.64	-0.10 to 0.64	-0.10 to 0.64	-0.10 to 0.64	-0.10 to 0.64	-0.10 to 0.64
Vis-Imm	—	—	—	—	—	—	—	—	—	—	—	—	—
		0.73 to 0.94	0.73 to 0.94	0.73 to 0.94	0.73 to 0.94	0.73 to 0.94	0.73 to 0.94	0.73 to 0.94	0.73 to 0.94	0.73 to 0.94	0.73 to 0.94	0.73 to 0.94	0.73 to 0.94
Vis-Del	—	—	—	—	—	—	—	—	—	—	—	—	—
		-0.36 to 0.45	-0.36 to 0.45	-0.36 to 0.45	-0.36 to 0.45	-0.36 to 0.45	-0.36 to 0.45	-0.36 to 0.45	-0.36 to 0.45	-0.36 to 0.45	-0.36 to 0.45	-0.36 to 0.45	-0.36 to 0.45
MAC-S-ability	—	—	—	—	—	—	—	—	—	—	—	—	—
		0.47 to 0.88	0.47 to 0.88	0.47 to 0.88	0.47 to 0.88	0.47 to 0.88	0.47 to 0.88	0.47 to 0.88	0.47 to 0.88	0.47 to 0.88	0.47 to 0.88	0.47 to 0.88	0.47 to 0.88

Abbreviations: Ability = ability subscale; Aud-Del = Auditory Delayed Memory Index; Aud-Imm = Auditory Immediate Memory Index; CES-D = Center for Epidemiologic Studies Depression Scale; Freq = frequency subscale; MAC-S = Memory Assessment Clinics Self-Rating Scale; NSC = night salivary cortisol; RAVLT = Rey Auditory Verbal Learning Test; STAI = State-Trait Anxiety Inventory; Vis-Del = Visual Delayed Memory Index; Vis-Imm = Visual Immediate Memory Index.

^a $p < 0.05$.

^b $p < 0.01$.

was conducted by the same investigator (K.U.) who was blinded to all clinical information, including cortisol results.

Statistical analyses. First, bivariate Pearson correlations were calculated to examine the strength of the relationships among cortisol level, mood, anxiety, memory, and hippocampal volumes (table 2). Then, to investigate mediating effects of cortisol, a series of regression equations were conducted, using methods described by Baron and Kenny.¹⁰

RESULTS Higher cortisol levels were associated with poorer delayed verbal memory performance (RAVLT delayed recall $r = -0.425$), weaker immediate and delayed visual memory (WMS-III visual memory indices $r = -0.444$ and -0.461), and greater trait anxiety (STAI trait anxiety $r = -0.427$). In addition, patients with smaller hippocampal volumes demonstrated poorer performance on a word-list learning task (left volume $r = 0.445$; right volume $r = 0.476$), and patients who endorsed greater symptoms of depression and anxiety reported more subjective memory complaints (CES-D $r = -0.464$, STAI trait anxiety $r = -0.444$). Interestingly, the memory indices as assessed by the WMS-III were not significantly related to hippocampal volume.

Unexpectedly, cortisol levels were not related to current symptoms of depression or anxiety, subjective memory ratings, or hippocampal volumes (range $r = -0.318$ to 0.258). Given the lack of a relationship between cortisol level and mood, cortisol could not mediate the relationship between depression and memory dysfunction or the relationship between depression and hippocampal atrophy in patients with medically intractable TLE (figure).

DISCUSSION Late-night salivary cortisol is an excellent indicator of biologically active, free cortisol concentration in serum, independent of salivary flow rate reference. Measurement of midnight salivary

cortisol is a simple, noninvasive, and reliable screening test for evaluation of patients suspected to have hypercortisolism. NSC has been advocated as a first-line screening test for patients suspected to have Cushing syndrome, and a recent meta-analysis reported excellent diagnostic characteristics for NSC in such patients.¹¹

Consistent with the existing literature in other populations, results of the current study suggest that higher NSC is related to lower objective memory performance and higher trait anxiety in patients with medically intractable epilepsy; however, NSC was not related to current symptoms of depression or anxiety. Unexpectedly, there was no significant relationship between NSC and hippocampal volume in this sample, and mediational analyses suggest that cortisol does not fully explain the relationship between depression and mesial temporal dysfunction in epilepsy. Supplemental analyses supported existing findings in epilepsy in that objective memory was related to hippocampal volume, on at least some memory measures, and subjective memory was related to mood and anxiety symptoms.

