Initial Posttraumatic Urinary Cortisol Levels Predict Subsequent PTSD Symptoms in Motor Vehicle Accident Victims

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Background: *This study was designed to examine the relationship between urinary hormone levels collected upon admission to the trauma unit following a motor vehicle accident and posttraumatic stress disorder symptomatology 1 month later.*

Methods: *Fifteen-hour urine samples were collected from 63 male and 36 female motor vehicle accident victims and were used to assess levels of catecholamines and cortisol reflecting peritraumatic and acute-phase posttraumatic levels. Presence of posttraumatic stress disorder symptomatology was assessed 1 month after the accident.*

Results: *Motor vehicle accident victims subsequently diagnosed with acute posttraumatic stress disorder excreted significantly lower levels of cortisol in 15-hour urines collected upon admission to the hospital. In addition, urinary levels of cortisol predicted a significant percentage of the variance in intrusive and avoidant thoughts 1 month after the accident.*

Conclusions: *The results of our study suggest that initial cortisol levels in the immediate aftermath of a traumatic event contribute, in part, to subsequent symptoms of posttraumatic stress disorder.* Biol Psychiatry 2000;48: 940–947

Key Words: Urinary cortisol, posttraumatic stress disorder, neuroendocrine

Introduction

The psychophysiology of posttraumatic stress disorder (PTSD) has been the focus of a number of recent studies, with research indicating that neuroendocrine levels may differentiate between those victims who meet PTSD criteria and those who do not. Despite mixed findings (for exceptions, see Lemieux and Coe 1995; Pitman and Orr 1990), the majority of studies have found lower 24-hour urinary cortisol excretion in victims with PTSD compared with victims without PTSD and normal control subjects (Mason et al 1986; Yehuda et al 1990, 1991, 1993, 1995a, 1995b; for a review, see Friedman 1991), suggesting a downregulation of the hypothalamic–pituitary–adrenal (HPA) axis in PTSD. Further support for altered HPA functioning stems from findings of greater numbers of lymphocyte glucocorticoid receptors (Yehuda et al 1991, 1993) and exaggerated dexamethasone (.5mg dose) suppression of cortisol excretion (Halbreich et al 1989; Yehuda et al 1993, 1995) in PTSD patients. Augmented adrenocorticotropic hormone response has also been noted after metyrapone treatment (Yehuda et al 1996), leading Yehuda and colleagues to suggest that PTSD is characterized by an enhanced sensitivity of the glucocorticoid negative feedback loop at the pituitary.

Studies examining basal catecholamine levels in patients with PTSD also have reported mixed results. Whereas PTSD patients and control subjects do not differ in levels of plasma norepinephrine and epinephrine (Blanchard et al 1991; McFall et al 1990; Southwick et al 1994), findings concerning 24-hour urinary catecholamine excretion have suggested greater catecholamine excretion in PTSD patients versus control subjects (Lemieux and Coe 1995; Kosten et al 1987; Yehuda et al 1992; see Friedman 1991 for a review).

The majority of these studies have examined neuroendocrine levels in chronic PTSD patients who have presented with PTSD for more than 20 years, making conclusions concerning onset of altered neuroendocrine levels difficult. Determining whether neuroendocrine abnormalities reflect altered acute responses to the traumatic stressors that persist long after the event or long-term presence of PTSD is impossible. Recent research has attempted to address this issue by examining neuroendocrine levels of trauma victims during the acute phase of responding to traumatic events. Resnick et al (1995) examined plasma cortisol levels within 51 hours after a rape (mean = 12.0 ± 15.6 hours) in 37 adult female victims. Women with prior assault or rape histories demonstrated lower plasma cortisol levels and were more likely to develop PTSD than were women without similar trauma histories;

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however, cortisol levels did not predict PTSD diagnosis at follow-up (ranging from 17 to 157 days after the rape). McFarlane et al (1997) examined 40 motor vehicle accident (MVA) victims and found that, although victims meeting PTSD criteria 6 months after the accident had significantly lower plasma cortisol levels than did victims with major depressive disorder, cortisol levels of PTSD patients did not differ from those of victims with no diagnosis. Both of these studies examined small numbers of participants and may not have had enough power to examine the relationship between initial cortisol levels and subsequent PTSD symptoms or to detect differences in cortisol levels between PTSD and non-PTSD patients.

