

Enhanced Memory Performance on an Internal–Internal Source Monitoring Test Following Acute Psychosocial Stress

T. Smeets, M. Jellicic, H. Merckelbach, M. Peters, A. Fett, J. Taverniers, C. Henquet, and J. Dautzenberg
Maastricht University

Research on the effect of acute stress and high levels of glucocorticoids on memory has largely focused on memory tasks involving the medial temporal lobe (e.g., declarative memory). Less is known, however, about the effects of stress and glucocorticoids on more strategic memory processes regulated by the prefrontal cortex (e.g., source monitoring). In the current study, the authors investigated whether exposure to acute psychosocial stress would result in altered source monitoring performance relative to the performance of a nonstressed control group. To this end, the authors assigned nonsmoking, healthy, young men to either a stress ($n = 22$) or a control ($n = 18$) condition, after which the men were given an internal source monitoring test. Results show that relative to control participants, stressed participants made fewer source monitoring errors. This study suggests that stress may have differential effects on memory, depending on whether the memory test is regulated by the prefrontal cortex or the medial temporal lobe.

Keywords: acute stress, glucocorticoids (GCs), source monitoring, prefrontal cortex (PFC), medial temporal lobe (MTL)

Source monitoring refers to cognitive processes that are involved in making attributions about the origins of memories, knowledge, and beliefs (Johnson, Hashtroudi, & Lindsay, 1993). Consistent with the source monitoring framework proposed by Johnson et al. (1993), three major types of source monitoring can be distinguished. Reality monitoring refers to the ability to discriminate between internally generated memories and externally generated memories (e.g., “Did I hear that on the news or did I imagine that?”). Alternatively, the ability to distinguish between memories of two externally derived sources is termed external source monitoring (e.g., “Did I hear that on the news or did Tim tell me that?”). Finally, internal source monitoring is said to occur when people have to discriminate between two internally generated memories (“Did I say that out loud or did I merely think that?”). Inaccuracies in judging the source of a memory (i.e., source monitoring errors) are by no means uncommon and can have important ramifications, as evidenced by mistaken eyewitness testimonies in which fragments of real experiences are accurately recalled but are attributed to the wrong person, time, or location (e.g., Ross, Ceci, Dunning, & Toglia, 1994).

Accurate source monitoring is dependent on cognitive processes that initially bind features into complex memories and on processes that reactivate and evaluate such features (Johnson et al., 1993). Brain areas involved in these cognitive processes include the medial temporal regions, which are essential for binding and reactivation, and frontal regions, particularly lateral frontal regions, which are of special importance for strategic retrieval and evaluation of features of memories (Johnson et al., 1993; Moscovitch, 1994). Evidence for the vital role of the prefrontal cortex (PFC) in source monitoring comes from clinical studies that involved patients with frontal lobe damage (e.g., Janowsky, Shimamura, & Squire, 1989; Schacter, Harbluk, & McLachlan, 1984) and, more recently, from studies that involved neuroimaging techniques (e.g., Dobbins, Foley, Schacter, & Wagner, 2002; Dobbins, Rice, Wagner, & Schacter, 2003; Mitchell, Johnson, Raye, & Greene, 2004; Nolde, Johnson, & D’Esposito, 1998; Ranganath, Johnson, & D’Esposito, 2000; Raye, Johnson, Mitchell, Nolde, & D’Esposito, 2000; Rugg, Fletcher, Chua, & Dolan, 1999; Slotnick, Moo, Segal, & Hart, 2003). On the basis of this type of empirical evidence, Johnson and Raye (1998, 2000; see also Nolde et al., 1998) suggested that the right PFC supports heuristic processing (e.g., item recognition), whereas the left PFC (possibly together with the right PFC) subserves source monitoring.

A vast amount of animal (e.g., McGaugh & Roozendaal, 2002; Roozendaal, 2000) and human (for reviews, see Het, Ramlow, & Wolf, 2005; Lupien & Lepage, 2001; Wolf, 2003) research has shown that glucocorticoid (GC) secretion from the adrenal cortex during stressful episodes may have facilitating as well as disruptive effects on memory formation, consolidation, and retrieval. Research suggests that memory facilitation may occur when GC receptors are moderately stimulated while high-affinity mineralocorticoid receptors are fully saturated. In contrast, when GC receptors are extremely occupied under stressful circumstances, high GC levels may exert detrimental effects on memory (e.g., de Kloet,

T. Smeets, M. Jellicic, H. Merckelbach, M. Peters, A. Fett, and J. Taverniers, Department of Experimental Psychology, Maastricht University, Maastricht, the Netherlands; C. Henquet and J. Dautzenberg, Department of Psychiatry and Neuropsychology, Maastricht University.

This research was supported by the Netherlands Organization for Scientific Research (NWO) Grant 452-02-006 awarded to M. Jellicic. We thank José Sulon for conducting the cortisol analyses at the Université de Liège (Belgium).

