Effects of Acute Psychosocial Stress on Visuospatial Information Processing in Healthy Males

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Abstract

Previous research has shown that stress can cause severe impairments in a number of different cognitive systems. There is scant research, however, investigating the effects of stress on visuospatial information processing. The few studies that have examined stress and visuospatial memory performance report inconsistent results, and there are no studies that examine stress and planning/organisation of visuospatial information directly. This study aimed to investigate the effects of acute psychosocial stress on visuospatial information processing in healthy males. Participants (22 undergraduate volunteers) were administered the copy trial of the Rey-Osterrieth Complex Figure Test (ROCF); those randomly assigned to the Stress group (n = 11) were then exposed to a psychosocial stressor, while the rest were exposed to an equivalent control condition. All were then administered the short- and long-delay recall trials of the ROCF. Physiological and self-report measures of stress indicated that the induction manipulation was effective. At long-delay recall, but not short-delay recall, Stress-group participants took longer to complete their drawing, and adopted a more piecemeal approach to it, than did those in the Non-Stress group. This observation of impaired visuospatial information processing at ROCF long-delay recall is consistent with (a) the peak in cortisol levels that occurs 20-40 minutes after the onset of the stressor, and (b) the idea that those raised cortisol levels lead to changes in prefrontal cortex functioning.

Keywords: stress; visuospatial; memory; planning; organisation; Rey-Osterrieth Complex Figure Test; cortisol.

Introduction

Stress is, for most humans, a part of everyday life. Relatively mild stressors, such as those experienced when preparing for and taking an exam, are more commonplace than relatively severe stressors, such as those associated with the experience of childhood abuse and neglect. Regardless of the source or severity of stress, however, the perception and experience of a stressor appears to have effects on particular aspects of cognition, including memory: Sometimes one goes blank when looking at an exam paper, even though test preparation has been good, and sometimes a vivid, intensely stressful childhood trauma is remembered for a lifetime (Wolf, 2009).

Physiological Response to a Stressful Event

An event is considered stressful when it threatens a major adaptive goal, and therefore threatens the physical or psychological well-being of an individual (Kemeny, 2003). All stressful events, psychological and physical, are capable of producing the same physiological responses.

The body mobilizes two main physiological systems (the autonomic nervous system (ANS) and the hypothalamic-pituitary-adrenal (HPA) axis) in response to stress. The ANS response is rapid and leads to increased activation of certain physiological mechanisms, such as heart rate and skin conductance response. The HPA axis response is slower and longer-lasting, and of greater relevance to brain-behaviour relations.

The following chain of events describes the typical HPA-axis physiological response to the perception of a physical or psychological stressor. When a stressor is perceived, the hypothalamus is stimulated and releases corticotrophin-releasing hormone (CRH). CRH stimulates the anterior pituitary to release adrenocorticotropic hormone (ACTH) into the bloodstream. ACTH stimulates the cortex of the adrenal gland to release glucocorticoids (GC; cortisol in humans). Cortisol is secreted to prepare the body to deal with the stress, and thus it has a major effect on the body and the brain.

Hence, in response to a stressor, there is an increase in the concentration of cortisol in the urine, saliva, and blood. As noted above, this process has more long-term effects than those associated with ANS activation. Cortisol levels peak 20-40 minutes after the onset of the stressor, and typically return to baseline 40-60 minutes after the termination of the stressor (Alderson & Novack, 2002; Kemeny, 2003; Wolf, 2003).

Cortisol is a hydrophilic hormone, and therefore it can easily cross the blood-brain barrier. Once in the brain, it affects numerous structures, including the hippocampus and prefrontal cortex (PFC), by binding to specific receptors that are highly concentrated in these regions (Alderson & Novack, 2002; Putman & Roelofs, 2011; Wolf, 2003). Both the hippocampus and the PFC play important roles in cognitive processing. The hippocampus is vital to learning and memory, while the PFC is similarly crucial to executive functions such as planning and organisation (Miller & Cohen, 2001; Squire, 1992; Wolf, 2003).

Effects of Stressors on Visuospatial Information Processing

Visuospatial information processing refers to the perception, encoding, construction, retrieval, organisation, planning and output of figural, pictorial, spatial, and other non-verbal bits of information. As such, the term subsumes a number of the traditionally-defined cognitive domains, including memory and executive functioning (Lezak, Howieson, & Loring, 2004).

Most previous studies examining the effects of stress on visuospatial information processing focus on measures of visuospatial memory (e.g., the ability to remember a complex figure, or the ability to remember a route through a maze).Visuospatial memory performance involves, essentially, the ability to learn and remember the location of objects, and relationships between objects, in environmental space (Astur, Taylor, Mamelak, Philpott, & Sutherland, 2002; Bechara, Damasio, Damasio, & Anderson, 1994; Johnson & Adamo-Villani, 2010; Kessels, Kappelle, Haan, & Postma, 2002; Pierrot-Deseilligny, Mu¨ri, Rivaud-Pechoux, Gaymard, & Ploner, 2002). This form of cognition may be classed, broadly, as a form of declarative memory (i.e., it refers to the ability of the individual to be explicitly aware of and to be able to make report on adaptively relevant non-verbal or verbal stimuli; Ullman, 2004).

Forming new declarative memories involves three processes: encoding, consolidation and retrieval. Each of these memory phases could be differently affected by the high levels of cortisol associated with the perception and experience of stress. Empirical studies suggest that high levels of cortisol have particularly adverse effects on the retrieval phase (Smeets, 2011; Wolf, 2003), but facilitate consolidation (Buchanan & Lovallo, 2001; Preuβ & Wolf, 2009) and have no effect on encoding (Wolf, 2009).

Most of the studies referred to in the previous paragraph were conducted using verbal declarative memory tasks, and the studies that have focused on non-verbal or visuospatial

declarative memory have not used consistent experimental procedures (e.g., they have not all examined the effects of stress on retrieval of visuospatial information). Hence, there is no consensus in the literature regarding the effects that stress has on visuospatial declarative memory. Some have found that stress enhances visuospatial memory performance (Luethi, Meier, & Sandi, 2009), whereas others have found that stress impairs that performance (Morgan, Doran, Steffian, Hazlett, & Southwick, 2006; Traverniers, Ruysseveldt, Smeets, & Grumbkow, 2010), or has no effect on it (Hoffman & al'Absi, 2004).

Taverniers et al. (2010) investigated the effects of high-intensity stress in a naturalistic environment on visuospatial memory performance. They simulated a prisoner of war (POW) experience with Belgian Special Forces candidates to induce high-intensity stress in one group. A control group completed a number of non-stressful filler tasks. Immediately after the experimental manipulation, participants were administered the Rey-Osterrieth Complex Figure Test (ROCF; Osterrieth, 1944; Rey, 1941). The results showed significant performance impairment on the ROCF. More specifically, stressed participants were unable to recall specific details of the figure, although they could recall the gestalt as accurately as the control group. These results are consistent with those of Morgan et al. (2006), who found that intense stress, induced using a simulated POW situation at a survival school for special operators, impaired ROCF recall.