It is difficult to measure acute physiologic responses to traumatic stress in naturalistic studies because traumatic stressors often involve a number of stressful events and do not have clear start and end points. For example, an MVA can include a number of potentially traumatic events: initial realization that a person is going to be in an accident, actual impact, pain experienced, hospital procedures, body disfigurement (facial scars), and so forth. Plasma cortisol levels provide an approximation of cortisol levels over the last hour or two (Baum and Grunberg 1995), and the acute time frame encompassed in a single blood draw may not be sufficient to examine more general systemic alterations in HPA hormones and their effects on the encoding and consolidating of these various traumatic memories. Urine samples average hormone levels across a longer time frame (the duration of the collection period) than do plasma samples and may provide a better measure of hormone levels throughout the acute phase of responding to a traumatic event (Baum and Grunberg 1995).

In our study, 15-hour urine samples were collected from MVA victims beginning upon admission to the trauma unit after their accidents. Urine samples were used to examine MVA victims' peri- and acute-phase posttraumatic neuroendocrine responses to their accident and to assess their efficacy in predicting PTSD symptomatology 1 month after the accident.

Although 24-hour urine samples would have provided hormone levels across the full circadian cycle, lesser injured victims were often not hospitalized for a full 24 hours, and the shorter sampling period allowed us to examine a wider range of patients.

Methods and Materials

Participants

Participants consisted of 63 male and 36 female motor vehicle (20% motorcycle) accident victims admitted to the level 1 trauma center of a Midwestern community hospital. Victims with Glasgow Coma Scale scores of less than 14 were excluded. Among the subjects, 73% were drivers. The average age of participants was 37.3 years (SD = 17.7, range = 18–84), and the sample was 85% European American, 13% African American, and 2% Asian American.

Procedures

Upon admission to the trauma center, patients are routinely catheterized and urine is collected as part of their medical treatment. For all potential participants, urine was collected on ice for the next 15 hours. Participants who were not catheterized (approximately 7%) urinated into polypropylene containers stored on ice for the 15-hour collection period. Compliance with these procedures was monitored by the on-duty nurse, and noncatheterized patients did not differ from catheterized patients in volume of urine excreted. At the end of the collection period, aliquots of the urine (60 mL) were frozen for catecholamine and cortisol analyses. Aliquots for catecholamine analysis were acidified to pH $\leq$ 3 before freezing.

Victims were approached by the head trauma nurse during their stay in the hospital. The nurse administered a brief Mini-Mental State exam (Folstein et al 1975) to determine the patient's ability to give informed consent. If the patient passed the exam, a researcher approached the patient. After completely describing the study to the patient, written informed consent was obtained. If the patient refused, the urine that had been previously collected was discarded. Researchers met with participants an average of 31.5 hours after admission (range = 4 to 144 hours depending on severity of injury). Of the 117 patients approached, 18 refused (acceptance rate of 85%). Demographics of refusers did not differ from participants enrolled in the study.

During the initial in-hospital assessment, behaviors prior to the accident (foods and drinks ingested, smoking, and drug or medication use) that may affect urinary hormone levels were measured. Similarly, injury severity scores (ISSs; Abbreviated Injury Scale, Association for the Advancement of Automotive Medicine 1990) were computed using objective injury data collected from patients' charts, and medications received at the hospital and time of admittance were also recorded. Subjects were paid $20, and arrangements for the follow-up interview were made.

Participants were assessed again at 1 month postaccident. This assessment was conducted in the participants' homes by one of two clinical psychology graduate students. The PTSD module of the structured clinical interview for the DSM-IV (SCID; First et al 1996) was administered, and a questionnaire packet including the Impact of Event Scale (IES; Horowitz et al 1979) was left with the participant to be completed and returned. Seventy-nine participants completed the SCID interview, and 68 participants returned the IES. Primary reasons for not completing the study included: participant moving without any forwarding information, participant being too busy to participate, and participant not responding to numerous phone calls and mailings. Noncompleters were younger [20.2 vs. 35.3 years, $F(1,89) = 7.8$, $p < .01$], and less severely injured [ISS scores of 2.4 vs. 6.6, $F(1,89) = 4.2$, $p < .05$] than the remaining group. Minorities were not significantly overrepresented among the noncompleters, but noncompleters were more likely to be men [18 men and two

women, $\chi^2(1, N = 99) = 7.5, p < .01$]. Completers did not differ from noncompleters in levels of urinary hormones.

