

The impact of stress on neutral and emotional aspects of episodic memory

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The present experiment demonstrates that exposure to a significant psychological stressor (administered *before* watching a slide show) *preserves* or even *enhances* memory for emotional aspects of an event, and simultaneously *disrupts* memory for non-emotional aspects of the same event. Stress exposure also disrupted memory for information that was visually and thematically central to the event depicted in the slide show. Memory for peripheral information, on the other hand, was unaffected by stress. These results are consistent with theories invoking differential effects of stress on brain systems responsible for encoding and retrieving emotional memories (the amygdala) and non-emotional memories (e.g., the hippocampal formation), and inconsistent with the view that memories formed under high levels of stress are qualitatively the same as those formed under ordinary emotional circumstances. These data, which are also consistent with results obtained in a number of studies using animals and humans, have implications for the traumatic memory debate and theories regarding human memory.

For at least the last two decades, the impact of stress on memory has been understood as largely disruptive. Both animal (e.g., Diamond, Park, Heman, & Rose, 1999; Sapolsky, Krey, & McEwen, 1986) and human (e.g., Lupien et al., 1997; Lupien, Lecours, Lussier, Schwartz, Nair, & Meaney, 1994; Newcomer, Craft, Hershey, Askins, & Bardgett, 1994; Newcomer et al., 1999) studies have demonstrated that exposure to high levels of stress can impair memory. Stress is typically induced by exposing subjects either to stress hormones directly (e.g., cortisol) or to a psychosocial manipulation (e.g., public speaking). Both techniques can be detrimental to the proper functioning of the hippocampus, thereby resulting in pronounced memory deficits (de Quervain et al., 2003; de Quervain, Roozendaal, Nitsch, McGaugh, & Hock, 2000; Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Newcomer

et al., 1994, 1999; Payne, Nadel, Allen, Thomas, & Jacobs, 2002).

In spite of this general pattern of memory disruption, other studies show that high levels of stress can enhance memory for emotionally arousing experiences (e.g., Cahill, Gorski, & Le, 2003; Cahill & McGaugh, 1998). These findings suggest that the nature of the memory task is important, and indeed, a close comparison of studies finding memory impairment with those finding memory enhancement reveals that the former typically use neutral memory materials (de Quervain et al., 2000, 2003; Kirschbaum et al., 1996; Newcomer et al., 1994, 1999; Payne et al., 2002; Wolkowitz, Reus, & Weingartner, 1990; Wolkowitz et al., 1993), while the latter use materials that are emotionally arousing (Buchanan & Lovallo, 2001; Cahill et al., 2003).

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Thus, while high stress levels often impair memory for neutral memory materials, they typically enhance memory for emotional memory materials (Buchanan & Lovallo, 2001; Cahill et al., 2003; Gold & van Buskirk, 1978; McGaugh, 2000). Consider, for example, the varied effects of administering a one-time, mild dose of cortisol (i.e., 20/25 mg) to human subjects. De Quervain et al. (2000, 2003) administered a 25 mg dose of cortisol to subjects learning word lists composed of unrelated, emotionally neutral nouns. Those who had received the cortisone retrieved significantly *fewer* of these neutral words than participants who received a placebo. Buchanan and Lovallo (2001), on the other hand, showed that a 20 mg dose of cortisol enhanced memory for emotional relative to neutral pictures. Subjects given cortisol recalled significantly *more* emotional pictures than did subjects in a placebo group, while both groups recalled a similar number of neutral pictures.

Similar effects have been achieved by exposing human subjects to psychological stress. For example, public speaking has been shown to disrupt memory for neutral word lists, both by reducing the amount of material recalled (e.g., Kirschbaum et al., 1996), and by producing gist-based false memory errors (Payne et al., 2002). These results likely stem from elevations in stress hormones that accompany the performance of stressful tasks—e.g., the Trier Social Stress Test (TSST), see Kirschbaum, Pirke, and Hellhammer, 1993; Kirschbaum et al., 1996. To our knowledge, psychosocial stress has not yet been used to examine emotional memory; thus, its impact on emotional tasks remains to be determined. However, to the extent that stressors effectively raise levels of stress-related neuromodulators, it follows that they should facilitate emotional memory.

Taken together, these results suggest that high stress levels should disrupt neutral memory and facilitate emotional memory, even if these two memory types are components of the same experience. However, the studies leading to this conclusion are separate and cross-sectional, with many methodological differences between them. Some researchers administered stress hormones directly, while others used more naturalistic social stressors to induce stress. Moreover, many of the studies tested memory for simple word lists or pictures, while others tested memory for events (Buchanan & Lovallo, 2001; Cahill et al., 2003; Cahill, Prins, Weber, & McGaugh, 1994). It thus remains to be seen which pattern of results would

be obtained in a single episodic memory experiment, after the administration of a specific psychosocial stressor. The primary goal of this experiment is to answer this question, predicting that stress exposure (induced via public speaking) will disrupt memory for the neutral features, and enhance memory for the emotional features of a single event.

These predictions are well grounded in the neurobiology of stress and memory. Both the stress-based enhancement of emotional memory and the stress-based disruption of neutral memory have been attributed specifically to the impact of stress hormones and neuromodulators on brain regions that are important for memory (Buchanan & Lovallo, 2001; de Quervain et al., 2000, 2003; Kim & Diamond, 2002; Kirschbaum et al., 1996; Lupien & McEwen, 1997; Payne et al., 2002; Roozendaal, 2003). Both norepinephrine and cortisol facilitate the functioning of the amygdala—a structure known to modulate emotional memory (Cahill & McGaugh, 1998; McGaugh, 2000; Roozendaal, 2003). High levels of cortisol, on the other hand, disrupt the functioning of the hippocampus and prefrontal cortex, structures integral to memory for neutral materials (Kim & Diamond, 2002).

Drawing specifically on the impact of stress hormones on brain function is nonetheless not the only, or best, way to understand the effects of stress on memory. A separate research tradition has demonstrated that physiological arousal (as opposed to stress *per se*) also produces an enhancement of emotional memory. Arousal can be dissociated from stress physiologically, with stress activating the hypothalamic-pituitary-adrenal (HPA) axis and ultimately resulting in the release of cortisol (Bondi & Picardi, 1999; Lovallo & Thomas, 2000). Arousal, on the other hand, while sharing some physiological functions with stress, such as increased heart rate, does not inevitably activate the HPA axis or produce cortisol release. Could it be that physiological arousal alone can account for the stress and memory findings discussed above? This is a possibility we must consider. Furthermore, the neutral versus emotional memory distinction may not be the best way of delineating the effects of stress on memory. Studies of arousal and memory show that memory for “central” aspects of a stimulus or event are enhanced, often at the expense of more “peripheral” aspects. It could be the case that stress produces the same, albeit undetected

result, with emotional/neutral information being confounded with central/peripheral information.

The central versus peripheral breakdown stems from laboratory studies of emotional memory that have aimed to test two intertwined hypotheses. The first concerns subjects' memory for the gist or "central" information contained within an event. The bulk of the evidence on this claim is reasonably clear at this point: in general, emotional arousal seems to enhance memory for an event's gist or centre (cf. Reisberg & Heuer, 2004). The second derives from data showing that arousal causes a narrowing of attention, so that an aroused subject attends less to information at the "periphery" of an event and more to information at the centre of an event. This is known as the Easterbrook hypothesis (Easterbrook, 1959; Loftus, 1982; Mandler, 1975). This attention to central details results in the "weapon focus" phenomenon, where eyewitnesses to crimes can often vividly remember the gun or knife used to perpetrate a crime, but not the features of the assailant (see Loftus, 1979). There is considerable evidence demonstrating enhanced memory for central information under conditions of emotional arousal. However, the data are mixed as to whether memory for peripheral information is substantially decreased under the same conditions (see Reisberg & Heuer, 2004). It seems to depend, at least in part, on how the terms "central" and "peripheral" are defined.