The two studies reviewed above induced high-intensity stress in extreme situations; hence, participants in their stress groups experienced large increases in cortisol levels. Cortisol affects central nervous system functioning: that is, too much *and* too little cortisol inhibits declarative memory functioning, while a moderate level of cortisol can facilitate declarative memory. Hence, the relationship between cortisol levels and declarative memory function takes on an inverted U-shaped function (Alderson & Novack, 2002; Luethi et al., 2009; Traverniers et al., 2010; Wolf, 2003). Therefore, it is possible that the results of those two studies cannot be generalized to normal humans in everyday situations (although they do provide evidence for the effects of stress on memory at the extreme right side of the inverted-U curve). Therefore, there is a need for studies that investigate the effect of an everyday psychosocial stressor on visuospatial declarative memory (thus, perhaps, providing evidence for what happens at other parts of the inverted-U curve).

Acute psychosocial stressors, as used in the studies reviewed below, are a part of everyday life; hence, they are not as intense as those stressors used in the studies of Taverniers et al. (2010) and Morgan et al. (2006). Luethi et al. (2009) found that recognition

and recall for a learned route on a map was enhanced by a public-speaking psychosocial stressor.

With regard to the effects of stress on other aspects of visuospatial information processing, there are no studies that directly examine the effects of stress on planning and organisation of nonverbal stimuli. A small group of studies has, however, investigated the impairing effects of stress on other frontal lobe functions, such as decision-making and working memory (Human, Henry, & Thomas, 2010; Luethi et al., 2009; Oei, Everaerd, Elzinga, van Well, & Bermond, 2006; Schoofs, Preuβ, & Wolf, 2008; Schoofs, Wolf, & Smeets, 2009; van den Bos, Harteveld, & Stoop, 2009). Morgan et al. (2006), in their study of ROCF performance following experience of an intense stressor, found that participants took a narrow and detailed approach to their copy of the figure. This approach suggests participants adopted a piecemeal strategy to copying, rather than the complete gestalt approach taken by control participants. This pattern of data suggests, in turn, that stress might have an impairing effect on planning and organisation of visuospatial information.

Rationale and Specific Hypotheses

Previous literature on the effects of stress on cognition has mainly focused on verbal declarative memory (see, e.g., Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Luethi et al., 2009; Stawski, Sliwinski, & Smyth, 2009). Limited research exists investigating the effect of acute psychological stressors on visuospatial information processing in healthy humans. Furthermore, the current literature on the effects of stress on visuospatial memory is equivocal, and the studies that have found an impairing effect of stress on visuospatial memory have used high-intensity stress inductions. These studies did not, therefore, investigate the effects of everyday stressors on visuospatial memory. My research will examine the effects of an acute psychosocial stressor on visuospatial memory performance and on planning and organisation of visuospatial information. It will use the ROCF, thus allowing my results to be comparable to those of previous studies, most of which have used that instrument.

We used an all-male sample to avoid cortisol confounds associated with female use of oral contraceptives and menstrual cycle stage (Hausmann, Schoofs, Rosenthal, & Jordan, 2009; Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Kirschbaum, Pirke, & Hellhammer, 1995).

Previous studies in this field have administered the stressor prior to taking the psychological measurement (i.e., prior to the copy trial (encoding phase) of the ROCF). Hence, they did not measure the effect of stress at any specific phase in the memory process: They could not distinguish whether the stressor affected the encoding (copy) or retrieval (recall) phase of the memory process. In the current study, stress was induced after the copy phase of the ROCF (i.e., after encoding had been completed) because this is the phase of the memory process most affected by stress (Smeets, 2011; Wolf, 2003).

Visuospatial information processing was measured using three outcome variables derived from ROCF performance: time to complete the drawing, accuracy of the drawing, and planning and organisational strategy underlying the drawing. Based on previous literature, I predicted that, compared to control participants, individuals exposed to the acute psychosocial stressor would (a) take more time to complete the ROCF recall trials; (b) create less accurate reproductions on those recall trials; and (c) show poorer planning and less gestalt-based organisational strategies in creating those reproductions.

Methods

Research Design and Setting

The study featured an experimental research design. Each of the participants was randomly assigned to either the Stress or Non-Stress group. Hence, the independent variable had two levels (stress versus no stress). The dependent variables were various outcomes related to administration of the ROCF copy, short-delay recall, and long-delay recall trials.

All experimental procedures took place between 14h00 and 18h00 to control for cortisol's diurnal cycle. Cortisol has a circadian rhythm, with levels peaking in the morning just after waking, and decreasing slowly over the course of the day, leading to the lowest levels in the late afternoon and evening (Dickerson & Kemeny, 2004; Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004; Lupien et al., 2002; Maheu, Kornik, Moszkowski, & Lupien, 2005). Studies using acute psychosocial stressors should take place in the late afternoon when cortisol levels are at their lowest and most constant, as this is when cortisol changes due to a stressor will be most easily identified (Dickerson & Kemeny, 2004; B. M. Kudielka, personal communication, June 5, 2008; Kudielka, Hellhammer, & Wüst, 2009). To maintain consistency of administration between the Stress and Non-Stress groups, control procedures also took place in the late afternoon.

The study took place in two venues at the Department of Psychology at the University of Cape Town (UCT). One venue was a computer laboratory where the cognitive testing, physiological measures, and questionnaire completion took place, and the second venue was the room where the participants underwent the experimental manipulation.

Participants

The study involved 22 male participants, recruited via the UCT Department of Psychology's Student Research Participation Program, aged between18 and 25 years. Each participant was randomly assigned (using procedures from the website www.random.org) to either the Stress group (n = 11) or to the Non-Stress group (n = 11).

All participants received course credit in exchange for participation. The UCT Department of Psychology's Research Ethics Committee and the UCT Faculty of Health Science's Human Research Ethics Committee granted ethical approval for all study procedures.

Exclusion criteria. These included (a) the presence of major depression and anxiety disorders, (b) the use of any steroid-based medication, and (c) a body mass index (BMI) of more than 25 or less than 19. Participants were also asked to refrain from eating or drinking anything (except water), smoking, and taking part in any form of exercise for at least 2 hours prior to their test session. These exclusion criteria have been identified as potentially confounding variables in research investigating the effects of psychosocial stress on cognitive performance (Kudielka et al., 2009), and are in line with criteria used in previous research in this field (Kirschbaum, Pirke, & Hellhammer, 1993; Schoofs et al., 2008; Schwabe & Wolf, 2010).

Materials

Participant self-report measurements.