Correspondence concerning this article should be addressed to T. Smeets, Department of Experimental Psychology, Maastricht University, P.O. Box 616, 6200 MD Maastricht, the Netherlands. E-mail: tom.smeets@psychology.unimaas.nl

Oitzl, & Joëls, 1999; Oitzl & de Kloet, 1992; Reul & de Kloet, 1985). Moreover, it seems that adrenergic activation of the basolateral amygdala and the hippocampus is a prerequisite for GCs to impair retrieval (e.g., Roozendaal, Hahn, Nathan, de Quervain, & McGaugh, 2004). Together with brain regions such as the PFC and the hippocampus, the activated basolateral amygdala along with the effects of emotional arousal can mediate the effects of GCs on memory (e.g., McGaugh & Roozendaal, 2002).

In a series of research articles, Moscovitch and colleagues have convincingly argued that medial temporal lobe (MTL) contributions to memory are distinct from PFC involvement in memory (e.g., Moscovitch, 1992; Moscovitch & Winocur, 1992; for a recent review, see Moscovitch & Winocur, 2002). These authors asserted that although there is no question as to its importance in memory processes, the MTL is primarily involved with the content of memories. The frontal cortex, on the other hand, operates on the input and output of the MTL in encoding and in performing heuristic and strategic operations (e.g., determining the source of a memory; Johnson et al., 1993; Moscovitch & Winocur, 2002). Whereas the effects of acute stress and high GC levels on memory recollection involving the MTL have been well studied, their effects on more strategic memory processes (e.g., retrieval cue generation and specification, verification, source monitoring) that are under the regulation of the PFC remain largely unknown.¹ It is interesting that animal studies have shown that the PFC is a significant target for negative feedback actions of GCs and that chronic GC administration and behavioral stress can result in dendritic reorganization in the medial PFC (e.g., Charney, 2004; Radley et al., 2004; Sanchez, Young, Plotsky, & Insel, 2000). In conjunction with other findings implicating the PFC as a target for circulating GCs (e.g., Lupien & Lepage, 2001), this suggests that high levels of circulating GCs may stimulate PFC-regulated source memory.

In the present study, we investigated whether exposure to an acute psychosocial stressor (i.e., the Trier Social Stress Test [TSST]; see Kirschbaum, Pirke, & Hellhammer, 1993) and/or the consequential cortisol elevations would result in altered source monitoring performance relative to a nonstressed control group. If acute psychosocial stress and high-GC stress responses affect PFC-regulated source monitoring performance similarly to MTL-mediated memory processes, one would expect that stressed participants would exhibit more source monitoring errors than nonstressed control participants. On the other hand, given that there are studies showing increased cerebral blood flow in the right PFC during psychological stress (e.g., Wang et al., 2005), one could speculate that acute stress and/or high levels of GCs have enhancing effects on source memory performance. Note, however, that source monitoring appears to be mainly lateralized to the left PFC and that increased blood flow in the right PFC may instead have impairing effects by depleting resources that are necessary for accurate source monitoring.

Method

Participants

Our sample consisted of 40 young, healthy undergraduate students. Participants were nonsmokers with a normal body mass index, and all were native Dutch speakers. To rule out the possibility that gender differences

could play a confounding role in cortisol reactions to the stress task (for a review, see Kudielka & Kirschbaum, 2005), we included only men in the study. The mean age was 19.2 years ($SD = 1.4$). Participants were excluded from the study if they suffered from cardiovascular diseases or endocrine disorders or if they were on any kind of medication. Test protocols were approved by the standing ethics committee of the Faculty of Psychology of Maastricht University. All participants signed a written informed consent and were given a small financial compensation (12.50€; approximately U.S.\$15) for completing the experiment.

Materials

Several personality questionnaires, including the Thought-Action Fusion Scale (Shafran, Thordarson, & Rachman, 1996), the Creative Experiences Questionnaire (Merckelbach, Horselenberg, & Muris, 2001), the Community Assessment of Psychic Experiences-Trait Scale (Stefanis et al., 2002), and the Community Assessment of Psychic Experiences-State Scale (Stefanis et al., 2002), were administered as filler tasks. Psychometric data derived from these questionnaires will not be addressed here.

Measures

TSST. The TSST (Kirschbaum et al., 1993) is a psychosocial challenge test that consists of a 10 min preparation period, a 5 min free speech task, and a 5 min mental arithmetic task performed in front of an audience while the participant is being videotaped. The TSST is a valid and reliable procedure to induce physiological stress responses in children, young adults, and older adults (e.g., Kirschbaum, Wüst, & Hellhammer, 1992; Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004; Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004). In a recent meta-analysis, the TSST was found to provoke the most robust physiological stress responses (i.e., cortisol stress responses) relative to various other laboratory stress tasks (Dickerson & Kemeny, 2004). Though the original TSST protocol has served our lab well in eliciting stress responses (e.g., Smeets, Jelicic, & Merckelbach, 2006a, 2006b), in the current study we used a personalized TSST. In this modified version of the TSST, participants were asked to describe their personality characteristics in a foreign language (i.e., in English) in front of an audience over a period of 10 min while the participant was standing. This performance was preceded by a 5 min preparatory period. During the final 5 min, the participants were asked to solve 30 mathematical problems of above-average difficulty. For each problem, they were given a maximum of 8 s.