There are two major ways of defining what is central and what is peripheral to a particular stimulus or event (see Reisberg & Heuer, 2004, for a review). The first is based on visual centrality, for example the focal point of a threatening picture, such as a gun or knife depicted in a picture of an attack. Peripheral details, on the other hand, would be any other aspect of the picture, such as background objects or even the physical appearance of the perpetrator. Christianson and Loftus (1991) studied memory for central versus peripheral features of an emotional event involving a bicycle accident. They defined these categories strictly in visual terms; peripheral details were those that were truly in the background (e.g., a car located in the distant background or extreme "periphery" of the slide). The second definition is more conceptual, and based on the theme or plot of an episode. Heuer and Reisberg (1990) defined as central any aspects of the story that were directly relevant to the plot, or in any way important for how the story unfolded; they defined as peripheral any information that could

be changed without changing the story. The data suggest that emotional arousal enhances central information, regardless of whether it is conceptualised as visual or thematic. Conversely, impaired memory for peripheral information (memory narrowing) seems to emerge only when testing memory for the visual periphery (see Reisberg & Heuer, 2004).

It is possible that an overlap exists between the emotional content of an event and what is central to that event. In the Heuer and Reisberg (1990) study, for example, much of the central, thematic, and plot-relevant information was emotional in nature, as the story is about a boy visiting his father at a hospital and watching him conduct gruesome surgeries. Indeed, Christianson (1992) points out that central information represents the source of the emotional arousal, including the most relevant information for extracting the emotional significance of an event. Likewise, there may well be overlap between what is neutral and what is peripheral, as many of the details that can be changed without altering the gist of the story are neutral in nature.

The current experiment aims to test the effect of stress on memory for emotional versus neutral information in an episodic memory experiment, where the episode unfolds in space and time, and emotional and neutral features are part of the same experience. We will also test the effect of stress on memory for central versus peripheral information, thus enabling us to determine which dichotomy better accounts for the data—the emotional/neutral distinction or the central/peripheral distinction. A larger, conceptual question follows from this distinction; namely, which account will better describe the data, the stress account or the arousal account? As discussed, the stress account posits that neutral information should be disrupted, and emotional information enhanced, by the actions of stress-related neuro-modulators at different brain sites (i.e., hippocampal regions and prefrontal cortex, amygdala). To the extent that stress *per se* (and not just physiological arousal) is an important manipulation, neutral, but not emotional, memory should suffer in the current experiment.

The arousal account, while similarly positing that emotional information should be preserved relative to neutral information due to high levels of emotional arousal, also posits that stress should lead to an enhancement of central information, and possibly (according to the Easterbrook hypothesis) a disruption of peripheral information. If

peripheral memory suffers and central memory improves under stress, we will have to consider at least two different possibilities: (1) that stress is an intense form of arousal having the same, uneven impact on central versus peripheral memory, or (2) that overlap necessarily exists between central and emotional information, and between peripheral and neutral information.

The present experiment examines the impact of highly emotional, psychosocial stress on memory for different aspects of a single event. As stated above, we hypothesised that stress would disrupt memory overall, but we also suspected that analysing “memory” part by part (e.g., emotional vs neutral information, and central vs peripheral information) would reveal a more complex interplay between stress and memory. Specifically, we expected stress to disrupt neutral but not emotional memory, which would be preserved, if not enhanced, by the same stressor. We also expected to see a significant overlap between emotional information and central information, and between neutral information and peripheral information, thus resulting in a similar pattern of preserved central information and disrupted peripheral information.

The current study used a variation on the “doctor/mechanic” memory task (Heuer & Reisberg, 1990, described above) to test these predictions. To date, most studies of stress and memory have investigated memory for simple word lists and paragraphs (e.g., de Quervain et al., 2000; Newcomer et al., 1994, 1999). Yet stressful experiences in the real world typically comprise some combination of emotional features (e.g., a car accident, injuries that resulted from the accident) and neutral features (e.g., the conversation one was having when the accident occurred), unfolding in space and time. It is of theoretical and practical interest to know if stress affects memory for the various aspects of an event in the same way. We thus modified the procedure to allow memory for neutral and emotional information to be evaluated separately.

METHOD

Participants

Participants were 117 undergraduate students recruited from lower-division psychology classes who received course credit for their participation. Participants were randomly assigned to a 2 (stress

group: stress vs control) \times 2 (delay condition: immediate memory test vs 1-week delay) between-subjects factorial design. There were 61 participants in the stress condition and 56 in the no-stress control condition. We tried to ensure relatively equivalent numbers of males and females in each of the four groups. Thus, of the 33 participants in the stress/immediate memory condition, 14 were males and 19 were females. Of the 28 participants in the stress/1-week delay condition, 12 were males and 16 were females. In the control condition, 17 males and 11 females were tested in the immediate memory condition, and 10 males and 18 females were tested in the delay condition.

Stimulus materials

All participants viewed a narrated slide show containing emotional and neutral information. The slides were taken from Heuer and Reisberg (1990), who kindly provided copies of their materials. In contrast to Heuer and Reisberg’s procedure where subjects viewed two different slide shows, one neutral and one emotionally arousing, participants in the present study viewed only the emotional version.

The slide show contained 12 detailed slides, the middle 3 of which were emotionally arousing. The material in the remaining 9 (4 shown before the emotional slides and 5 after) was neutral. Cahill and McGaugh (1995) have shown that the middle emotional slides (along with their corresponding narrative) account almost entirely for participants’ enhanced memory for the arousing story relative to the neutral story. Heuer and Reisberg (1990) showed the same pattern in their own data, where recognition memory was most pronounced for the emotionally arousing middle slides (see Heuer & Reisberg, 1990, Figure 2). The emotionally neutral slides in this version of the story are typically no better remembered than the corresponding slides in the neutral version of the story. Using a single set of slides thus allowed us to compare emotional and neutral slides as a within-subjects variable, eliminating the interpretive difficulties posed by using different stimuli in the emotional and neutral slide shows.

Each of the 12 slides was accompanied by a recorded narration. The slide show depicted a mother taking her young son to visit his father (a surgeon) at work. Midway through the slide show, participants learn that a terrible car accident has

occurred, and the father must fight to save the victims. The son watches his father work, and at this point participants see the emotionally arousing slides (the car accident, a team of surgeons operating on the open chest cavity of a victim, and a victim's reattached severed legs).

Procedure

Participants in the experimental group were exposed to the Trier Social Stress Test (TSST) (Kirschbaum et al., 1993), a procedure that reliably elicits moderate psychological stress in laboratory settings (Kirschbaum et al., 1996). These subjects were required to deliver a speech in front of a one-way mirror. They were told that three trained investigators, located behind the mirror, would evaluate the speech. Ten minutes were given for speech preparation, notes were abruptly taken from participants at the end of this time, and they were asked to give the speech extemporaneously. Participants also believed that the speech was video and audio recorded and that verbal and nonverbal aspects of their performance were being analysed. All speeches were made under intense lighting, in the presence of two tripod-mounted 1000-watt halogen stage lights. Following the 5-minute speech, participants performed a moderately difficult subtraction task aloud, also for 5 minutes. Control participants sat and listened to relaxing music for a corresponding amount of time (c 20 minutes).

All participants were tested individually. After completing the TSST (or the relaxation period in the case of controls), participants sat at a computer and two Ag-AgCl electrodes were attached to their fingers. They were told (deceptively) that physiological measurements, including heart rate and skin conductance, were being taken to assess sensitivity to various types of auditory and visual stimulation, that they would be watching a slide show, that they might find some slides pleasant, some unpleasant, and some neutral, and to carefully attend to each slide for the duration of its presentation. After indicating that they understood the instructions, participants viewed the slide show while listening to an accompanying narration through headphones. Each slide was presented for 6 seconds via computer; the narration began immediately after presentation of each slide. Subjects were not informed that their memory would be tested for the materials later on.