The lack of significant mediational relationships in this study may be due to the few patients with clinically elevated cortisol levels ($n = 3$). Most studies in the literature have been conducted in patient populations with cortisol hypersecretion (e.g., depression, Cushing syndrome). Future studies with larger sample sizes and greater cortisol variability will be required to further delineate the relationships among negative affect, cortisol, and the functional and structural integrity of mesial temporal structures in patients with intractable TLE. If the relationship between cortisol and memory is replicated in larger samples, it may provide a target for intervention to reduce memory morbidity in patients with TLE.

AUTHOR CONTRIBUTIONS

Dr. Busch: design/conceptualization of study, analysis/interpretation of data, drafting/revising manuscript. Dr. Frazier: design/conceptualization of study, analysis/interpretation of data, drafting/revising manuscript. Dr. Chapin: design/conceptualization of study, analysis/interpretation of data, drafting/revising manuscript. Dr. Hamrahian: design/conceptualization of study, analysis/interpretation of data, drafting/revising manuscript. Dr. Diehl: design/conceptualization of study, analysis/interpretation of data, drafting/revising manuscript. Dr. Alexopoulos: design/conceptualization of study, analysis/interpretation of data, drafting/revising manuscript. Dr. Unnwongse: analysis/interpretation of data, drafting/revising manuscript. Dr. Naugle: design/conceptualization of study, analysis/interpretation of data, drafting/revising manuscript. Dr. Kubu: design/conceptualization of study, analysis/interpretation of data, drafting/revising manuscript. Dr. Tesar: design/conceptualization of study, analysis/interpretation of data, drafting/revising manuscript. Dr. Najm: design/conceptualization of study, analysis/interpretation of data, drafting/revising manuscript.

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Figure Examination of cortisol as a potential mediator in the relationship between depression and memory

For purposes of this illustration, depression was reported using Center for Epidemiologic Studies Depression Scale score and memory was reported using the long delayed free recall trial of the Rey Auditory Verbal Learning Test. Similar relationships were observed between depression and other measures of memory as well as between depression and hippocampal volumes. NSC = night salivary cortisol.

DISCLOSURE

Dr. Busch has received research support from the Epilepsy Foundation and the NIH/NCRR. Dr. Frazier serves on the editorial board of *Assessment*; has served as a consultant for Shire plc; and has received research support from Bristol-Myers Squibb, the NIH/NCRR, and the Brain and Behavior Research Foundation. Dr. Chapin has received research support from the American Parkinson Disease Association and the American Psychological Association, Division 40. Dr. Hamrahian and Dr. Diehl report no disclosures. Dr. Alexopoulos serves on the editorial board of *Epileptic Disorders*; is employed as a full-time adult epileptologist, who performs video-EEG, EEG, and other clinical neurophysiology studies at the Cleveland Clinical Epilepsy Center and bills for these procedures; serves on the speakers' bureau of UCB; and has received research support from UCB, Pfizer Inc., the US Department of Defense, and the American Epilepsy Society. Dr. Unnwongse reports no disclosures. Dr. Naugle serves on editorial advisory boards for *Neuropsychology* and *The Clinical Neuropsychologist*. Dr. Kubu serves as a consultant for Medtronic, Inc.; is co-author on a patent filed examining deep brain stimulation in the treatment of neuropsychological disorders; and receives research support from the NIH (NIMH, NINDS) and the Greenwall Foundation. Dr. Tesar reports no disclosures. Dr. Najm serves as an Associate Editor for *Epileptic Disorders*; serves on the speakers' bureau for UCB; and receives research support from the US Department of Defense.

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