Source monitoring test (SMT). This study's SMT was based on Parks's (1997) study and was developed to determine participants' ability to discriminate mere thoughts from actually verbalized thoughts. That is, participants had to indicate whether they had really verbalized or only imaged answers to earlier presented questions. This test has been proven to be sensitive to differences in source monitoring abilities (see Henquet, Krabbendam, Dautzenberg, Jolles, & Merckelbach, 2005). SMT materials consisted of 24 nonintrusive questions concerning personal history (e.g., "When were you born?") and opinion (e.g., "What foods do you like?"). Questions were presented on a computer screen, with a computer program specifically developed for this purpose. The SMT involved 16 trials. On half of the trials, single questions were presented. On the other half, questions were presented in pairs, with one question located at the top half of the computer screen and the other located at the bottom half of the screen. Questions were presented, after which a white screen was shown for 3 s. Participants had to prepare an answer to the presented questions. On single question trials, the word "answer" appeared 1 s after presentation of

¹ One important exception to this is work on the link between stress and working memory (for examples, see al'Absi, Hugdahl, & Lavallo, 2002; Elzinga & Roelofs, 2005; Lupien, Gillin, & Hauger, 1999).

the white screen. On dual question trials, either the words “answer top” or “answer bottom” appeared. Thus, here participants had to prepare an answer to both questions, but they verbalized only one answer. This resulted in verbalizing answers to 16 questions, whereas 8 answers were covertly prepared but never verbalized. Single questions and pairs were presented in a quasi-random order, and two parallel versions were used that were counterbalanced across participants. An experimenter was present to monitor whether participants actually verbalized the answers. All participants were capable of answering the questions; which indicated that the questions were simple and direct. Participants were instructed to indicate when they had prepared an answer for the questions. Preparation time varied among participants but never took longer than 10 s. Given this constellation, we have every reason to believe that the participants actually did prepare answers during the preparation phase of the paired-question trials, as opposed to the participants merely remembering the questions. Following the presentation of the questions (i.e., source memory acquisition [SMA]) and exposure to the TSST or filler task, participants were given a surprise recognition test and SMT (R + SMT). In this test, participants saw original questions, each paired with a new question of the same content and form. For example, the old item, “When were you born?” was presented along with the new item “Where were you born?” Participants were asked to identify for each of the 24 pairs of old and new items the question they had seen before (i.e., the recognition memory aspect of the test). Further, the participants had to indicate whether they verbalized answers to the old items or only thought about an answer (i.e., the source memory aspect of the test).

Saliva sampling and biochemical analyses. Cortisol data were obtained with cotton Salivette (Sarstedt, Nümbrecht, Germany) devices. The saliva samples that were not centrifuged were stored at -40°C immediately on collection. Salivary free cortisol levels were determined in duplicate by direct radioimmunoassay (University of Liège, Belgium), including a competition reaction between ^{125}I iodohistamine-cortisol and anticortisol serum made against the 3-carboxymethyl-oxime-bovine serum albumin conjugate. After overnight incubation at 4°C of 100 μl saliva, separation of free and antibody-bound ^{125}I iodohistamine-cortisol was performed via a conventional second-antibody method. In order to reduce sources of variability, we analyzed all 7 samples taken from each participant (see below) in the same assay. Two Salivette devices contained insufficient saliva for analyses and were treated as missing values. Mean intra- and interassay coefficients of variation were less than 4.5% and 8.5%, respectively.

Design

Participants were quasi-randomly assigned to one of two groups. Participants who were exposed to a modified version of the TSST (Kirsch-

baum et al., 1993) served as the stress group ($n = 22$), whereas other participants were assigned to a control group that included a filler task ($n = 18$). The two groups did not differ with respect to age (stress group: $M = 19.3$ years, $SD = 1.6$; control group: $M = 19.0$ years, $SD = 1.0$), $t(38) = 0.73$, $p = .47$.

Procedure

All participants were individually tested in experimental sessions run between 9 a.m. and 10 a.m., 10 a.m. and 11 a.m., or 11 a.m. and 12 noon. To allow for objective controlled cortisol sampling, we deprived all participants of food, drinks, and heavy exercise at least 1 hr prior to the test phase. After arrival in the laboratory, participants were given a resting phase of 20 min during which they signed a consent form and completed the Community Assessment of Psychic Experiences–Trait Scale. Subsequently, participants performed the SMA. All participants then either took part in the modified version of the TSST or received a filler task (i.e., reading a neutral text) of equal duration (see Figure 1). Afterward, participants filled out the Creative Experiences Questionnaire and completed the R + SMT. Finally, during a 10-min recovery period, participants completed the Community Assessment of Psychic Experiences–State Scale and the Thought-Action Fusion Scale. Cortisol samples were obtained over a 60-min period at seven measurement points (i.e., -20 , -10 , 0 , $+10$, $+20$, $+30$, and $+40$ min, with reference to the onset time [T] of the stressor or filler task; also see Figure 1). The entire test session never exceeded 1 hr. Participants were debriefed, paid, and thanked for their participation.