Because our primary predictor variables were urinary hormone levels, we excluded participants who suffered from chronic diseases (diabetes, asthma, arthritis) or were taking medications (high blood pressure medications, thyroid medications) that could affect urinary hormone levels. In addition, those who were administered steroids (for possible cervical spine fractures) in the hospital were also excluded. This resulted in a total sample of 74 participants (49 men and 25 women) for whom we had urine data. Of these, 55 completed the SCID interview, and 48 returned the IES questionnaire.

Measures

The Mini-Mental State Exam (MMSE; Folstein et al 1975) is a 30-point exam testing orientation, attention, calculation, and ability to follow verbal and written instructions. A score of 25 is required to participate in the study. The MMSE demonstrates good test–retest reliabilities (.83–.98; Folstein et al 1975), has been used extensively in medical and emergency situations, and has served as a test for ability to give consent to medical procedures (Dziedzic et al 1998; Holzer et al 1997).

MEASURES OF PTSD. During the initial assessment in the hospital, no systematic examination of PTSD symptoms was conducted; however, after defining intrusive thoughts as "thoughts that pop into your head concerning the accident even when you were not particularly trying to think about the accident," we asked participants whether they had experienced intrusive thoughts.

One month after the accident, the PTSD module of the SCID (First et al 1996) was administered. The SCID is a semistructured interview that enables trained interviewers to make both current and lifetime psychiatric diagnoses based on DSM-IV criteria. In our study, only the PTSD module was administered, and current and lifetime incidence of PTSD was assessed. Diagnostic interviews were conducted by one of two trained clinical psychology graduate students. Interviews were taped, and interrater reliability for PTSD diagnoses was 93%. In those cases in which interviewers differed in diagnosis, interviewers met and came to a consensus on the diagnosis.

Posttraumatic stress disorder symptomatology 1 month after the accident was also assessed with the IES (Horowitz et al 1979). The IES is a 15-item questionnaire used to assess the presence and intensity of intrusive thoughts and avoidant behaviors relevant to the MVA occurring in the week before the follow-up session. Items can be summed to provide a total score (Chronbach's α for the present study = .92) as well as two subscale scores that assess the frequency of intrusive thoughts (Intrusion subscale; $\alpha = .85$) and avoidant thoughts and behaviors (Avoidance subscale; $\alpha = .85$).

URINARY CATECHOLAMINES AND CORTISOL. Free urinary catecholamine assays were conducted by Quest Diagnostics (San Juan Capistrano, CA), and cortisol assays were conducted by the Cleveland Clinic. Free urinary catecholamines were assayed following standard high-pressure liquid chromatography procedures using reverse phase chromatography with electrochemical detection. Only dopamine levels were significantly correlated with volume of urine excreted ($r = .48$, $p < .01$), therefore dopamine levels were computed as μg/dL of urine. Epinephrine and norepinephrine levels were computed as amount per 15-hour sample (μg/15 hours). Urinary cortisol levels were measured by flourescent polarization immunoassay (Abbott TDx Diagnostics, Abbott Laboratories, Abbott Park, IL) and were calculated as amount per 15-hour sample (μg/15 hours). Because urinary cortisol levels were also correlated with urine volume ($r = .27$, $p = .05$), analyses were also conducted on cortisol levels per volume of urine excreted (μg/dL). According to laboratory procedures, all samples were run in duplicate, and those differing by more than 20% were reassayed. No duplicates in the present study differed by more than 20%. Participants who were and were not subsequently diagnosed with PTSD did not differ in urinary excretion of creatinine [$F(1,53) = 1.3$, $p = .26$].