Subjective state anxiety reports, using the Spielberger state-trait anxiety scale (Spielberger, 1983), were collected at (1) the beginning of the experiment, before the experimental manipulation, (2) immediately after the manipulation (participants were asked, "How do you feel *now*?"), (3) after the manipulation, immediately after the second form (participants were asked, "How did you feel *during* the speech?"), and (4) after viewing the slides, at the end of the experiment. Although saliva samples were not analysed for cortisol content in this study, a sister study (Jackson, Payne, Nadel, & Jacobs, 2004) demonstrates that, in this laboratory, the TSST reliably elevates cortisol (see also Kirschbaum et al., 1996).

Depending on experimental condition, participants were administered the memory test immediately after viewing the slides or asked to return 1 week later for "additional physiological testing". Participants in the delay condition were told they were participating in an experiment of sensory sensitivity. As part of this deception, they took home journals to record episodes of sensitivity.

The memory assessment consisted of both a recall and a recognition memory test. After Heuer and Reisberg (1990) and Cahill et al. (1994), participants were first asked to recall all they could about the narrated story they had previously seen, both about the general story line and any details they recalled. They were then reminded that they had seen a total of 12 slides, and were asked to remember each slide in as much detail as possible. Recall data were taped for later transcription and analysis.¹ Participants were credited with remembering a slide if they described visual information that could only be known from having viewed that particular slide (Cahill et al., 1994). Two judges, blind to experimental condition, scored these data. Each judge also scored the data for false recall errors. Memories were considered "false" only if participants recalled information that was clearly not part of the slide show or narration (e.g., "an ambulance came and took the boy away"). Percent agreement between the judges was greater than 90% in both cases. The few disagreements were discussed until a consensus was reached and were ultimately decided by the second judge (JP).

¹With the exception of four subjects, who were excluded from the analysis due to problems with the recording equipment.

Following free recall, participants were given a 4AFC recognition questionnaire (137 questions), where questions pertained to visual elements of the slide show as well as the narration. The questions and answer choices were read aloud to all participants in the same order. For each question, a confidence judgement was made, indicating whether the participant was (1) not at all confident of the answer to (5) extremely confident in the answer. These confidence ratings were used to construct a “false memory” variable. False memories were considered confident answers (i.e., a rating of 5) that were nonetheless incorrect.

An emotional memory score was obtained for each subject by computing the average number of correct responses on the emotionally arousing slides 5, 6, and 7. A neutral memory score was obtained by computing the average correct responses to all other slides. Finally, two variables were created to measure “central information” and “peripheral information”. The first of these defined “central information” as information that was central to the plot of the story. Three scorers assessed each question on the recognition questionnaire, and determined whether it “could be removed without changing the gist or plot of the story”. If so, it received a score of “peripheral”. If not, the question received a score of “central”. For example, a question asking whether mother and son (as opposed, for example, to a mother and daughter, or a father and son) were depicted is central to this story. So, for that matter, is the fact that a car accident occurred and that father performs surgery on the victims. Changing these items would change the plot of the story. Examples of peripheral facts, on the other hand, are that mother was wearing a pink collar, that the boy had blond hair, that he was carrying a soccer ball, and so on. One could change these details without changing the gist or theme of the story.

The questions were next scored for visual centrality. In this case, the three scorers were instructed to determine whether each question corresponded to a feature that was “visually central or visually peripheral relative to what the viewer sees when looking at each slide”. By this second scoring system, facts that were learned via the narration (as opposed to looking at the slides) would necessarily be peripheral because they were not visible on the slides. Scorers placed a “VC” (for visually central) next to central questions and a “VP” (for visually peripheral) next to peripheral questions. There was approximately 95% concurrence between scorers for both thematically

and visually defined central/peripheral variables. Again, disagreements were discussed and settled by the second judge (JP).

RESULTS

Exposure to the TSST produced a robust increase in mean subjective stress ratings (see Table 1). Although stress and control participants gave equivalent stress ratings at the beginning of the experiment, before stressor administration, those in the experimental condition rated themselves as significantly more stressed, both during ($M = 59$) and immediately after ($M = 51$) the speech, than those in the control condition at the same two times ($M_s = 26$ and 27 , respectively). *T*-tests comparing groups at each time point confirmed that these were significant differences, $t_s(114) = 16.6$ (during) and 12.8 (after), p_s both $< .0001$. The difference between the groups disappeared on the fourth subjective anxiety measure, given shortly before participants left the lab, indicating that subjective stress had returned to baseline by the end of the experiment.

Recall memory overall

We first analysed free recall memory with a $2 \times 2 \times 2$ between-subjects ANOVA comparing group (stress, control), sex (male, female), and delay (immediate memory, 1-week delay). Exposure to the stressor significantly disrupted recall memory, but largely in men, producing a significant group \times sex interaction effect, $F(1, 105) = 4.54$, $p < .05$. Follow-up analyses revealed a trend towards recall memory suffering more in males, $t(50) = 1.9$, $p = .069$, than in females, $t(59) = -1.1$ (ns), for

TABLE 1
Subjective stress ratings

	1st	2nd	3rd	4th
Control group	35 (8.7)	26 (6.5)	27 (5.2)	34 (9.8)
Stress group	37 (8.3)	59 (13.1)	51 (13.3)	36 (9.9)
<i>p</i> value	NS	$< .0001$	$< .0001$	NS

Mean (SD) subjective stress ratings at the four time points during the experiment:

1st = Stress assessment, before manipulation.

2nd = Stress assessment, after manipulation.

3rd = Stress assessment, “How did you feel during . . .?”

4th = Stress assessment, prior to leaving lab.

whom recall memory was statistically equivalent in both stress and control conditions. We next analysed false recall using a similar 2 (group) \times 2 (sex) ANOVA. Analyses of false recall errors revealed further memory impairments in stressed subjects, with subjects in the stress group recalling more false information than those in the control group, $F(1, 105) = 4.11, p < .05$. This false memory effect was unrelated to sex.

Emotional vs neutral recall memory

We next examined the effect of stress on the type of memory presented in the different phases of the slide show. Recall that recognition memory scores were computed for emotional and neutral information, yielding a two-level within-subjects “memory type” variable. We thus used a 2 \times 2 \times 2 \times 2 mixed ANOVA, this time comparing stress group (stress, control), sex (male, female), delay (immediate memory test, 1-week delay), and memory type (emotional, neutral) as a within-subjects variable (see Table 2). This analysis revealed that emotional information ($M = .81$) was better recalled than neutral information overall ($M = .66$), $F(1, 105) = 45.3, p < .0001$, an effect that did not interact with delay condition. We also found a significant group \times memory type interaction, $F(1, 105) = 4.4, p < .05$, indicating that neutral memory, but not emotional memory, was impaired by the stress manipulation (see Figure 1). Unlike neutral information, emotional information was preserved under stress. Participants in the stress ($M = .807$) and control ($M = .809$) groups remembered an equivalent amount of emotional information.

This result was accompanied by a marginally significant group \times memory type \times sex interaction, $F(1, 109) = 3.6, p = .059$, and a significant group \times memory type \times sex \times delay interaction, $F(1, 109) = 4.6, p < .05$ (see Table 2). Splitting the data by “delay” showed that the group \times sex interaction reached significance only in the 1-week delay condition, $F(1, 50) = 9.3, p < .01$. Stressed females carried this effect, apparently by showing facilitated memory for the emotional slides recalled after a 1-week delay. Follow-up t -tests confirmed that stressed females recalled significantly more emotional information than both stressed males ($p < .01$), and control females ($p < .01$) (see Figure 2). Importantly, then, stress did enhance emotional recall, although only in females.

Recognition memory overall

We next analysed overall recognition memory scores with a 2 \times 2 \times 2 between-subjects ANOVA, comparing stress group (stress, control), sex (male, female), and delay (immediate memory test, 1-week delay). Exposure to the stressor significantly disrupted recognition memory overall, as revealed by a main effect of stress condition, $F(1, 109) = 6.03, p < .02$. Stressed subjects remembered significantly less information from the slide show overall than did non-stressed control subjects. There was also a significant effect of delay. Participants tested immediately after viewing the slides remembered more information than those tested after a 1-week delay, $F(1, 109) = 91.69, p < .0001$. However, the delay variable did not interact with the other variables of interest.