Statistical Analyses

Cortisol responses were analyzed with a 2 (Group: stress, control) \times 7 (Time: T $- 20$, T $- 10$, T0, T $+ 10$, T $+ 20$, T $+ 30$, T $+ 40$) analysis of variance, with the last factor being a repeated measure. For each participant, we computed an individual cortisol response (i.e., delta increase in cortisol) defined as peak cortisol level (T $+ 10$, T $+ 20$, T $+ 30$, or T $+ 40$) after the TSST or filler task minus prestress cortisol level (T $- 10$). Note that T $- 10$ rather than T $- 20$ was used as the prestress cortisol level because the novelty of the first cortisol sampling procedure may have amplified the T $- 20$ reading. Delta responses were analyzed with an independent samples t test. Performance on the R + SMT was analyzed as follows. First, we calculated a recognition memory score by summing the number of correctly identified old items divided by the number of items (i.e., proportion of correct recognition of memory items presented during the SMA). Second, a proportion of correct source hits was calculated, conditioned on correct memory recognition of the items. That is, a source hit was only scored if the matching stimulus sentence was correctly

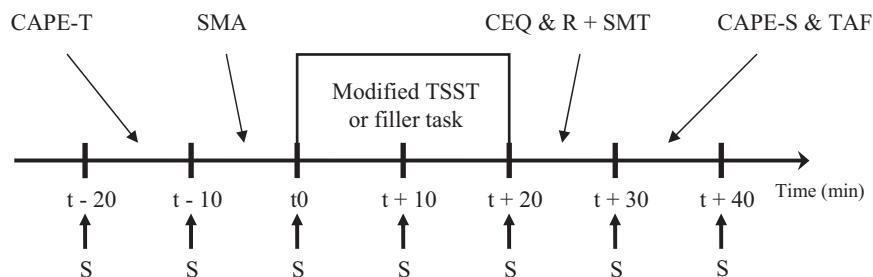


Figure 1. Timeline for completing personality questionnaires, performing memory tests, and sampling saliva are shown. The Community Assessment of Psychic Experiences–Trait Scale (CAPE-T), Community Assessment of Psychic Experiences–State Scale (CAPE-S), Thought-Action Fusion Scale (TAF), and Creative Experiences Questionnaire (CEQ) served as filler tasks. The CAPE-T was 10 min; the source memory acquisition (SMA) test was 10 min; the CEQ was 5 min; the recognition and source monitoring test (R + SMT) was 5 min; the CAPE-S was 8 min; the TAF was 2 min. S = Salivette device used for saliva sampling; t = time of the onset of the stressor or filler task; TSST = Trier Social Stress Test.

recognized. Similarly, source false-alarm rates (i.e., the number of erroneous claims of having verbalized an answer that in fact was covertly prepared) were calculated conditioned on correct memory recognition of the items. Finally, measures of accurate and biased discrimination between internal and external thoughts (i.e., a discrimination index [Pr] and bias index [Br], respectively) were determined with the two-high threshold theory (Snodgrass & Corwin, 1988; see also Corwin, 1994). Thus, the Pr was defined as $Pr = [\text{number of source hits} - \text{number of source false alarms}]$. The Br was defined as $Br = [\text{number of source false alarms} / (1 - Pr)]$. For each of the R + SMT parameters, an independent samples (group: stress, control) t test was conducted. To evaluate the role of cortisol in acute stress effects on source monitoring indices, we defined low- and high-cortisol responder groups with a median split (see below). Independent samples t tests were then used to check whether low-cortisol responders differed from high-cortisol responders in their source monitoring performance. Within the stress group, Spearman's rho correlations (two-tailed test) between delta increases in cortisol and source monitoring indices were calculated. When sphericity assumptions were violated, Greenhouse-Geisser corrected p values were reported. Alpha was set at .05 unless otherwise specified and adjusted (Bonferroni) for multiple comparisons as necessary.

Results

Prestress Cortisol Analyses

None of the participants self-reported any violations of the prohibition on eating, drinking, and heavy exercise. Stress and control groups did not differ with regard to prestress cortisol levels at $T - 20$, $T - 10$, and $T0$ ($t_s < 1.60$; $p_s > .12$).

Cortisol Stress Responses

As expected, a significant main effect of time, $F(6, 216) = 6.12$, $p = .003$, and a significant Group \times Time interaction, $F(6, 216) = 24.39$, $p < .001$, were found in the absence of a main effect of group, $F(1, 36) = 1.32$, $p = .26$. Delta increases in cortisol differed significantly between groups, $t(38) = -5.66$, $p < .001$, with means of 7.47 nmol/L ($SD = 5.11$) and 0.24 nmol/L ($SD = 1.96$) for the stress and control groups, respectively. Previous research has indicated that a cortisol increase larger than 2.5 nmol/L reflects a cortisol secretory episode (Van Cauter & Refetoff, 1985) and can be taken as a clear-cut cortisol response (see, e.g., Kirschbaum et al., 1993; Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Schommer, Hellhammer, & Kirschbaum, 2003). In our study, 18 out of 22 (i.e., 82%) participants in the stress group displayed a clear-cut cortisol response. Because of the high variance in cortisol responses to the TSST and following Domes, Heinrichs, Reichwald, and Hautzinger (2002; also see Elzinga & Roelofs, 2005), we conducted a post hoc median split between low- and high-cortisol responders. This resulted in a group of 11 low-cortisol responders, whose mean delta increase was 3.17 nmol/L ($SD = 2.46$), a 75% mean increase, and a group of 11 high-cortisol responders whose mean increase was 11.77 nmol/L ($SD = 2.84$), a 185% increase, $t(20) = 7.59$, $p < .001$. Figure 2 shows increases in cortisol levels throughout the experimental session for low- and high-cortisol responders and non-stressed controls.