Results

Statistical Analyses

Initial one-way analyses of variance (ANOVAs) and chi-square tests were conducted to examine differences in demographics between PTSD and non-PTSD patients. Pearson product correlations were conducted to determine covariates for analyses. Differences between PTSD groups in urinary hormone levels were examined using ANOVAs and analyses of covariance (ANCOVAs). Finally, the relationship between PTSD symptomatology and urinary hormone levels were analyzed with hierarchical multiple regression analyses.

PTSD Incidence

One month after the accident, 9 out of the 55 patients (16%) met full PTSD diagnostic criteria. There were no differences in age, income, or education between those patients subsequently diagnosed with PTSD and those who did not meet diagnostic criteria. In addition, PTSD and non-PTSD participants did not differ in time of admission and thus, initiation of urine collection [$F(1,52) = 0.00$, $p <= .997$]; however, African American women were significantly overrepresented amongst the PTSD patients [$\chi^2(3, N = 55) = 24.6$, $p < .01$]. All four African American women in the study met threshold PTSD diagnostic criteria. For means of comparison, one African American man, one European American man, and three European American women met PTSD criteria. Because of the small number of minority participants, it is impossible to draw any conclusions based on ethnicity. Excluding these participants from our analysis of differences between PTSD and non-PTSD victims would dramatically decrease power to examine between-group differences in an already small sample; however, examining

Table 1. Mean (SD) Urinary Hormone Levels between Participants Who Did and Did Not Report Experiencing Intrusive Thoughts at the Initial, In-Hospital Assessment

	Intrusive Thoughts (n = 21)	No intrusive Thoughts (n = 30)
Cortisol (μg/dL; square root transformations)	3.82 (2.24)[a]	5.50 (2.68)
Cortisol (μg/15 hours; square root transformations)	14.49 (6.68)[a]	22.80 (10.85)
Epinephrine (μg/15 hours)	17.18 (11.40)[a]	29.36 (24.01)
Norepinephrine (μg/15 hours; square root transformations)	7.22 (2.25)	6.41 (2.37)
Dopamine (μg/dL)	0.17 (0.14)	0.14 (0.06)

[a] $p < .05$.

Table 2. Means (SDs) of Urinary Hormones and IES Scores for the PTSD and Non-PTSD Groups

	PTSD (n = 9)	Non-PTSD (n = 46)
Cortisol (μg/dL; square root transformations)	2.93 (1.42)[b]	5.10 (2.60)
Cortisol (μg/15 hours; square root transformations)	11.45 (5.24)[c]	21.04 (9.83)
Epinephrine (μg/15 hours)	14.78 (8.78)	27.63 (24.11)
Norepinephrine (μg/15 hours; square root transformations)	5.43 (1.10)[a]	7.17 (2.57)
Dopamine (μg/dL)	0.12 (.06)	0.16 (.11)
Total IES score	48.67 (11.60)[d]	17.31 (14.22)
IES intrusion subscale score	25.44 (5.7)[d]	9.18 (7.24)
IES avoidance subscale score	23.22 (7.56)[d]	8.13 (7.67)

IES, Impact of Event Scale; PTSD, posttraumatic stress disorder.
[a] $p = .053$.
[b] $p < .05$.
[c] $p < .01$.
[d] $p < .001$.

differences in urinary hormones across a continuous measure of PTSD symptomatology (IES scores) allows us to conduct analyses both with and without the African American women. Therefore, initial correlation–regression analyses were run on all participants, and to demonstrate that significant results were not driven by the African American women, identical analyses were performed excluding this subset of participants.

Differences in Urinary Hormones between PTSD Groups

Initial one-way ANOVAs were computed to examine differences in urinary hormone levels between participants who did ($n = 21$) and did not ($n = 30$) report experiencing intrusive thoughts at the initial assessment. Because of nonnormal variation, square root transformations were conducted on the cortisol and norepinephrine data. Results revealed that victims who reported experiencing intrusive thoughts had significantly lower urinary cortisol and epinephrine levels than victims who did not report having intrusive thoughts [Fs(1,50) = 5.47 and 9.70, ps $< .05$ for cortisol per volume and per 15 hours respectively [$F(1,47) = 4.58$, $p < .05$ for epinephrine; Table 1].