TABLE 2
Recognition memory scores

Stress				Control			
Males		Females		Males		Females	
No delay	Delay	No delay	Delay	No delay	Delay	No delay	Delay
<i>Neutral memory</i>							
.49 (.08)	.42 (.06)	.55 (.07)	.43 (.07)	.60 (.08)	.45 (.07)	.60 (.06)	.47 (.07)
<i>Emotional memory</i>							
.64 (.09)	.52 (.1)	.68 (.06)	.52 (.1)	.68 (.06)	.54 (.1)	.68 (.08)	.52 (.09)

Mean (*SD*) recognition memory scores for neutral and emotional memory, broken down by stress, sex, and delay conditions.

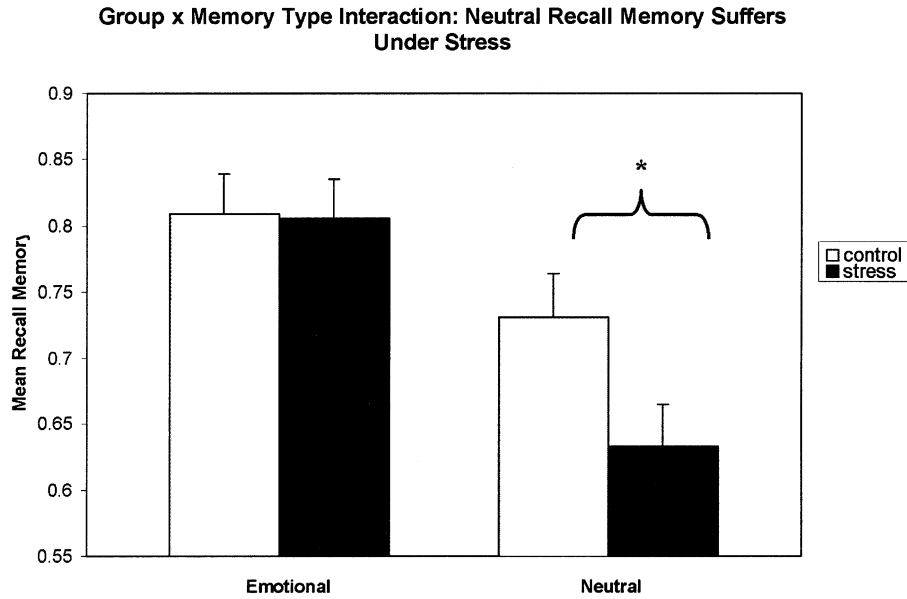


Figure 1. Recall memory for neutral and emotional slides: Proportion of slides correctly recalled by stress and control groups. * $p < .05$.

Finally, there was no main effect of sex ($p = .37$), nor did sex interact with any of the other variables.

Emotional vs neutral recognition memory

We next examined the effect of stress on the type of memory presented in the different phases of the

slide show. We thus used a $2 \times 2 \times 2 \times 2$ mixed ANOVA, this time comparing group (stress, control), sex (male, female), delay (immediate memory test, 1-week delay), and memory type (emotional, neutral) as a within-subjects variable. In addition to the main effects of stress and delay noted above, a main effect of memory type emerged, $F(1, 109) = 117.7, p < .0001$, which was qualified by a significant stress by memory type

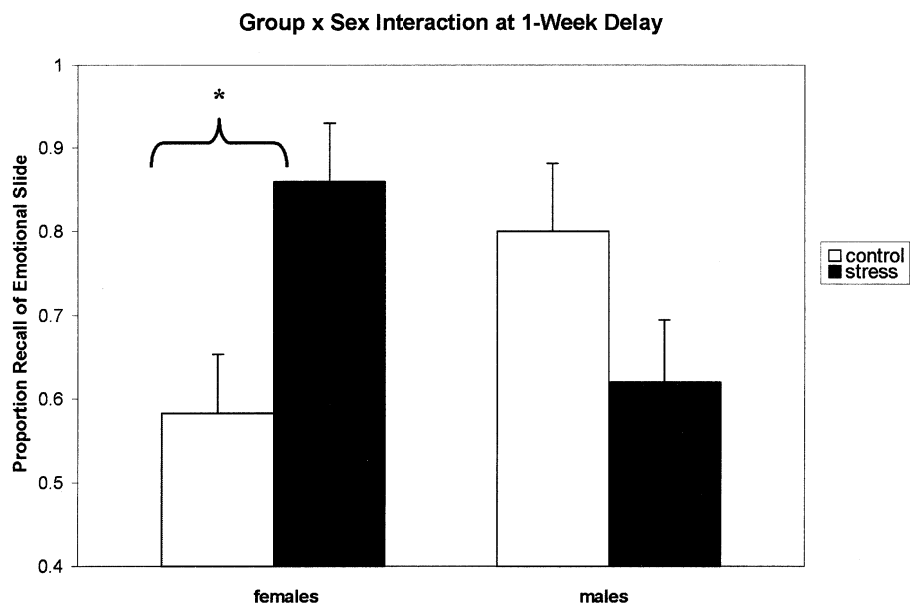


Figure 2. Recall of emotional slides in male and female participants at a 1-week delay.

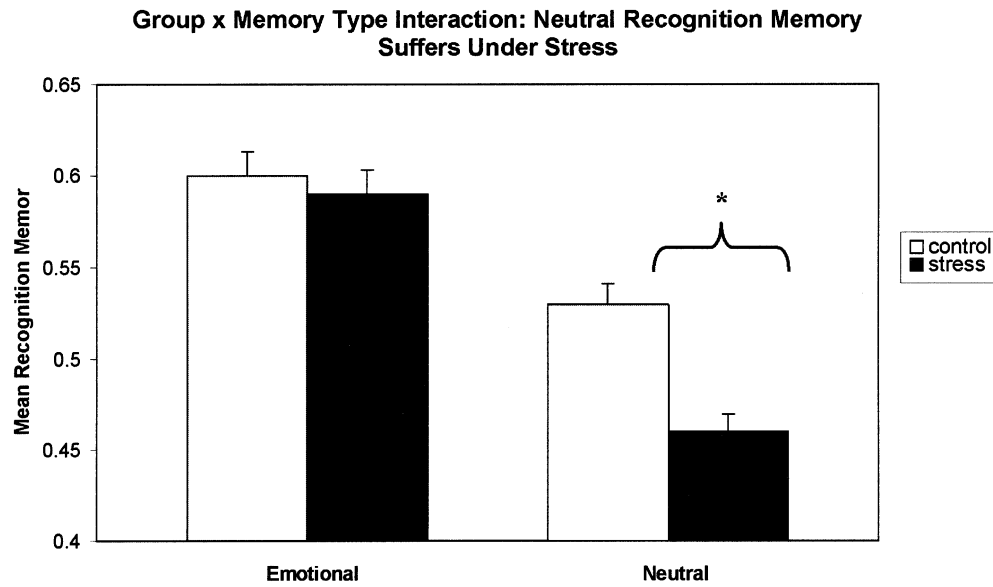


Figure 3. Recognition memory for neutral and emotional slides: Proportion of questions correctly answered by stress and control groups. * $p < .05$.

interaction, $F(1, 109) = 4.78$, $p < .05$. As can be seen in Figure 3, and consistent with our predictions, only neutral information suffered under stress. Memory for emotional information was preserved relative to neutral information.

Strange, Hurlmann, and Dolan (2003) report that memory for neutral information suffers when it precedes emotional information. We thus recreated the memory type variable, this time yielding a three-level variable where the neutral slides were further broken into “pre” and “post” emotional information (neutral pre, emotional, neutral post). “Pre” refers to the neutral content (slides 1–4) preceding the emotional slides, and “post” refers to the neutral material (slides 8–12) following the emotional slides (4–7). We con-

ducted another 2 (stress group; between subjects) \times 2 (sex; between subjects) \times 2 (delay; between subjects) \times 2 (memory type with three levels, pre, emotional, and post; within subjects) mixed ANOVA to assess the impact of stress, sex, and delay on the three-level memory type variable.