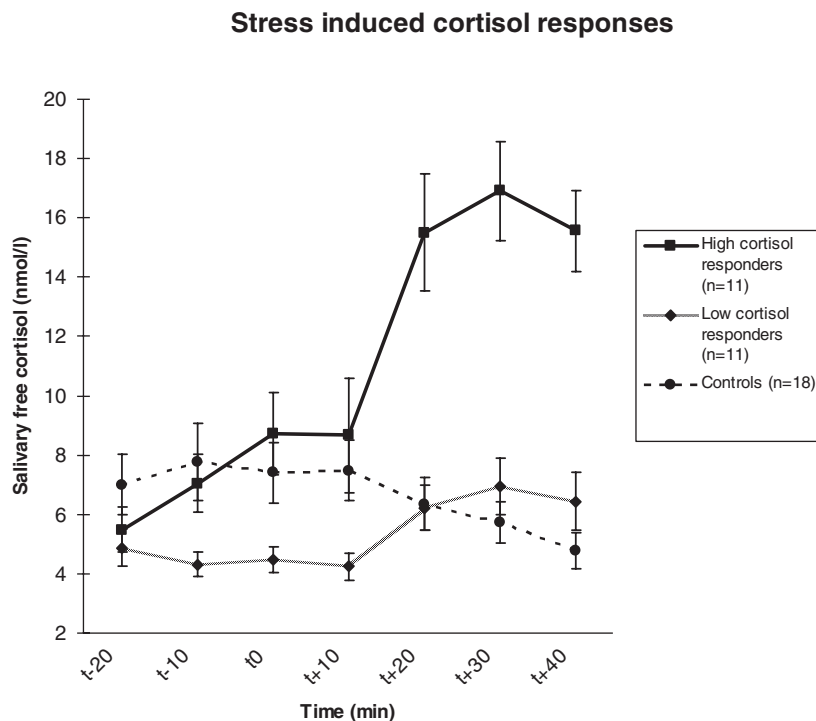


Figure 2. Mean free salivary cortisol levels (nmol/l) for controls and high- and low-cortisol responders in the stress group are shown. Data points indicate cortisol levels throughout the session. Error bars represent standard error of mean (SE). t = time of the onset of the stressor or filler task.

Source Monitoring Performance

Mean scores derived from the SMT for the stress and control group can be found in Table 1. Independent samples *t* tests showed that participants in the stress group outperformed controls with regard to the proportion of correct source hits, the proportion of source false alarms, *Pr*, and the recognition memory component of the SMT ($ps < .05$). Groups, however, did not differ in terms of biased responding ($p > .10$). To check whether these overall group differences could be mediated by the extent of the cortisol elevations, we compared low- and high-cortisol responders. No differences were found ($ts < 1.08$; $ps > .29$) between low- and high-cortisol responders on the proportion of correct source hits (.88 and .88, respectively), proportion source false alarms (.04 and .03, respectively), recognition memory score (.99 and .98, respectively), *Pr* (.84 and .85, respectively), and response *Br* (.24 and .21, respectively). Furthermore, within-stress group correlations between delta increases in cortisol and source monitoring indices remained nonsignificant ($rs < .28$; $ps > .09$).

Discussion

The main results of this study can be summarized as follows. First, participants exposed to the stressor were better at correctly identifying verbalized and internally prepared items (i.e., source hits) than nonstressed controls. Second, relative to controls, stressed participants were more accurate at discriminating between targets and distracters when the number of errors that were made was taken into account (i.e., *Pr*). Third, participants in the stress group less frequently misclassified imagined thoughts as verbalized answers (i.e., source false alarms). Fourth, these results were obtained in the absence of differences in response bias. Furthermore, relative to nonstressed controls, stressed participants were slightly better at correct recognition of previously presented items. Finally, scores derived from the R + SMT did not differ between low- and high-cortisol responders, suggesting that the magnitude of the cortisol response does not directly influence the effects of acute stress on source monitoring performance.

To the best of our knowledge, this is the first time that the effects of acute stress on source memory have been studied. Our findings are in support of theories contending that the role of the MTL in memory is markedly different from PFC contributions to

memory (e.g., Moscovitch & Winocur, 2002). Although acute stress and/or elevated GC levels tend to undermine declarative memory in a variety of ways (Het et al., 2005; Wolf, 2003), this study suggests that PFC-regulated source monitoring performance benefits from stress. Perhaps it is improved cerebral blood flow to the PFC during psychological stress (e.g., Wang et al., 2005) that accounts for the beneficial effect of stress on source monitoring performance. Alternatively, results showing that source monitoring indices did not differ between high- and low-cortisol responders and that these indices showed no significant correlation with delta cortisol increases seem to suggest that the noradrenergic system (e.g., via the release of noradrenaline) is implicated in the source memory enhancing effect of stress. Clearly, this issue is open to empirical testing. In any event, our findings seem to indicate that stress has differential effects on memory performance, depending on whether the memory test is regulated by the PFC or the MTL.