Beck Depression Inventory-II (BDI-II). The BDI-II (Beck, Steer, & Brown, 1996) is a 21-item self-report questionnaire. Each item has four possible responses, with each response indicating a different degree of possible depressive symptomatology. Respondents are asked to choose the response that best describes their mood over the previous 2 weeks; higher scores indicate greater levels of depression. The distinct ranges of scores are: 0-13, minimally depressed; 14-19, mildly depressed; 20-28, moderately depressed; 29-63, severely depressed (Beck et al., 1996).

The BDI-II was developed in order to comply with the criteria for major depressive disorder set out in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). It is highly internally consistent (Dozois, Dobson, & Ahnberg, 1998) and has good test-retest reliability (Beck et al., 1996).

For the purposes of this study, the BDI-II was used as a screening measure, with participants scoring 29 or above being excluded from the study. All participants who were excluded based on this criterion were given the contact details for the Student Wellness Centre.

State-Trait Anxiety Inventory (STAI). The STAI (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) consists of two parts. Form Y-1 measures an individual's anxiety at a specific point in time (state anxiety), while Form Y-2 is an indicator of general levels of anxiety (trait anxiety). The STAI has good psychometric properties in that it has a reliable factor structure, is highly internally consistent, and has high levels of validity (Spielberger & Vagg, 1984).

For the purposes of this study, the STAI-Trait was used as a control measure to establish participants' general levels of anxiety (i.e., to ensure that, across groups, participants were experiencing similar levels of anxiety in their everyday lives). The STAI-State was used to measure changes in self-reported anxiety across the experimental procedures.

Positive and Negative Affect Schedule (PANAS). The PANAS (Watson, Clark, & Tellegen, 1988) is comprised of two 10-item mood scales, the Positive Affect (PA) scale and Negative Affect (NA) scales. The PA scale measures the extent to which the respondent feels enthusiastic, active, and alert, whereas the NA scale measures to extent to which the person feels unpleasant and distressed. The NA scale and not the PA scale is related to self-reported stress and coping. Intra-subject fluctuations in self- reported stress are highly correlated with fluctuations in NA but not in PA (Watson et al., 1988). For the purposes of this study, the NA scale was most relevant, and so it alone was used as a self-reported measure of stress.

The PANAS can be used with reference to a number of different time-frames. In the current study, the time-frame adopted was 'at the present moment'. When using this time frame, the PANAS is sensitive to changes in internal or external circumstances; thus, it is useful to use when measuring intra-individual mood fluctuations.

With regard to psychometric properties of the PANAS, the PA and NA scales have been shown to be highly internally consistent, and to be largely uncorrelated. The scales are

reliable and precise, and independent of the time frame and subject population (Watson et al., 1988).

Physiological measurements.

Heart rate and skin conductance measurements. ECG and skin conductance measurements were taken throughout the experimental session using the Vrije Universiteit Ambulatory Monitoring System, version 5fs (VU-AMS; Vrije Universiteit, Amsterdam, Holland). This non-invasive device is portable; hence, participants were able to move around and walk between the two study venues while wearing it.

The device was fitted at the beginning of the session. Following fitting, 5 minutes was allowed in order for the participants' heart rates to stabilise before the first measurement was taken. Average heart rate and skin conductance measurements were taken during each of the following periods: (a) a 2-minute baseline measurement immediately following the 5-minute stabilization period referred to above, (b) during the experimental manipulation, and (c) 5 minutes before the end of the session.

The acute psychosocial stressor. Stress group participants were exposed to the *Fear-Factor Stress Test* (*FFST*), a stress induction procedure developed in our laboratory (C. Du Plooy, personal communication, April 19, 2011) that combines two already existing and commonly used laboratory stressors, the Trier Social Stress Test (Kirschbaum et al., 1993) and/or the cold pressor test (Schoofs et al., 2009). The room in which the induction took place featured bright lights, a video camera, and a two-person judging panel.

Each participants was read a set of standardized instructions asking him to imagine that he was undergoing an audition for *Fear Factor,* and that he must therefore convince a panel of two judges that he is a suitable person to be on the reality television show. He was told that the judges were behavioural health experts who, with the aid of a video recording would analyse his verbal and nonverbal behaviour.

The participant was further told that the audition would be comprised of three tasks: 1) a 5-min free motivational speech stating why he should be included on *Fear Factor*; 2) a 5-min mental arithmetic task, demonstrating that he is able to think under pressure; and 3) a 2-min submersion of the dominant arm in cold water, demonstrating that he is able to withstand the physical demands of the television show.

The participant was then given 10 minutes to prepare the speech. The participant was then given 5 minutes to present his speech. If he stopped speaking before the time elapsed, one of the judges said, "You still have time left, please continue." If the participant was unable to continue, the judges asked a series of prompting questions (e.g., "What are your weaknesses?" or "Why should we not take you?"). Following the 5-min speech, the participant was asked to perform a serial subtraction (of 17, starting from 2043) for 5 minutes. If the participant performed an incorrect subtraction, he was asked to re-start the task from the beginning. Finally, the participant was asked to submerge his dominant arm in cold water (between 0 and 4ºC) for as long as possible (up to a maximum of 2 minutes). The participant remained standing throughout.

Cortisol samples were not analysed in this study as an indicator of the stress response. This is because similar studies previously run in the same laboratory, using an identical or very similar stressor and sampling from the same population, have demonstrated that this stress manipulation procedure is effective and increases salivary cortisol levels reliably (du Plooy, Henry, Human, & Thomas, 2011; Human et al., 2010).

The control condition. Administration of this condition occurred in the same room as that of the stress-induction condition. In this case, however, the room had normal lighting, and no video camera and judges were present.

Participants in the Non-Stress group underwent tasks that were similar in physical and mental type to those stress condition, but without the negative stress-inducing components (i.e., the social evaluative and pain components were absent). Specifically, each participant was provided with a blank sheet of paper and was given 10 minutes to write a summary of everything he had done on that day. After that 10-min period, he was asked to stand and read aloud out of a magazine. The researcher would then leave the room so that the participant was left alone in the room while he read aloud for 5 minutes. The researcher then re-entered the room and asked the participant to count upwards in multiples of five, starting from zero. Again, the researcher left the room and the participant would perform this task aloud for 5 minutes while standing alone in the room.

After completion of that task, the researcher re-entered the room and asked the participant to submerge his arm into warm water (35-37 ºC) for a maximum of 2 minutes. The researcher remained in the room but did not watch the participant directly.

Measure of visuospatial information processing. The *Rey-Osterrieth Complex Figure Test* (*ROCF*; Osterrieth, 1944; Rey, 1941) consists of three trials: copy, short-delay

recall, and long-delay recall. The copy trial requires the participant to draw, as precisely as possible, a complex geometric figure modelled on a card presented to him by the experimenter. The short-delay and long-delay (typically after a 15-40 minute filled delay) recall trials requires the participant to draw the figure again from memory (Shin, Park, Park, Seol, & Kwon, 2006). The recall trials can be a measure of visuospatial memory performance and visuoconstructional and planning ability (Strauss, Sherman, & Spreen, 2006).