Regardless of its position relative to the emotional material, memory for neutral information was marginally impaired under stress $F(1, 109) = 3.35$, $p = .07$ (see Figure 4). To clarify this result, we analysed the three story phases (neutral pre, emotional, neutral post) in separate ANOVAs (after Cahill & McGaugh, 1995), which revealed that stressed participants remembered significantly less neutral information in both the pre, $F(1, 109) = 13.59$, $p < .01$, and post, $F(1, 109) =$

TABLE 3
Recall memory scores

<i>Stress</i>				<i>Control</i>			
<i>Males</i>		<i>Females</i>		<i>Males</i>		<i>Females</i>	
<i>No delay</i>	<i>Delay</i>	<i>No delay</i>	<i>Delay</i>	<i>No delay</i>	<i>Delay</i>	<i>No delay</i>	<i>Delay</i>
<i>Neutral memory</i>							
.67 (.06)	.52 (.07)	.78 (.05)	.54 (.06)	.77 (.06)	.68 (.07)	.88 (.07)	.61 (.06)
<i>Emotional memory</i>							
.88 (.06)	.61 (.06)	.88 (.05)	.85 (.06)	.92 (.06)	.80 (.07)	.93 (.07)	.58 (.06)

Mean (*SE*) recall memory scores for neutral and emotional memory, broken down by stress, sex, and delay conditions.

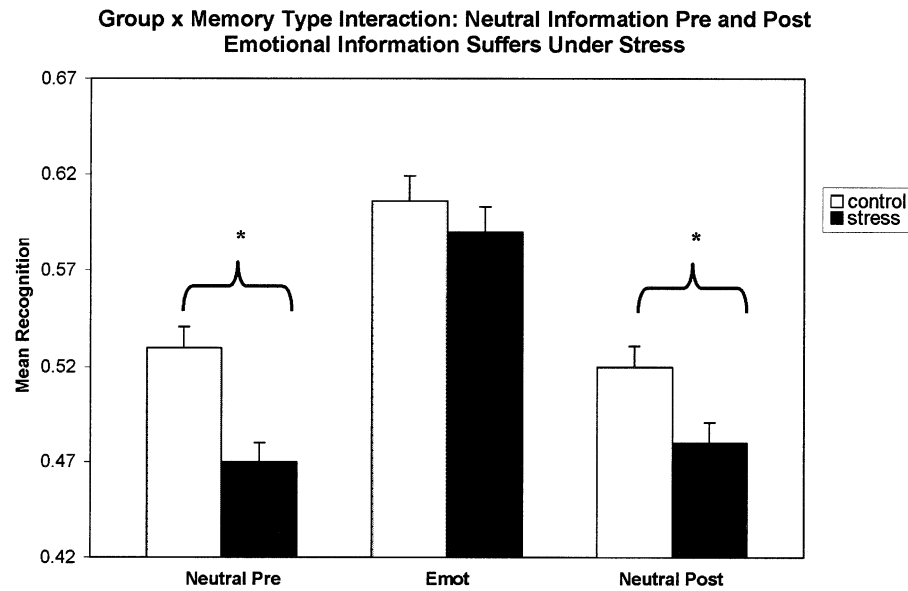


Figure 4. Recognition memory for neutral-pre, emotional, and neutral-post slides: Proportion of questions correctly answered by stress and control groups. * $p < .05$.

5.74, $p < .02$, phases, but remembered an equivalent amount of information in the emotional phase, $F(1, 109) = 0.71$, $p = .40$ ns.

Finally, despite the changes to the Reisberg and Heuer (1990) procedure, we obtained an “emotional memory enhancement effect”. Emotional information was better remembered (in this case recognised) than neutral information, $F(1, 114) = 75.2$, $p < .0001$. Interestingly, this main effect of memory type did not interact with delay, suggesting that the emotional memory enhancement effect was present in both the immediate and delay conditions (see Figure 5). Follow-up ANOVAs confirmed this to be the case in both the immediate memory condition, $F(1, 59) = 55.1$, $p < .0001$, and the 1-week delay condition, $F(1, 54) = 24.6$, $p < .0001$.

However, remembering emotional information well does not mean that it is remembered accurately. Looking at false memories, which we defined as confident false recognition responses (i.e., false recognition responses that had a confidence rating of 5 or “extremely certain”), a group \times sex interaction emerged, $F(1, 109) = 6.69$, $p < .01$. Stressed females falsely recognised significantly more emotional information than control females ($p < .05$). Correlational analyses revealed that these false memories were positively related to the *details* of the emotional slides (e.g., the colour of a door, the number of cars in a particular slide) ($r = .32$, $p < .01$), but not the gist

(e.g., general questions about the story line) ($r = .13$, ns). This finding did not apply to males, nor did it reach statistical significance for neutral information.

Central vs peripheral recognition memory

We next examined the effect of stress on memory for central versus peripheral information, as assessed by the recognition questionnaire. A 2 (group; stress vs control) \times 2 (sex; male vs female) \times 2 (delay; immediate memory test vs 1-week delay) \times 2 (memory type; central vs peripheral, within subjects) mixed ANOVA revealed a main effect of group, qualified by a two-way interaction between group and memory type; $F(1, 109) = 3.84$, $p < .05$. Stress significantly *disrupted* memory for central, thematic information ($M = .71$ versus $M = .77$ in the control condition). Memory for thematically peripheral information, on the other hand, though reduced relative to thematically central information, was not significantly impaired by the stress manipulation ($M = .36$ in the stress group, $M = .385$ in the control group). A similar pattern emerged for visually defined central and peripheral information (e.g., $M = .487$ for visually central information in the stress condition, $M = .534$ in the control condition), although in this case the relevant interaction

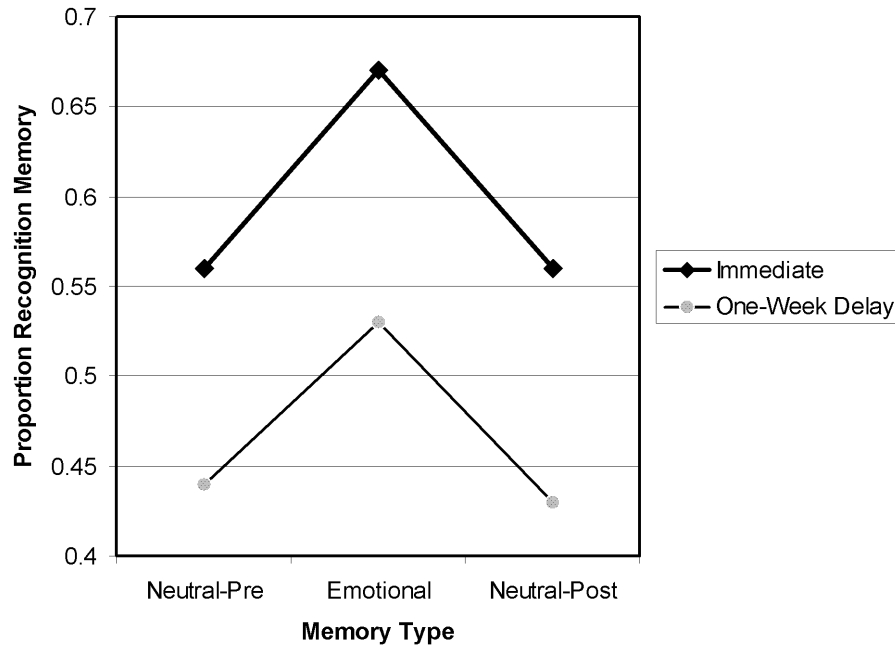


Figure 5. Recognition memory for different slides: Proportion of questions correctly answered by immediate and 1-week delay groups.

was not significant. Also similarly, visually peripheral information was unaffected by stress ($M = .52$, stress group; $M = .55$, control group).