Additional ANOVAs were computed to examine differences in 15-hour urinary hormone levels between those participants who did (PTSD group) and did not (non-PTSD group) subsequently meet threshold PTSD criteria 1 month after their accidents. Results revealed that PTSD patients had significantly lower levels of urinary cortisol (μg/15 hours) in the immediate aftermath of their accidents than did patients who did not meet diagnostic criteria [$F(1,54) = 8.0$, $p < .01$; Table 2]. Similar results were found for cortisol corrected for volume [μg/dL; $F(1,54) = 5.9$, $p < .05$]. Because our study did not examine 15-hour urinary cortisol levels in a healthy control group, it may be informative to present values provided by prior research. Schaeffer and Baum (1984) reported 15-hour urinary cortisol levels in 38 chronically stressed residents of Three Mile Island (TMI) and 83 control participants. Cortisol levels of control participants averaged 5.8–6.6 μg/15 hours, whereas TMI residents averaged 19.3 μg/15 hours. Their collection procedures differed from those of our study in that Schaeffer and Baum's participants were not catheterized, and all urine samples were collected from 6 PM to 9 AM. Nonetheless, these results suggest that, in comparison with control participants, participants in our study excreted elevated levels of cortisol, with PTSD patients demonstrating lower elevations.

To determine potential covariates, initial zero-order Pearson product correlations were calculated between urinary hormone levels and time of admission, ISS scores, and foods and beverages (caffeine and alcohol) ingested and cigarettes smoked in the 24 hours before the accident (Table 3). Only ISS scores were significantly correlated with hormone levels, so ISS scores were used as covariates in the following analyses. Prior history of PTSD diagnosis has also been shown to increase the risk of subsequent PTSD (Ursano et al 1999). Two PTSD patients and one non-PTSD patient had previously met diagnostic criteria for PTSD stemming from a previous car accident, finding a child who committed suicide, and an attempted rape. The average cortisol levels for these participants were 2.02 ± .72 μg/dL (9.10 ± 3.66 μg/15 hours). Chi-square analyses revealed that current PTSD patients were more likely to have previously met PTSD diagnosis [$\chi^2(1, n = 55) = 5.87$, $p < .05$], so lifetime incidence of PTSD was also included as a covariate. After covarying for injury severity and lifetime incidence of PTSD, previous significant differences in urinary cortisol between PTSD and non-PTSD participants became nonsignificant ($p > .17$).

Table 3. Correlations between Selected Control Variables and Urinary Hormone Levels

Control variable	Cortisol (μg/dL)	Cortisol (μg/15 hour)	Epinephrine (μg/15 hour)	Norepinephrine (μg/15 hour)	Dopamine (μg/dL)
Time of admit	−.009	−.063	.161	.043	−.052
Injury Severity Score	.468[b]	.496[b]	.476[b]	.244	.051
Lifetime incidence of PTSD	−.257[a]	−.255[a]	−.130	−.083	−.023
Number of caffeinated beverages	−.007	−.115	−.043	.155	−.126
Blood alcohol concentration	−.034	.219	−.086	.136	.176
Number of cigarettes	.078	.047	−.062	.018	.143

PTSD, posttraumatic stress disorder.
[a] $p = .06$.
[b] $p < .01$.

Testing Relationships between PTSD Symptomatology and Urinary Hormone Levels

Zero-order Pearson product correlations were calculated to examine the relationship between urinary hormone levels in the first 15 hours after the accident and intrusive and avoidant thoughts 1 month later (Table 4). In addition, to test the ability of urinary hormone levels to predict intrusive and avoidant thoughts, we conducted hierarchical multiple regressions regressing total IES scores on urinary hormone levels. Lifetime incidence of PTSD was entered on the first step, followed by ISS score on the second, and cortisol on the third. After removing variance associated with lifetime PTSD incidence and injury severity, urinary cortisol (μg/15 hours) continued to predict a significant percentage of the variance in total IES score ($\Delta R^2 = .097$, $p < .05$; Table 5). Similar regressions conducted separately on the intrusion ($\Delta R^2 = .088$, $p < .05$) and avoidance ($\Delta R^2 = .092$, $p < .05$) subscales of the IES produced similar results, suggesting that urinary cortisol levels were related equally to both intrusive and avoidant thoughts and behaviors. Running the identical analyses excluding African American women did not change the ΔR^2 or significance values. Analyses predicting total IES and avoidance subscale scores using cortisol per volume of urine excreted (μg/dL) revealed similar results, ($\Delta R^2 = .071$ and .074, $p < .05$, respectively). Cortisol per volume of urine did not predict a significant percentage of the variance in intrusion subscale scores, however ($\Delta R^2 = .058$, $p < = .077$). Analyses predicting IES scores from urinary epinephrine revealed that epinephrine levels did not predict a significant percentage of the variance in intrusive and avoidant thoughts and behaviors ($p > .15$)