Numerous quantitative and qualitative scoring methods have been developed to measure ROCF performance (Strauss et al., 2006). Quantitative scoring methods are most popular. The 36-point system first developed by Osterrieth (1944) and refined into a more stringent form by Taylor (1991) is the most widely used quantitative measure of accuracy of the drawing. Within this scoring system, the figure is broken down into 18 scorable elements, with the participant awarded a score of 0-2 for each element, depending on accuracy and placement of the element. Specifically, no points are awarded if the element is absent or non-recognisable; 0.5 points are awarded if the element is distorted and poorly placed; 1 point is awarded if the element is distorted but placed correctly, or if the element is drawn correctly but placed poorly; 2 points are awarded if the element is drawn correctly and placed correctly (Strauss et al., 2006).

Quantitative scoring systems, although popular and a conventional way to measure ROCF performance, cannot capture qualitative aspects of the approach taken to completing the copy and recall tasks (Shin et al., 2006). Hence, researchers have developed various qualitative scoring methods to assess the process individuals use when drawing the figure. These methods mostly require the participant to be given different coloured pencils to use as they draw the figure, thus allowing approach and strategy to be identified.

One example of such a scoring system is the Rey Complex Figure Organisational Strategy Score (RCF-OSS; Anderson, Anderson, & Garth, 2001). This is a 7-point scoring system that identifies seven levels of conceptual strategies, based on organisation and planning, commonly utilised when completing the ROCF. The score given depends on these seven levels. The most basic level exhibits no attempt to draw the figure so that the drawing is unrecognisable. The second and third levels feature poor and random organisation. At these levels, there is some attempt to draw the figure. The third level has at least one configural element. The fourth level indicates that a piecemeal approach was taken. The fifth to seventh levels have the main configural elements drawn early, with the most advanced strategy

featuring the drawing of the main configural elements before any other part of the figure (Anderson et al., 2001).

With regard to the psychometric properties of the ROCF, it measures visuospatial memory performance with high validity (Poulton & Moffitt, 1995). This validity is shown by the high correlation (r = .88) between performance on the short-delay and long-delay recall trials, indicating a single aspect of memory is being measured. Furthermore, convergent validity studies have found that copy, short-delay, and long-delay recall scores correlate significantly with scores on other tasks measuring memory and constructional abilities. Moreover, ROCF performance does not correlate with performance on verbal memory tasks, suggesting the measure has discriminant validity (Strauss et al., 2006).

Regarding internal consistency, both split-half and coefficient alpha reliabilities have been reported to be greater than .60 for the copy trial and greater than .80 for the short-delay and long-delay recall trials (Berry, Allen, & Schmitt, 1991; Fastenau, Bennet, & Denberg, 1996). Test-retest reliability is also reportedly high, with a 91.7% correlation between two measurement sessions 6 months apart (Meyers & Meyers, 1995). Interrater reliability of the Taylor scoring system has also been shown to be high (> .90) for total scores (Strauss et al., 2006). Interrater reliability of the RCF-OSS is similarly acceptable and is estimated at .85 to .92 (Anderson et al., 2001).

Procedure

All participants were met at the computer laboratory where the memory testing was to take place. They were asked to read and sign the consent form (Appendix A), complete the BDI-II, PANAS, and STAI-Trait questionnaires, and have their BMI measured. Only participants meeting the BDI-II or BMI inclusion criteria continued. The participant was then fitted with the VU-AMS device. A 5-min period was allowed for the device to normalise to the participants' heart rate, and then a 2-min baseline reading was taken.

Participants were then administered the ROCF copy trial, following conventional procedures described by Strauss et al. (2006). They then underwent the 22-minute FFST procedure in the second venue; and during the procedure a second heart rate and skin conductance measurement was taken. This was followed by a 5-min relaxation period back in the computer laboratory. Afterwards, the participants completed a second STAI-State and PANAS questionnaire. At this point, the ROCF short-delay recall trial was administered. Twenty to thirty minutes following the short-delay recall trial, the long-delay recall trial was

administered. Afterwards, 5 minutes before the end of the session, the third heart rate and skin conductance measurement was taken, and the third STAI-State and PANAS questionnaires were completed. Finally, the participants were debriefed and dismissed.

Statistical Analysis

All statistical analyses were completed using SPSS version 19.0. The level for statistical significance was set at $\alpha = 0.05$. Details for the specific analyses are provided before the presentation of the results. Unless otherwise stated, all of the required assumptions were upheld for each of the statistical analyses.

Results

Sample Characteristics

Table 1 shows descriptive statistics regarding the sample characteristics.

Table 1
Sample Characteristics: Descriptive statistics

	Group	
	Stress	Non-Stress
Measure	(n = 11)	(n = 11)
Age	20.18 (1.40)	20.18 (1.41)
BDI-II score	12.27 (6.29)	10.73 (6.21)
BMI	23.09 (1.83)	22.37 (3.75)
STAI - Trait score	42.73 (10.99)	39.82 (10.94)

Note. Means are provided with standard deviations in parentheses.

Participants' ages ranged from 18 to 25 years ($M = 20.17 \pm 1.34$); an independent samples t-test detected no statistically significant between-group differences, $t(20) = 0.00$, $p = 1.00$, Cohen's $d = 0.0$. It is important that there were no between-group age differences as cortisol levels tend to increase with age (Larsson, Gullberg, Råstam, & Lindblad, 2009).

BDI-II scores were, on average, relatively low for both of the groups, with the mean for each falling with the range conventionally described as 'minimally depressed' (0-13). An independent-samples t-test detected no statistically significantly between-group differences on this measure, $t(20) = -0.58$, $p = .569$, $d = 0.25$. Additionally, the sample seemed to be

representative of the general population: When compared to the normative data for male college students (M = 12.56 ± 9.93) supplied by the test manual (Beck et al., 1996), a single-sample t-test was not statistically significant, $t(21) = -0.81$, $p = .428$.

Regarding STAI - Trait anxiety scores, an independent-samples t-test detected no statistically significant between-group differences, $t(20) = -0.62$, $p = .541$, $d = 0.27$, suggesting that, across groups, participants experienced equivalent general levels of anxiety. Additionally, the sample seemed to be representative of the general population: When compared to the normative data for male college students (M = 38.30 ± 9.18) supplied by the test manual (Spielberger et al., 1983), a single-sample t-test was not statistically significant, $t(21) = 0.85$, $p = .406$.

Taken together, the BDI and STAI-Trait data imply that, with regard to current mood and trait anxiety, the Stress and Non-Stress groups were not significantly different from each other and from the general population of male college students. Hence, results related to the experimental manipulation were not confounded by pre-existing high levels of negative emotional states in either group.