It should be acknowledged, though, that with the study's design we cannot rule out the possibility that the source memory enhancing effect may be related to the MTL. That is, although neuropsychological (e.g., Janowsky et al., 1989; Schacter et al., 1984) and functional imaging studies (e.g., Dobbins et al., 2002, 2003; Mitchell et al., 2004; Raye et al., 2000; Slotnick et al., 2003) have demonstrated that source monitoring relies critically on the PFC, source monitoring is also related to MTL functioning. However, given that exposure to acute stress prior to retrieval has typically been associated with memory impairing effects (e.g., Domes, Heinrichs, Rimmele, Reichwald, & Hautzinger, 2004; Kuhlmann, Piel, & Wolf, 2005), the present data thus seem to indicate that the source memory enhancing effect is PFC driven.

Because in the current study participants were exposed to the psychosocial stressor only after the SMA phase, our findings showing enhanced source monitoring performance cannot be attributed to differences in encoding between the two groups. Whether acute psychosocial stress given before or during the encoding phase can result in altered source monitoring performance therefore remains to be determined. Research on the effects of acute stress on MTL-regulated declarative memory performance indicates that memory facilitation may occur when GC receptors are moderately stimulated while mineralocorticoid receptors are simultaneously totally saturated. When GC receptors are fully

Table 1
Source Monitoring Performance of Participants Exposed to the Trier Social Stress Test (Stress Group) or the Filler Task (Control Group)

Variable	Stress group		Control group		<i>t</i> (38)	<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>		
Recognition memory ^a	0.99	0.02	0.95	0.07	-2.26	.03
Source hits ^b	0.88	0.07	0.80	0.11	-2.74	.01
Source false-alarm rate ^c	0.03	0.03	0.08	0.09	2.23	.03
Discrimination index ^d	0.85	0.10	0.72	0.18	-2.80	.01
Bias index ^e	0.22	0.25	0.24	0.15	0.21	.83

Note: For the stress group, $n = 22$. For the control group, $n = 18$

^a Proportion of correctly recognized old memory items. ^b Proportion of total correct source attributions.

^c Proportion of false positives. ^d Measure of accurate discrimination (two-high threshold). ^e Measure of biased responding (two-high threshold).

occupied, on the other hand, high GC levels may be disadvantageous to memory (e.g., de Kloet et al., 1999). At present, one can only speculate as to whether such differential effects can also be found for PFC-regulated SMTs. Also, it remains unclear whether higher doses (e.g., via GC administration) would result in enhanced source monitoring performance.

Some comments as to the limitations of this study are worthy of note. First, the study used a relatively homogeneous (i.e., male undergraduate) sample of participants. Therefore, it remains to be seen whether the findings can be generalized to other populations (e.g., older adults, women). Second, in the present study we used an SMT that was specifically designed to elicit internal–internal source monitoring errors. Future work should investigate whether these findings also translate to other (i.e., external–external and internal–external) SMTs. Also note that if participants were able to correctly remember the presentation format for the 8 single (but not the 16 paired) items of the SMA that were verbalized at all times, the participants could infer that they must have verbalized the answer. Finally, to directly compare the effects of acute stress on MTL- and PFC-regulated memory tasks, studies including independent memory tests of both types are needed.

In sum, the present study showed that exposure to acute psychosocial stress can result in improved internal–internal source monitoring performance. Although the current results seem to suggest that cortisol does not play a major role in these effects, the precise neurobiological mechanisms of these effects remain unclear. Future work with functional neuroimaging techniques such as positron emission tomography or functional magnetic resonance imaging could shed further light on this issue.