Discussion

Numerous recent reports have demonstrated reliable differences in levels of urinary catecholamines and cortisol in PTSD and non-PTSD patients who were similarly exposed to a traumatic event. Nonetheless, these studies have typically examined victims presenting with chronic (often decades long) PTSD, making it impossible to conclude whether altered neuroendocrine levels reflect long-term presence of psychopathology or altered or persistent response to the initial traumatic event. Our study demonstrated differing urinary cortisol levels during and immediately following the traumatic event in patients subsequently diagnosed with acute PTSD and those who did not meet PTSD criteria.

Although we had no formal measure of PTSD administered at the hospital, MVA victims who reported experiencing intrusive thoughts within the first few days of their accident had lower cortisol and epinephrine levels than victims who did not report the presence of intrusive thoughts. These findings suggest that initial hormone levels in the immediate aftermath of an accident may be associated with the development of intrusive thoughts.

Table 4. Correlations between IES Scores and Urinary Hormone Levels

	Cortisol (μg/dL)	Cortisol (μg/15 hour)	Epinephrine (μg/15 hour)	Norepinephrine (μg/15 hour)	Dopamine (μg/dL)
IES total score	−.424[b]	−.463[b]	−.368[a]	−.178	−.325[a]
	(−.391[b])	(−.415)[b]	(−.375[a])	(−.068)	(−.285)
Intrusion subscale	−.408[b]	−.456[b]	−.355[a]	−.201	−.363[a]
	(−.368[a])	(−.410[b])	(−.362[a])	(−.113)	(−.332[a])
Avoidance subscale	−.412[b]	−.440[b]	−.353[a]	−.143	−.264
	(−.384[b])	(−.387[b])	(−.356[a])	(−.015)	(−.210)

Correlations excluding African American women are presented in parentheses. IES, Impact of Event Scale.
[a] $p < .05$.
[b] $p < .01$.

Table 5. Hierarchical Regressions Predicting Impact of Event (IES) Total Scores 1 Month after the Accident from Urinary Cortisol Levels (μg/dL and μg/hour) during the First 15 Hours after the Accident

Step no.	Variables	R^2	β	df1	df2	ΔR^2	F of ΔR^2
1	Lifetime incidence of PTSD	.079	.281	1	46	.079	3.93[a]
2	Injury Severity Score	.151	−.276	1	45	.072	3.84
3	Cortisol (μg/15 hours)	.248	−.369	1	44	.097	5.66[a]
3	Cortisol (μg/dL)	.222	−.308	1	44	.071	4.01[a]

Because the initial two steps are identical for both regressions, only the third step is shown for the second regression. Significance of first regression model, $F(3,44) = 4.83$, $p = .005$; Significance of second regression model, $F(3,44) = 4.19$, $p = .011$.

[a] $p < .05$.

Additional research is necessary before any strong conclusions can be drawn concerning the relationship between initial intrusive thoughts and hormonal responses to trauma.

Motor vehicle accident victims subsequently diagnosed with acute PTSD had lower urinary cortisol levels in the first 15 hours after their accident than did victims who did not meet diagnostic criteria. These differences disappeared after controlling for injury severity and lifetime incidence of PTSD, however, perhaps because of a lack of power afforded by small sample size. Further, African American women comprised the majority of our PTSD patients, so interpretation of the present results is complicated by potential differences in race and gender. We were unable to find any reference to differing levels of urinary cortisol levels between African American and European American women; however, Yanovski and colleagues have reported that black and white girls and women do not differ in plasma cortisol levels despite differences in plasma adrenocorticotropic hormone (Yanovski et al 1996a, 1996b). This suggests that our observed differences in cortisol may have been driven primarily by responses to the MVA and not by differences in race.