Average BMI was within the normal range of 19-25 for both groups, and there were no statistically significant between-group differences, $t(20) = -0.57$, $p = .577$, $d = 0.24$. Thus, between-group differences in BMI did not confound the effects of the stress manipulation by influencing cortisol levels. It is important to control for BMI because there is a positive correlation between cortisol excretion rate and BMI ($\chi^2 = 0.34$, $p < .001$; Fraser et al., 1999).

Experimental Manipulation Check

The following analyses sought to ensure that, with regard to stress-related self-report and physiological measures, (a) participants in the two groups were not significantly different before the experimental manipulation, (b) they were different after the manipulation, and (c) they had returned to baseline when they left the session (i.e., that the experimental manipulation did not have long-lasting effects on them). For each of the relevant measures, 2 x 3 (Group [Stress/Non-Stress] x Testing Stage [baseline/post-manipulation/end of session]) mixed-designs ANOVAs were conducted and planned comparisons were run to test pre-existing hypotheses about where exactly between- and within-group differences would exist. Table 2 shows the descriptive statistics for these measures.

Table 2
Stress-Related Self-Report and Physiological Measures: Descriptive statistics

Measure	Group	
	Stress (n = 11)	Non-Stress (n = 11)
STAI - State		
Baseline	40.18 (10.57)	36.36 (7.06)
Post-manipulation	41.45 (11.34)	31.64 (5.94)
End of Session	37.09 (7.79)	33.81 (5.62)
PANAS - NA Scale		
Baseline	14.64 (4.48)	14.45 (3.36)
Post-manipulation	16.55 (7.01)	11.82 (1.66)
End of Session	13.09 (2.81)	12.09 (1.92)
Heart rate[a]		
Baseline	71.07 (11.22)	74.60 (13.34)
During manipulation	87.60 (12.68)	86.75 (15.33)
End of Session	69.87 (9.84)	68.97 (7.21)
Skin conductance response[b]		
Baseline	8.64 (2.47)	5.64 (3.19)
During manipulation	16.19 (6.18)	10.26 (4.82)
End of Session	14.89 (5.38)	11.50 (5.39)

Note. Means are provided with standard deviations in parentheses. Heart rate levels are measured in beats per minute (bpm). Skin conductance response is measured in uS. [a]n = 6 at each measurement point. [b]n = 7 at each measurement point.

Self-report measures of stress. Regarding STAI - State anxiety scores, a single-sample t-test confirmed that, at baseline, the overall mean of the sample (M = 38.17 ± 8.78) was not significantly statistically different from the normative data for college students (M = 36.47 ± 10.02) supplied by the test manual (Spielberger et al, 1983), $t(21) = 0.94$, $p = .358$. Again, these data indicate that the current sample was representative of the general population and that the participants were not feeling excessively anxious at the start of the session.

The mixed-design ANOVA conducted on the data depicted in Figure 1 revealed that there was no statistically significant within-subjects main effect of Testing Stage, $F(2, 40) = 1.07$, $p = .352$, partial $\eta^2 = .051$ (baseline: $M = 38.27 \pm 8.99$; post-manipulation: $M = 36.55 \pm 10.16$; end of session: $M = 35.46 \pm 6.84$). There was also no statistically significant between-group main effect, $F(1, 20) = 4.16$, $p = .055$, partial $\eta^2 = .172$ (Stress group: $M = 39.58 \pm 9.89$; Non-Stress group: $M = 33.94 \pm 12.79$). Furthermore, there was no statistically significant Group x Testing Stage interaction effect, $F(2, 40) = 1.75$, $p = .187$, partial $\eta^2 = .08$.

Because the between-group main effect tended towards statistical significance, it was important to examine this effect more closely via the following set of planned pairwise comparisons:

1) Stress group vs. Non-stress group at baseline
2) Stress group vs. Non-stress group at post-manipulation
3) Stress group vs. Non-stress group at end of session

These three comparisons test the questions of interest directly: First, were the groups different before at baseline; second, were they different after the manipulation (i.e., was the manipulation effective?); third, were their subjective experiences of stress different at the end of the study? The analyses revealed the following: First, there were no statistically significantly between-group differences at baseline, $p = .331$, $d = 0.43$; second, participants in the Stress group reported significantly higher levels of state anxiety post-manipulation than did participants in the Non-Stress group, $p = .019$, $d = 1.08$; third, there were no statistically significantly between-group differences at the end-of-session measurement point, $p = .272$, $d = 0.48$.

Ethically, it was important to demonstrate that the FFST procedure did not have lasting effects on the participants in the Stress group; in other words, I had to ensure that they did not leave the study still feeling anxious from their exposure to the experimental manipulation, but rather left in an affective state similar to that in which they arrived. A repeated measures *t*-test showed that, for participants in that group, subjective levels of anxiety were not significantly different at the end-of-session measurement point than at baseline, $t(1,10) = 1.46$, $p = .255$, partial $\eta^2 = .127$. Given that their baseline levels of state anxiety were comparable to those in the general population, the implication here is that the Stress group participants were not still in a subjectively stressed state at the end of the session.