Stress did not simply disrupt memory for the comparatively trivial details of the story; the detrimental effect of stress applied even to the most central elements—the who, what, when, and where—of the story. For example, individual chi-square analyses confirmed that stressed subjects were significantly less able than control subjects to remember critical facts, such as the fact that the son watched the father perform the surgery (91% of stressed subjects remembered this fact as compared to 100% of control subjects, ($\chi^2 = 3.15$; $df = 1$; $p = .06$); that the father was pleased that his son watched (77% compared to 90%), ($\chi^2 = 4.85$; $df = 1$, $p < .05$); that the father was smiling after the surgery (42% compared to 66%), ($\chi^2 = 6.59$; $df = 1$, $p < .01$); and that the mother left the hospital alone (78% compared to 93%), ($\chi^2 = 4.72$; $df = 1$, $p < .03$).

DISCUSSION

In the past decade, cognitive and neuroscientific studies have shown that both stress and arousal augment emotional memory (e.g., Cahill & McGaugh, 1998; Reisberg & Heuer, 2004). We

replicated this effect here, demonstrating that memory for emotional material is superior to memory for neutral material both immediately and after a 1-week delay. In typical experiments using this design, memory is tested only after a delay, on the assumption that emotion specifically augments consolidation processes (Cahill & McGaugh, 1998). Only recently have encoding effects been considered (e.g., Cahill, 2003a; Quevedo et al., 2003; Strange & Dolan, 2004; Strange et al., 2003). Quevedo et al. (2003) found enhanced memory for emotional slides after a 1-week delay (LTM), but not after a 1-hour delay (STM), suggesting that emotional arousal effects are not related to STM. Given this, the authors concluded that the amygdala does not modulate short-term emotional memory mechanisms. However, Strange et al. (2003) found enhanced memory for emotional relative to neutral words after a very brief delay, and they were able to relate this enhancement to the adrenergic and amygdala mechanisms that are known to participate in long-term emotional memory enhancement (Cahill, 2003a; Strange et al., 2003). Our immediate memory findings thus add to the burgeoning evidence that emotional arousal can act on both short-term and long-term memory processes (see also Hamann, 2001). Although our use of a pre-learning stressor may have made

amygdala involvement more likely in the emotional memory enhancement effect seen in the immediate memory condition, further studies, perhaps that administer β -adrenergic antagonists as in Strange et al. (2003) and in Strange and Dolan (2004), are needed to investigate this assumption directly.

Our data also demonstrate that stress has differential effects on memory for the various features or components of what, in its entirety, is an emotional episode. The episode depicted in our slides is complex, containing features presented in different modalities (visual and auditory), and containing both emotional and neutral information—much as would be the case during a stressful episode in the real world. As predicted, stress disrupted memory for the neutral, but not the emotional, features of the episode.

Contrary to our predictions, however, stress failed to disrupt memory for peripheral information relative to central information. In fact, the opposite was true: While stressed subjects had worse memory than control subjects overall (i.e., worse memory for both central and peripheral information), they had particular difficulty remembering the central information, performing far more poorly than controls. This pattern applied to central information regardless of whether it was defined conceptually or visually. So while memory for emotional information was preserved, and in some cases even enhanced, under stress, memory for central information suffered under stress. These rather different patterns may indicate that there is *not* substantial overlap between what is emotional about an event and what is central to an event, at least in this particular paradigm.

Interestingly, Heuer and Reisberg (1990) found that arousal *enhanced* both central and peripheral features of the emotional slide show. Here, stress *disrupted* both central and peripheral features of the slide show. Taken together, these results suggest that the arousal induced by our stress manipulation cannot account for the current findings. Rather, stress may have a unique impact on memory. The stressed subjects in our experiment had difficulty remembering even the most central facts about the story—critical facts such as who, what, when, and where—not just the minor details. The fact that experimental and control subjects did not differ significantly in terms of their memory for peripheral details (e.g., the fact that the little boy was carrying a soccer ball or was wearing tennis shoes) suggests that these details

are difficult to remember regardless of whether or not one is under stress. The most detrimental impact of stress was on the more important, central information that was critical to the plot of the story. Thus, while general emotional arousal facilitates memory for these central story elements, stress appears to disrupt them.

The Easterbrook hypothesis was also not supported in the current experiment, in that peripheral information was not selectively impaired relative to central information. This is a particularly surprising result given that stress induces physiological and emotional arousal. Consistent with the current study, Heuer and Reisberg (1990) found that arousal failed to impair memory for peripheral information. While this might have been due to their definition of central information (defined thematically rather than visually), it might also have been because the magnitude of arousal in their study (generated simply by viewing the emotional slides) was not sufficient to produce the attentional narrowing effect found in other experiments. As stress undoubtedly produces physiological arousal, however, this latter explanation now seems unlikely.

It appears, then, that the stress account provides a better explanation of our data than the arousal account. As the stress account predicts, we found preserved (or enhanced) emotional memory and impaired neutral memory after the stress manipulation. This is not to say that arousal makes no contribution to the preservation of emotional information seen in the stress group—it almost certainly does. However, it does suggest that arousal alone cannot account for the full pattern of findings, particularly the pronounced disruption of neutral information. Nor can it account for the disruption of central information we observed. Given these findings, we suggest that arousal and stress have somewhat distinct effects on memory, with stress adding to and changing the impact of arousal.

It is worth noting that most studies of arousal and memory have used forms of arousal that derive from the studied memory materials (i.e., the arousal is generated by viewing the emotional materials), and it is these studies that have led to the established pattern of enhanced memory for central relative to peripheral information. However, several studies have aroused subjects via extrinsic means, such as the administration of stimulants or vigorous exercise (e.g., Christianson & Mjorndal, 1985). These studies typically fail to reveal an impact on memory, suggesting that

arousal must be intrinsic to the to-be-remembered materials to have an effect on memory. The current data show a strong effect of the stress manipulation, which is independent of the to-be-remembered materials. This may be another hint that a critical difference exists between the impact of “arousal” and “stress” on memory, even though the stress response includes an increase in physiological arousal. On top of this increase, however, is the activation of the HPA axis, which occurs specifically during the stress response. The stress response triggers the release of cortisol and norepinephrine, both of which affect memory in specific ways. High levels of cortisol have repeatedly been shown to disrupt memory for neutral materials, and we believe that cortisol elevations are most likely behind the neutral memory impairments found here.² The Trier Social Stress Test (TSST) is associated with average cortisol elevations of around 12–13 nmol/L—a level that has produced memory disruption both in our lab (Jackson, Payne, Nadel, & Jacobs, *in press*; Payne et al., 2002) and in other labs (e.g., Kirschbaum et al., 1993, 1996). Thus, while we did not take cortisol samples in the current experiment, we have good reason to believe that cortisol was elevated by the TSST.

Previous studies on the neural mechanisms of stress and memory (e.g., Jackson et al., 2003; Kirschbaum, Wust, & Hellhammer, 1992; Wolf, Shommer, Hellhammer, McEwen, & Kirschbaum, 2001), and emotion and memory (e.g., Cahill & van Stegeren, 2003; Canli et al., 2002; Shors, 1998) have pointed to substantial sex differences. In line with these findings, we found three differing response patterns between the sexes.

The first was a pattern of enhanced emotional memory for women, but not men, after a 1-week delay. Examining both sexes together, emotional memory appeared to be “preserved”, but not enhanced, by stress. Examining them separately, however, revealed that women showed the emotional memory enhancement effect, consistent with Buchanan and Lovallo’s (2001) finding of enhanced emotional memory following a low dose of the stress hormone cortisol, and other studies showing an augmenting effect of stress on memory for emotional materials (e.g., Cahill et al., 2003;

see McGaugh, 2000). The question, however, is why this effect emerged only in women in the current experiment. Although we did not measure cortisol in the current study, it is well documented that females give greater subjective stress ratings to social stressors than males (Kirschbaum et al., 1992; Stroud, Salove, & Epel, 2002). That our speech task resulted in stronger memory effects in females than in males might reflect this difference in perceived stress.