References

- al'Absi, M., Hugdahl, K., & Lovallo, W. R. (2002). Adrenocortical stress responses and altered working memory performance. *Psychophysiology*, *39*, 95–99.
- Charney, D. S. (2004). Psychobiological mechanisms of resilience and vulnerability: Implications for successful adaptation to extreme stress. *American Journal of Psychiatry*, *161*, 195–216.
- Corwin, J. (1994). On measuring discrimination and response bias: Unequal numbers of targets and distractors and two classes of distractors. *Neuropsychology*, *8*, 110–117.
- de Kloet, E. R., Oitzl, M. S., & Joëls, M. (1999). Stress and cognition: Are corticosteroids good or bad guys? *Trends in Neurosciences*, *22*, 422–426.
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*, 355–391.
- Dobbins, I. G., Foley, H., Schacter, D. L., & Wagner, A. D. (2002). Executive control during episodic retrieval: Multiple prefrontal processes subserve source memory. *Neuron*, *35*, 989–996.
- Dobbins, I. G., Rice, H. J., Wagner, A. D., & Schacter, D. L. (2003). Memory orientation and success: Separable neurocognitive components underlying episodic recognition. *Neuropsychologia*, *41*, 318–333.
- Domes, G., Heinrichs, M., Reichwald, U., & Hautzinger, M. (2002). Hypothalamic-pituitary-adrenal axis reactivity to psychological stress and memory in middle-aged women: High responders exhibit enhanced declarative memory performance. *Psychoneuroendocrinology*, *27*, 843–853.
- Domes, G., Heinrichs, M., Rimmele, U., Reichwald, U., & Hautzinger, M. (2004). Acute stress impairs recognition for positive words: Association with stress-induced cortisol secretion. *Stress*, *7*, 173–181.
- Elzinga, B. M., & Roelofs, K. (2005). Cortisol-induced impairments of working memory require acute sympathetic activation. *Behavioral Neuroscience*, *119*, 98–103.
- Henquet, C., Krabbendam, L., Dautzenberg, J., Jolles, J., & Merckelbach, H. (2005). Confusing thoughts and speech: Source monitoring and psychosis. *Psychiatry Research*, *133*, 57–63.
- Het, S., Ramlow, G., & Wolf, O. T. (2005). A meta-analytic review of the effects of acute cortisol administration on human memory. *Psychoneuroendocrinology*, *30*, 771–784.
- Janowsky, J. S., Shimamura, A. P., & Squire, L. R. (1989). Source memory impairment in patients with frontal lobe lesions. *Neuropsychologia*, *27*, 1043–1056.
- Johnson, M. K., Hashtroudi, S., & Lindsay, D. S. (1993). Source monitoring. *Psychological Bulletin*, *114*, 3–28.
- Johnson, M. K., & Raye, C. L. (1998). False memories and confabulation. *Trends in Cognitive Sciences*, *2*, 137–145.
- Johnson, M. K., & Raye, C. L. (2000). Cognitive and brain mechanisms of false memories and beliefs. In D. L. Schacter & E. Scarry (Eds.), *Memory, brain, and belief* (pp. 35–86). Cambridge, MA: Harvard University Press.
- Kirschbaum, C., Pirke, K.-M., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test'—A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, *28*, 76–81.
- Kirschbaum, C., Wolf, O. T., May, M., Wippich, W., & Hellhammer, D. H. (1996). Stress- and treatment-induced elevations of cortisol levels associated with impaired declarative memory in healthy adults. *Life Sciences*, *58*, 1475–1483.
- Kirschbaum, C., Wüst, S., & Hellhammer, D. (1992). Consistent sex differences in cortisol responses to psychological stress. *Psychosomatic Medicine*, *54*, 648–657.
- Kudielka, B. M., Buske-Kirschbaum, A., Hellhammer, D. H., & Kirschbaum, C. (2004). HPA axis responses to laboratory psychosocial stress in healthy adults, younger adults, and children: Impact of age and gender. *Psychoneuroendocrinology*, *29*, 83–98.
- Kudielka, B. M., & Kirschbaum, C. (2005). Sex differences in HPA axis responses to stress: A review. *Biological Psychology*, *69*, 113–132.
- Kudielka, B. M., Schommer, N. C., Hellhammer, D. H., & Kirschbaum, C. (2004). Acute HPA axis responses, heart rate, and mood changes to psychosocial stress (TSST) in humans at different times of day. *Psychoneuroendocrinology*, *29*, 983–992.
- Kuhlmann, S., Piel, M., & Wolf, O. T. (2005). Impaired memory retrieval after psychosocial stress in healthy young men. *Journal of Neuroscience*, *25*, 2977–2982.
- Lupien, S. J., Gillin, C. J., & Hauger, R. L. (1999). Working memory is more sensitive than declarative memory to the acute effects of corticosteroids: A dose-response study in humans. *Behavioral Neuroscience*, *113*, 420–430.
- Lupien, S. J., & Lepage, M. (2001). Stress, memory, and the hippocampus: You can't live with it, can't live without it. *Behavioral Brain Research*, *127*, 137–158.
- McGaugh, J. L., & Roozendaal, B. (2002). Role of adrenal stress hormones in forming lasting memories in the brain. *Current Opinion in Neurobiology*, *12*, 205–210.
- Merckelbach, H., Horselenberg, R., & Muris, P. (2001). The Creative Experience Questionnaire (CEQ): A brief self-report measure of fantasy proneness. *Personality and Individual Differences*, *31*, 987–995.
- Mitchell, K. J., Johnson, M. K., Raye, C. L., & Greene, E. J. (2004). Prefrontal cortex activity associated with source monitoring in a working memory task. *Journal of Cognitive Neuroscience*, *16*, 921–934.
- Moscovitch, M. (1992). Memory and working-with-memory: A component process model based on modules and central systems. *Journal of Cognitive Neuroscience*, *4*, 257–267.
- Moscovitch, M. (1994). Cognitive resources and dual-task interference effects at retrieval in normal people: The role of the frontal lobes and medial temporal cortex. *Neuropsychology*, *8*, 524–534.