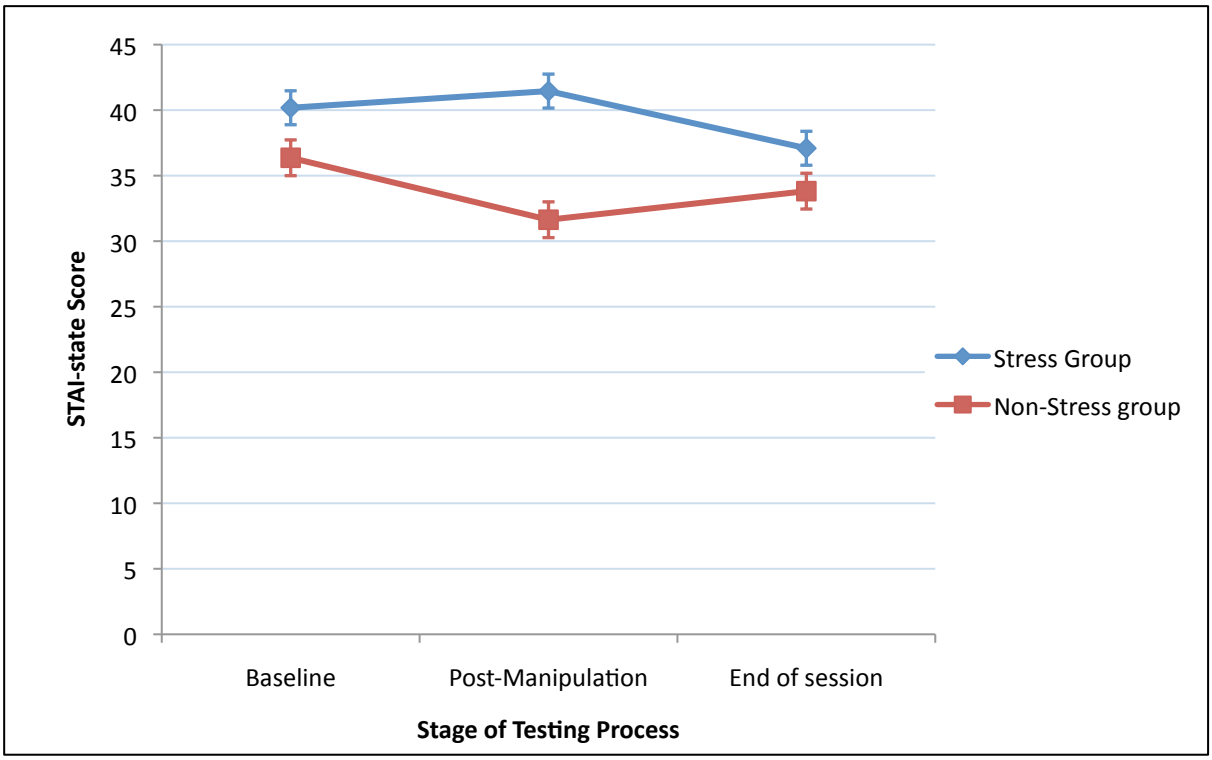

Figure 1. Changes in self-reported state anxiety levels for the Stress and Non-Stress groups. Error bars indicate standard error of means.

Regarding data from the PANAS – NA Scale, the assumption of homogeneity of variance was violated at the post-manipulation measure of the between-group main effect, Levene's test for equal variances, $F(1, 20) = 10.29$, $p = .004$. However, ANOVA is a robust enough test to withstand such violations of this assumption if there are equal group sizes. Hence, I continued with the analysis in the conventional manner.

The mixed-design ANOVA conducted on the data depicted in Figure 2 revealed that there was no statistically significant within-subjects main effect of Testing Stage, $F(2, 40) = 2.52$, $p = .093$, partial $\eta^2 = .112$ (baseline: $M = 14.55 \pm 3.86$; post-manipulation: $M = 14.18 \pm 5.53$; end of session: $M = 12.59 \pm 2.40$). There was also no statistically significant between-groups main effect, $F(1, 20) = 2.25$, $p = .149$, partial $\eta^2 = .101$ (Stress group: $M = 14.76 \pm 5.11$; Non-Stress group: $M = 12.79 \pm 2.64$). There was, however, a statistically significant Group x Testing Stage interaction effect, $F(2, 40) = 3.42$, $p = .043$, partial $\eta^2 = .146$.

The same set of three planned pairwise comparisons as used above allowed closer examination of this significant interaction effect. The analyses revealed the following: First, there were no statistically significantly between-group differences at baseline, $p = .915$, $d = 0.05$; second, participants in the Stress group reported significantly higher levels of negative affect post-manipulation than did participants in the Non-Stress group, $p = .042$, $d = 0.93$;

third, there were no statistically significantly between-group differences at the end-of-session measurement point, $p = .341$, $d = 0.42$.

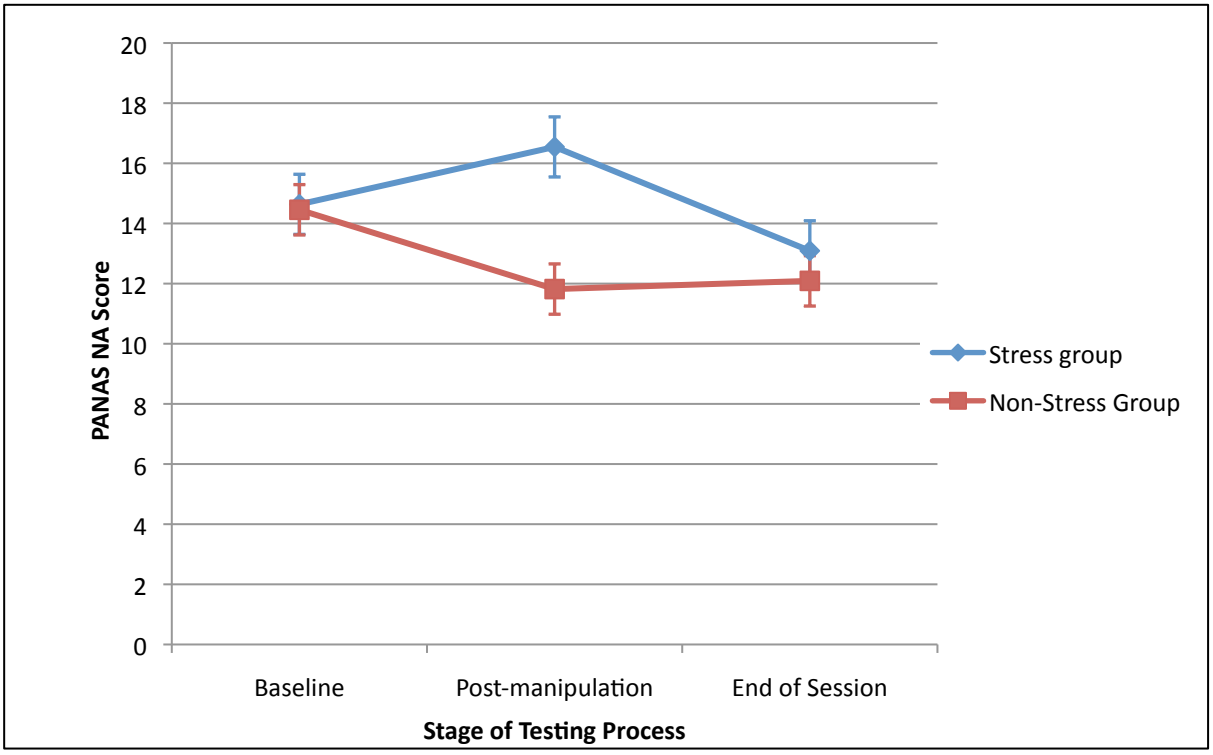

Figure 2. Changes in self-reported negative affect levels for the Stress and Non-Stress groups. Error bars indicate standard error of means.

Again, from an ethical standpoint it was important to demonstrate that the FFST procedure did not have long-lasting effects on PANAS – NA scale scores. A repeated measures *t*-test showed that, for Stress group participants, these scores were not significantly different at the end-of-session measurement point than at baseline, $t(1,10) = 1.35$, $p = .272$, partial $\eta^2 = .119$. The implication here, then, is that the Stress group participants did not report higher levels of negative affect at the end of the session than they did when they arrived.

Physiological stress measures of stress. Numerous sets of physiological data were lost due to hardware malfunctioning. Heart rate measures could only be collected for 6 participants in each group, and skin conductance rate (SCR) data could only be collected for 7 participants in each group. This rate of data loss is similar to that reported by other studies using the VU-AMS in our laboratory (Henry & Human, 2008; Human et al., 2010).