The second sex difference emerged in the proportions of false recognition errors that men and women made under stress. Stressed females falsely recognised more emotional information than stressed males, particularly for peripheral detail (as opposed to central information or gist). Cahill and van Stegeren (2003) have shown that, in women, the effect of arousal on emotional memory is modulated by the left amygdala, whereas in men it is modulated by the right amygdala. As the left hemisphere is also associated with the processing of detail information (as opposed to gist information, which is the domain of the right hemisphere), it may be that stress specifically impaired the processing of emotional detail information in women (see Cahill, 2003b). However, this is probably not the best explanation, as stressed females, in spite of their false memories for emotional detail, also showed generally superior overall emotional memory (see above). This superiority may have led to a false confidence in their memory, even for difficult to remember, highly specific details.

The third sex difference emerged in the overall recall data, and suggested that men’s recall memory was more impaired by stress than females’ recall memory. While this result did not emerge in the recognition memory data, or in any of the analyses where memory type (whether emotional/neutral or central/peripheral) was assessed, it is nonetheless consistent with other findings in the literature. For instance, Wolf et al. (2001) demonstrated a sex difference in the relationship between cortisol and short-term memory. In spite of an overall negative correlation between cortisol and memory in a mixed-sex sample, additional analyses showed that the effect was specific to men. Thus, higher cortisol values were associated with poorer memory performance in men, but not in women. Cahill et al. (2003), however, in a study of post-learning stress administration (cold pressor stress), failed to find this sex difference, although this may have been due to a small number of male subjects in the

²Given cortisol’s facilitatory effect on the amygdala (McGaugh, 2000), cortisol elevations probably also help to explain why emotional information was preserved in our experiment.

sample. Further studies are clearly needed to pinpoint the impact of stress and cortisol on memory in male versus female human participants.

The fact that stress has a variable impact on different types of memory seems relevant to the traumatic memory debate. Some researchers have suggested that intensely stressful or traumatic memories are always remembered well. For example, Shobe and Kihlstrom (1997, p. 74) argued that "... the preponderance of laboratory evidence indicates that memory is more likely to be enhanced than impaired by high levels of emotion and stress". However, we have shown, in numerous experiments including the one presented here, that stress leads to complex patterns of memory disruption and enhancement (Jackson et al., in press; Nadel & Jacobs, 1998; Payne et al., 2002; Payne, Nadel, Britton, & Jacobs, 2004).

As mentioned, stress-related memory impairment is found in studies of memory dependent on the hippocampus (de Quervain et al., 2000, 2003), a brain structure known to be essential for normal episodic memory function. The hippocampus is densely packed with receptors for the stress hormone cortisol, which at high levels disrupts hippocampal function and impairs episodic memory (Kim & Diamond, 2002; Lupien & McEwen, 1997; Payne et al., 2004). Other studies show that emotional arousal, even strongly negative emotional arousal, enhances forms of memory modulated by the amygdala (Cahill & McGaugh, 1998).

The amygdala plays an important role in memory for emotional events, an effect that is also governed by norepinephrine and the beta-adrenergic system (McGaugh, 2000). Until recently, some thought that such arousal had a net facilitating effect on *all* types and *all* aspects of memory. Livingston's (1967) "Now print!" concept, for example, suggested that the brain "prints" all information immediately preceding and following an arousing event. Similarly, "flashbulb memories"—vivid, long-lasting memories for emotionally arousing and significant events—were once thought to strongly and faithfully represent past emotional experience. It appears that neither of these original conceptions is completely accurate. Emotional arousal may only facilitate memory for those emotional aspects of events that engender arousal at the time of encoding (Cahill & Alkire, 2003), and flashbulb memories, while maintaining perceived vividness and detail, are often highly inconsistent across time (Talarico & Rubin, 2003).

How victims remember trauma has been termed the "most divisive issue facing psychology today" (McNally, 2003, p. 1). While both cognitive and neuroscientific studies have made it clear that emotional arousal and stress augment memory (e.g., Cahill & McGaugh, 1998; Reisberg & Heuer, 2004), it seems premature to conclude that traumatic memories are always well remembered, or that "... people remember horrific experiences all too well" (McNally, 2003, p. 2).

We believe that stress can simultaneously impoverish memory for neutral aspects, and preserve emotional aspects, of an emotional or traumatic episode. What, then, might a traumatic memory look like when recalled? It would certainly not be a perfect rendition of the original emotional experience. Rather, it might be vague for important emotionally neutral details—like time of occurrence, spatial context, or perhaps details about a perpetrator (e.g., what he or she was wearing)—while being relatively sharp for focal emotional information (e.g., the pain of being hurt, the fact that one was assaulted). Even so, one must keep in mind that some "memories" of "well-remembered" emotional events are inaccurate. A hint of this exists in our own data, albeit only in women, and only in memory for minor emotional details, not emotional gist (see also Payne et al., 2004). It also exists in flashbulb memories, which lack accuracy and consistency even in those memories that people believe they remember in sharp detail (e.g., September 11th 2001, Talarico & Rubin, 2003).

In summary, the present study highlights the fact that stress has complex effects on memory. Memory for an emotional experience may be preserved or even strengthened, while memory for the neutral elements of that emotional experience is diminished. This pattern of results can be understood if one assumes that these two aspects of memory may be modulated by the amygdala and hippocampus respectively, and that a sufficiently high concentration of stress hormones might potentiate the former but inhibit the latter. Clinicians often report that memory for trauma is characterised not only by intrusive memories, but also by gaps in memory and general memory impairment (see Payne et al., 2004). Our data suggest that a brain-based account reflecting the role of stress hormones and their actions in different memory-relevant systems in the brain, might explain why this is so.