- Moscovitch, M., & Winocur, G. (1992). Frontal lobes and memory. In L. R. Squire (Ed.), *The encyclopedia of learning and memory: A volume in neuropsychology* (pp. 182–187). New York: Macmillan.
- Moscovitch, M., & Winocur, G. (2002). The frontal cortex and working with memory. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function* (pp. 188–209). New York: Oxford University Press.
- Nolde, S. F., Johnson, M. K., & D'Esposito, M. (1998). Left prefrontal activation during episodic remembering: An event-related fMRI study. *NeuroReport*, *9*, 3509–3514.
- Oitzl, M. S., & de Kloet, E. (1992). Selective corticosteroid antagonists modulate specific aspects of spatial orientation learning. *Behavioral Neuroscience*, *106*, 62–71.
- Parks, T. E. (1997). False memories of having said the unsaid: Some new demonstrations. *Applied Cognitive Psychology*, *11*, 485–494.
- Radley, J. J., Sisti, H. M., Hao, J., Rocher, A. B., McCall, T., Hof, P. R., et al. (2004). Chronic behavioral stress induces apical dendritic reorganization in pyramidal neurons of the medial prefrontal cortex. *Neuroscience*, *125*, 1–6.
- Ranganath, C., Johnson, M. K., & D'Esposito, M. (2000). Left anterior prefrontal activation increases with demands to recall specific perceptual information. *Journal of Neuroscience*, *20*, 1–5.
- Raye, C. L., Johnson, M. K., Mitchell, K. J., Nolde, S. F., & D'Esposito, M. (2000). fMRI investigations of left and right PFC contributions to episodic remembering. *Psychobiology*, *28*, 197–206.
- Reul, J. M., & de Kloet, E. R. (1985). Two receptor systems for corticosterone in rat brain: Microdistribution and differential occupation. *Endocrinology*, *117*, 2505–2511.
- Roozendaal, B. (2000). Glucocorticoids and the regulation of memory consolidation. *Psychoneuroendocrinology*, *25*, 213–238.
- Roozendaal, B., Hahn, E. L., Nathan, S. V., de Quervain, D. J., & McGaugh, J. L. (2004). Glucocorticoid effects on memory retrieval require concurrent noradrenergic activity in the hippocampus and basolateral amygdala. *Journal of Neuroscience*, *24*, 8161–8169.
- Ross, D. F., Ceci, S. J., Dunning, D., & Togli, M. P. (1994). Unconscious transference and mistaken identity: When a witness misidentifies a familiar but innocent person. *Journal of Applied Psychology*, *79*, 918–930.
- Rugg, M. D., Fletcher, P. C., Chua, P. M.-L., & Dolan, R. J. (1999). The role of the prefrontal cortex in recognition memory and memory for source: An fMRI study. *NeuroImage*, *10*, 520–529.
- Sanchez, M. M., Young, L. J., Plotsky, P. M., & Insel, T. R. (2000). Distribution of corticosteroid receptors in the rhesus brain: Relative absence of glucocorticoid receptors in the hippocampal formation. *Journal of Neuroscience*, *20*, 4657–4668.
- Schacter, D. L., Harbluk, J. L., & McLachlan, D. (1984). Retrieval without recollection. *Journal of Verbal Learning and Verbal Behavior*, *23*, 593–611.
- Schommer, N. C., Hellhammer, D. H., & Kirschbaum, C. (2003). Dissociation between reactivity of the hypothalamus-pituitary-adrenal axis and the sympathetic-adrenal-medullary system to repeated psychosocial stress. *Psychosomatic Medicine*, *65*, 450–460.
- Shafraan, R., Thordarson, D. S., & Rachman, S. (1996). Thought-action fusion in obsessive compulsive disorder. *Journal of Anxiety Disorders*, *10*, 379–391.
- Slotnick, S. D., Moo, L. R., Segal, J. B., & Hart, J., Jr. (2003). Distinct prefrontal cortex activity associated with item memory and source memory for visual shapes. *Cognitive Brain Research*, *17*, 75–82.
- Smeets, T., Jelicic, M., & Merckelbach, H. (2006a). Stress-induced cortisol responses, sex differences, and false recollections in a DRM paradigm. *Biological Psychology*, *72*, 164–172.
- Smeets, T., Jelicic, M., & Merckelbach, H. (2006b). The effect of acute stress on memory depends on word valence. *International Journal of Psychophysiology*, *62*, 30–37.
- Snodgrass, J. G., & Corwin, J. (1988). Pragmatics of measuring recognition memory: Applications to dementia and amnesia. *Journal of Experimental Psychology: General*, *117*, 34–50.
- Stefanis, N. C., Hanssen, M., Smirnis, N. K., Avramopoulos, D. A., Evdokimidis, I. K., Stefanis, C. N., et al. (2002). Evidence that three dimensions of psychosis have a distribution in the general population. *Psychological Medicine*, *32*, 347–358.
- Van Cauter, E., & Refetoff, S. (1985). Evidence for two subtypes of Cushing's disease based on the analyses of episodic cortisol secretion. *New England Journal of Medicine*, *312*, 1343–1349.
- Wang, J., Rao, H., Wetmore, G. S., Furlan, P. M., Korczykowski, M., Dinges, D. F., et al. (2005). Perfusion functional MRI reveals cerebral blood flow pattern under psychological stress. *Proceedings of the National Academy of Sciences, USA*, *102*, 17804–17809.
- Wolf, O. T. (2003). HPA axis and memory. *Best Practice & Research Clinical Endocrinology and Metabolism*, *17*, 287–299.

Received March 17, 2006

Revision received August 14, 2006

Accepted August 24, 2006 ■