Regarding heart rate, the mixed-design ANOVA conducted on the data depicted in Figure 3 revealed that there was a statistically significant within-subjects main effect of Testing Stage, $F(2, 20) = 70.05$, $p < .001$, partial $\eta^2 = .875$ (baseline: $M = 72.83 \pm 11.90$; post-manipulation: $M = 87.18 \pm 13.42$; end of session: $M = 69.42 \pm 8.24$). There was no statistically significant between-groups main effect, $F(1, 10) = 0.01$, $p = .930$, partial $\eta^2 = .001$ (Stress group: $M = 76.178 \pm 13.24$; Non-Stress group: $M = 76.77 \pm 13.97$), or Group x Testing Stage interaction effect, $F(2, 20) = 1.28$, $p = .300$, partial $\eta^2 = .113$.

Despite this lack of statistically significant effects related to Group, it was still important to determine whether the Stress and Non-Stress groups were different at baseline and at the end of the session, for the reasons mentioned above. Hence, the same set of three planned pairwise comparisons as above were performed. The analyses revealed the following: There were no statistically significantly between-group differences at baseline, at post-manipulation, or at the end of the session, $p = .630$, $d = 0.29$, $p = .919$, $d = 0.06$ and $p = .860$, $d = 0.11$ respectively. Possible reasons for this unexpected result will be discussed later.

A second set of planned pairwise comparisons allowed further examination of the statistically significant within-subjects main effect of Testing Stage. The comparisons were:

1) Baseline vs. during manipulation for the Non-Stress group
2) Baseline vs. during manipulation for the Stress group

These comparisons allow direct examination of the question of whether, for each group separately, there was a significant increase in heart rate from baseline to the during-manipulation measurement point for each of the groups. The analyses revealed the following: First, in both the Non-Stress and Stress groups there was a statistically significant increase from the first to the second measurement point, $p < .001$, partial $\eta^2 = .871$, and $p < .001$, partial $\eta^2 = .952$, respectively. This increase in heart rate within the Stress group is consistent with a priori predictions and points to the effectiveness of the experimental manipulation; however, the fact that there was an increase of similarly large magnitude in the Non-Stress group is a surprising result that will be discussed in more detail later.

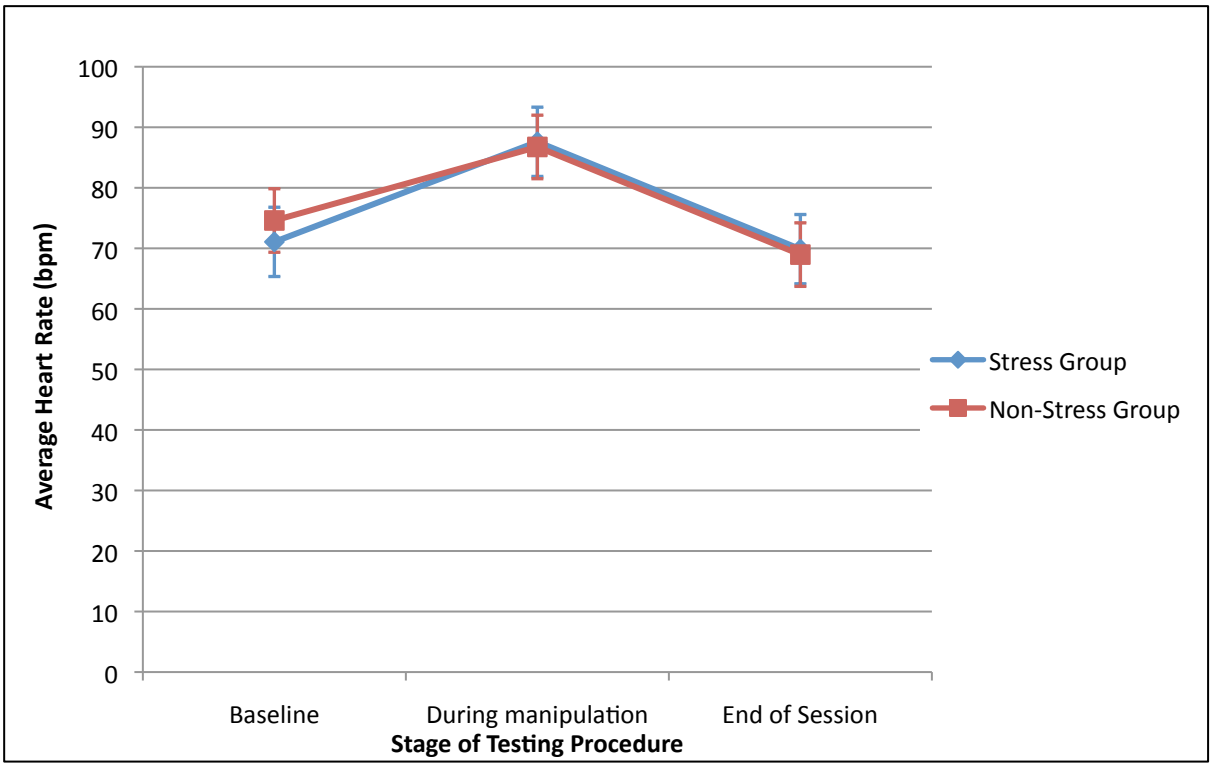

Figure 3. Changes in average heart rate for the Stress and Non-Stress groups. Error bars indicate standard error of means.

Regarding SCR data, the assumption of sphericity required for the repeated-measures ANOVA was violated Mauchly's test of sphericity, $\chi^2(2) = 9.67$, $p = .008$. Hence, a Greenhouse-Geisser degrees of freedom correction ($\varepsilon = .631$) was performed, and the rest of the analysis proceeded in the conventional manner.

The mixed-design ANOVA conducted on the data depicted in Figure 4 revealed that there was a statistically significant within-subjects main effect of Testing Stage, $F(2, 24) = 34.45$, $p < .001$, partial $\eta^2 = .742$ (baseline: $M = 7.14 \pm 3.15$; post-manipulation: $M = 13.22 \pm 6.15$; end of session: $M = 13.19 \pm 5.46$). There was no statistically significant between-groups main effect, $F(1, 12) = 3.06$, $p = .106$, partial $\eta^2 = .203$ (Stress group: $M = 13.24 \pm 5.78$; Non-Stress group: $M = 9.13 \pm 5.04$), or Group x Testing Stage interaction effect, $F(2, 24) = 1.78$, $p = .204$, partial $\eta^2 = .129$.