REFERENCES

- Bondi, M., & Picardi, A. (1999). Psychological stress and neuroendocrine function in humans: The last two decades of research. *Psychotherapy and Psychosomatics*, *68*, 114–150.
- Buchanan, T. W., & Lovallo, W. R. (2001). Enhanced memory for emotional material following stress-level cortisol treatment in humans. *Psychoneuroendocrinology*, *26*, 307–317.
- Cahill, L. (2003a). Similar neural mechanisms for emotion-induced memory impairment and enhancement. *Proceedings of the National Academy of Sciences USA*, *100*, 13123–13124.
- Cahill, L. (2003b). Sex- and hemisphere-related influences on the neurobiology of emotionally influenced memory. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *27*, 1235–1241.
- Cahill, L., & Alkire, M. (2003). Epinephrine enhancement of human memory consolidation: Interaction with arousal at encoding. *Neurobiology of Learning and Memory*, *79*, 194–198.
- Cahill, L., Gorski, L., & Le, K. (2003). Enhanced human memory consolidation with post-learning stress: Interaction with the degree of arousal at encoding. *Learning & Memory*, *10*, 270–274.
- Cahill, L., & McGaugh, J. L. (1995). A novel demonstration of enhanced memory associated with emotional arousal. *Consciousness and Cognition*, *4*, 410–421.
- Cahill, L., & McGaugh, J. L. (1998). Mechanisms of emotional arousal and lasting declarative memory. *Trends in Neuroscience*, *21*, 294–299.
- Cahill, L., Prins, B., Weber, M., & McGaugh, J. L. (1994). Beta adrenergic activation and memory for emotional events. *Nature*, *371*, 702–704.
- Cahill, L., & van Stegeren, A. (2003). Sex-related impairment of memory for emotional events with β -adrenergic blockade. *Neurobiology of Learning and Memory*, *79*, 81–88.
- Canli, T., Desmond, J. E., Zhao, Z., & Gabrieli, J. D. (2002). Sex differences in the neural basis of emotional memories. *Proceedings of the National Academy of Sciences USA*, *99*(16), 10789–10794.
- Christianson, S.-A. (1992). Emotional stress and eyewitness memory: A critical review. *Psychological Bulletin*, *112*, 284–309.
- Christianson, S.-A., & Loftus, E. (1991). Remembering emotional events: The fate of detailed information. *Cognition & Emotion*, *5*, 81–108.
- Christianson, S.-A., & Mjorndal, T. (1985). Adrenalin, emotional arousal, and memory. *Scandinavian Journal of Psychology*, *26*, 237–248.
- de Quervain, D. J., Henke, K., Aerni, A., Treyer, V., McGaugh, J. L., Berthold, T. et al. (2003). Glucocorticoid-induced impairment of declarative memory retrieval is associated with reduced blood flow in the medial temporal lobe. *European Journal of Neuroscience*, *17*(6), 1296–1302.
- de Quervain, D. J., Roozendaal, B., Nitsch, R. M., McGaugh, J. L., & Hock, C. (2000). Acute cortisone administration impairs retrieval of long-term declarative memory in humans. *Nature Neuroscience*, *3*, 313–314.
- Diamond, D. M., Park, C. R., Heman, K. L., & Rose, G. M. (1999). Exposing rats to a predator impairs spatial working memory in the radial arm maze. *Hippocampus*, *9*, 542–552.
- Easterbrook, J. A. (1959). The effect of emotion on cue utilization and the organization of behavior. *Psychological Review*, *66*, 183–201.
- Gold, P. E., & van Buskirk, R. (1978). Posttraining brain norepinephrine concentrations: Correlation with retention performance of avoidance training and with peripheral epinephrine modulation of memory processing. *Behavioral Biology*, *23*, 509–520.
- Hamann, S. (2001). Cognitive and neural mechanisms of emotional memory. *Trends in Cognitive Sciences*, *5*, 394–400.
- Heuer, F., & Reisberg, D. (1990). Vivid memories of emotional events: The accuracy of remembered minutiae. *Memory & Cognition*, *18*, 496–506.
- Jackson, E. D., Payne, J. D., Nadel, L., & Jacobs, W. J. (in press). Stress differentially modulates fear conditioning in healthy men and women. *Biological Psychiatry*.
- Kim, J. J., & Diamond, D. M. (2002). The stressed hippocampus, synaptic plasticity and lost memories. *Nature Reviews Neuroscience*, *3*, 453–462.
- 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., Wiplich, 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., Wust, S., & Hellhammer, D. (1992). Consistent sex differences in cortisol responses to psychological stress. *Psychosomatic Medicine*, *54*, 648–657.
- Livingston, R. B. (1967). Reinforcement. In G. Quarton, T. Melnechuk, & F. Schmitt (Eds.), *The neurosciences: A study program* (pp. 514–576). New York: Rockefeller Press.
- Loftus, E. (1979). *Eyewitness testimony*. Cambridge, MA: Harvard University Press.
- Loftus, E. (1982). Remembering recent experiences. In L. Cermak (Ed.), *Human memory and amnesia* (pp. 239–255). Hillsdale, NJ: Lawrence Erlbaum Associates Inc.
- Lovallo, W. R., & Thomas, T. L. (2000). Stress hormones in psychophysiological research: Emotional, behavioral and cognitive implications. In J. T. Cacioppo, L. G. Tassinary & G. G. Berntson (Eds.), *Handbook of psychophysiology* (2nd ed.). Cambridge: Cambridge University Press.
- Lupien, S. J., Gaudreau, S., Tchiteya, B. M., Maheu, F., Sharma, S., Nair, N. P. V. et al. (1997). Stress-induced declarative memory impairment in healthy elderly subjects: Relationship to cortisol reactivity. *Journal of Clinical Endocrinology and Metabolism*, *82*, 2070–2075.
- Lupien, S. J., Lecours, A. R., Lussier, I., Schwartz, G., Nair, N. P. V., & Meaney, M. J. (1994). Basal cortisol levels and cognitive deficits in human aging. *Journal of Neuroscience*, *14*, 2893–2903.

- Lupien, S. J., & McEwen, B. S. (1997). The acute effects of corticosteroids on cognition: Integration of animal and human model studies. *Brain Research Reviews*, *24*, 1–27.
- Mandler, G. (1975). *Mind and emotion*. New York: Wiley.
- McGaugh, J. L. (2000). Memory: A century of consolidation. *Science*, *287*, 248–251.
- McNally, R. J. (2003). *Remembering trauma*. Cambridge, MA: Harvard University Press.
- Nadel, L., & Jacobs, W. J. (1998). Traumatic memory is special. *Current Directions in Psychological Science*, *7*, 154–157.
- Newcomer, J. W., Craft, S., Hershey, T., Askins, K., & Bardgett, M. E. (1994). Glucocorticoid-induced impairment in declarative memory performance in adult humans. *Journal of Neuroscience*, *14*, 2047–2053.
- Newcomer, J. W., Selke, G., Melson, A. K., Hershey, T., Craft, S., Richards, K., et al. (1999). Decreased memory performance in healthy humans induced by stress-level cortisol treatment. *Archives of General Psychiatry*, *56*, 527–533.
- Payne, J. D., Nadel, L., Allen, J. J. B., Thomas, K. G. F., & Jacobs, W. J. (2002). The effects of experimentally induced stress on false recognition. *Memory*, *10*, 1–6.
- Payne, J. D., Nadel, L., Britton, W. B., & Jacobs, W. J. (2004). The biopsychology of trauma and memory. In D. Reisberg & P. Hertel (Eds.), *Memory and emotion* (pp. 76–128). London: Oxford University Press.
- Quevedo, J., Sant'Anna, M. K., Madruga, M., Lovato, I., de-Paris, F., Kapczinski, F. et al. (2003). Differential effects of emotional arousal in short- and long-term memory in healthy adults. *Neurobiology of Learning and Memory*, *79*, 132–135.
- Reisberg, D., & Heuer, F. (2004). Memory for emotional events. In D. Reisberg & P. Hertel (Eds.), *Memory and emotion* (pp. 3–41). London: Oxford University Press.
- Roozendaal, B. (2003). Systems mediating acute glucocorticoid effects on memory consolidation and retrieval. *Progress in Neuro-Psychopharmacology & Biological Psychiatry*, *27*, 1213–1223.
- Sapolsky, R. M., Krey, L. C., & McEwen, B. S. (1986). The neuroendocrinology of stress and aging: The glucocorticoid cascade hypothesis. *Endocrinology Reviews*, *7*, 284–301.
- Shobe, K. K., & Kihlstrom, J. F. (1997). Is traumatic memory special? *Current Directions in Psychological Science*, *6*(3), 70–74.
- Shors, T. J. (1998). Stress and sex effects on associative learning: For better or for worse. *The Neuroscientist*, *4*, 353–364.
- Spielberger, C. D. (1983). *Manual for the state-trait anxiety inventory* (2nd ed.). [In collaboration with R. L. Gorusch, R. Lushene, P. R. Vagg, & G. A. Jacobs.] Palo Alto, CA: Consulting Psychologists Press.
- Strange, B. A., & Dolan, R. J. (2004). β -Adrenergic modulation of emotional memory-evoked human amygdala and hippocampal responses. *Proceedings of the National Academy of Sciences USA*, *101*(31), 11454–11458.
- Strange, B. A., Hurlmann, R., & Dolan, R. J. (2003). An emotion-induced retrograde amnesia in humans is amygdala- and beta-adrenergic-dependent. *Proceedings of the National Academy of Sciences USA*, *100*(23), 13626–13631.
- Stroud, L. R., Salove, P., & Epel, E. S. (2002). Sex differences in stress responses: Social rejection versus achievement stress. *Biological Psychiatry*, *52*, 318–327.
- Talarico, J. M., & Rubin, D. C. (2003). Confidence, not consistency, characterizes flashbulb memories. *Psychological Science*, *14*, 455–461.
- Wolf, O. T., Shommer, N. C., Hellhammer, D. H., McEwen, B. S., & Kirschbaum, C. (2001). The relationship between stress induced cortisol levels and memory differs between men and women. *Psychoneuroendocrinology*, *26*, 711–720.
- Wolkowitz, O. M., Reus, V. I., & Weingartner, H. (1990). Cognitive effects of corticosteroids. *American Journal of Psychiatry*, *147*, 1297–1303.
- Wolkowitz, O. M., Weingartner, H., Rubinwo, D. R., Jimerson, D., Lking, M., Berretini, W. et al. (1993). Steroid modulation of human memory: Biochemical correlates. *Biological Psychiatry*, *33*, 744–746.