Hierarchical multiple regression analyses indicated that urinary cortisol levels predicted a significant percentage of the variance in intrusive and avoidant thoughts and behaviors even after controlling for the influence of injury severity and lifetime PTSD incidence. Although the percentage of variance accounted for by urinary cortisol was small (7–10%), the correlations between urinary levels and PTSD symptomatology are quite robust considering that we are predicting self-report measures of intrusive and avoidant thoughts from physiologic hormone levels collected a month earlier (and before the participant knew about the study). This relationship persisted even after eliminating African American women from the analyses.

The present finding that subsequent PTSD symptomatology is associated with initial hormonal responses to the MVA suggests that initial cortisol levels may contribute, in part, to subsequent acute PTSD. Pitman was the first to hypothesize that the pattern of neuroendocrine responding observed in chronic PTSD could, if present during the traumatic event, lead to the development of "overconsolidated" memories or "superconditioning" during periods of extreme stress (Pitman 1989; Pitman et al 1991). Yehuda and colleagues expanded this theory to suggest that exaggerated catecholamine increases without the regulatory influence of accompanying cortisol increases could lead to inappropriate memory consolidation and subsequent PTSD (Yehuda and Harvey 1997; Yehuda et al 1998).

The present study did not find consistent relationships between catecholamine levels during and immediately following the MVA and subsequent PTSD symptomatology. Although urinary epinephrine levels were significantly correlated with IES scores, PTSD and non-PTSD patients did not differ in levels of epinephrine. Studies that have reported differences in catecholamine levels between PTSD and non-PTSD participants typically have examined urinary hormone levels decades after the traumatic event and may reflect chronic hyperarousal to reminiscent and nonreminiscent stimuli (Kosten et al 1987; Lemieux and Coe 1995; Yehuda et al 1992). Although epinephrine levels were negatively correlated with intrusive and avoidant symptoms in our sample, the levels we observed are much higher than those observed in other samples (Baum et al 1983; McKinnon et al 1989). It is possible that during the initial life-threatening traumatic event, all victims respond with relatively high levels of catecholamines; however, elevated catecholamines combined with low levels of cortisol during and immediately following the traumatic event may contribute to abnormalities in memory consolidation during the initial phases of responding to the MVA, resulting in PTSD symptoms (Charney et al 1994; van der Kolk 1996).

Our study has a number of limitations. The small sample size and relatively few participants meeting PTSD diagnostic criteria suggest viewing the between-group analyses with caution. In addition, because all the African American women in our sample developed PTSD, making conclusions regarding contributors to group differences is difficult. To reduce participant load, we only administered the PTSD module of the SCID, and as such, we have no measure of possible conditions occurring comorbid with PTSD (i.e., depression, substance abuse) or the relation of

urinary hormones to comorbid disorders. Further, a number of variables could contribute to alterations in urinary hormone levels. This necessitates decreasing the sample size by eliminating patients who suffered from chronic diseases or who had been taking any medications that may influence urinary neuroendocrine levels.

In addition, a 15-hour urine sample reflects responses to much more than just the MVA. These hormone levels reflect levels present from the last void before the accident through urine collected 15 hours after admission and can be affected by a number of pre-, peri-, and posttraumatic variables. We attempted to control for as many of these confounds as possible by eliminating patients who were taking any medications that could affect hormone levels and by controlling for injury severity. Future research may examine shorter sampling durations to limit the impact of postaccident variables on urinary hormone levels.

Despite these shortcomings, the present research demonstrates that patients subsequently diagnosed or not diagnosed with PTSD after a serious car accident differed in initial urinary cortisol levels and that initial cortisol levels can predict subsequent PTSD symptomatology. Further research is needed to determine possible preexisting variables (e.g., history of traumatic experiences, personality variables) that may affect physiologic responses to traumatic events, in effect predisposing certain victims to PTSD.

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