Despite this lack of statistically significant effects related to Group, it was still important to determine whether the Stress and Non-Stress groups were different at baseline and at the end of the session, for the reasons mentioned above. Hence, the same set of three planned pairwise comparisons as above were performed. The analyses revealed the following: There were no statistically significantly between-group differences at baseline, at post-manipulation, or at the end of the session, $p = .073$, $d = 1.05$, $p = .069$, $d = 1.07$, and $p = .262$,

$d = 0.63$, respectively. Possible reasons for this unexpected result will be discussed later. The same set of planned pairwise comparisons as used above investigated the significant interaction effect. The analyses revealed the following: First, in both the Non-Stress and Stress groups there was a statistically significant increase from the first to the second measurement point, $p = .010$, partial $\eta^2 = .761$, and $p < .001$, partial $\eta^2 = .733$, respectively. This increase in heart rate within the Stress group is consistent with a priori predictions and points to the effectiveness of the experimental manipulation; however, the fact that there was an increase of similarly large magnitude in the Non-Stress group is a surprising result that will be discussed in more detail later.

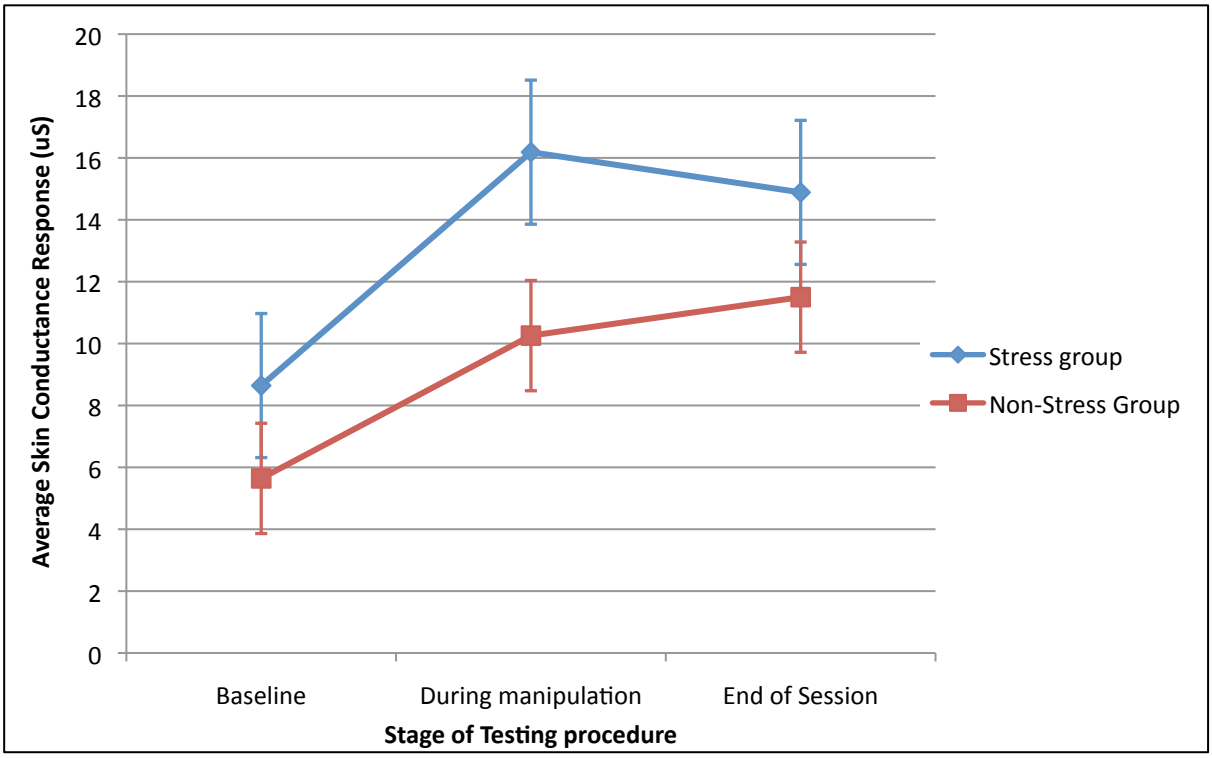

Figure 4. Changes in average skin conductance response for the Stress and Non-Stress groups. Error bars indicate standard error of means.

Visuospatial Memory Performance

Two independent raters blind to the condition scored the participants' ROCFs to ensure reliability in scoring. For the quantitative system, inter-rater reliability was $r = 0.98$. For the qualitative system outline above inter-rater reliability was $r = 0.89$. The averages of the two raters' scores were used as data in the analyses presented below.

Table 3 shows the descriptive statistics for all of the ROCF outcome variables. Analyses of these data proceeded as follows: First, independent-samples *t*-tests were used to

compare the groups on each of the copy-stage measures. Second, 2 x 2 (Group [Stress/Non-Stress] x ROCF Trial [Short-delay Recall/Long-delay Recall]) mixed-design ANOVAs were conducted on the recall measures. Third, a set of two planned pairwise comparisons (1. Stress group vs. Non-stress group at short-delay recall; 2. Stress group vs. Non-stress group at long-delay recall) were run to test pre-existing hypotheses about where exactly between- and within-group differences would exist on these measures.

Table 3
ROCF Performance: Descriptive statistics

	Group	
Measure	Stress (n = 11)	Non-Stress (n = 11)
Time		
Copy	179.73 (65.37)	153.82 (55.12)
Short-delay recall	178.73 (74.53)	131.27 (49.41)
Long-delay recall	163.27 (76.11)	96.82 (36.48)
Accuracy		
Copy	34.41 (1.38)	34.43 (1.26)
Short-delay recall	19.70 (6.30)	20.11 (4.42)
Long-delay recall	19.86 (5.85)	21.18 (2.97)
Planning and Organisation		
Copy	5.09 (1.18)	5.63 (0.64)
Short-delay recall	4.41 (1.02)	4.68 (0.84)
Long-delay recall	4.09 (1.14)	4.86 (0.71)

Note. Means are provided with standard deviations in parentheses. Time is measured in seconds. The maximum possible score for accuracy (quantitative scoring of the figure) is 36 points. The maximum possible score for planning (qualitative scoring of approach to the figure) is 7 points.

Time. As expected, there were no between-group differences on the copy trial, $t(1,20) = -1.01$, $p = .327$, $d = 0.43$. Regarding the mixed-design ANOVA conducted on time taken to complete the two recall trials (see Figure 5), the assumption of homogeneity of variance was violated for the long-delay recall data at the between-group main effect, Levene's test for equal variances, $F(1, 20) = 9.54$, $p = .006$. However, ANOVA is a robust enough test to

handle such violations if there are equal group sizes. Hence, the analysis proceeded in conventional fashion.

There was a statistically significant within-subject main effect of Trial, $F(1, 20) = 6.88$, $p = .016$, partial $\eta^2 = .256$ (short-delay: $M = 155 \pm 66.32$; long-delay: $M = 130.05 \pm 67.47$), and a statistically significant between-groups main effect, $F(1, 20) = 5.43$, $p = .030$, partial $\eta^2 = .214$ (Stress group: $M = 171 \pm 73.94$; Non-Stress group: $M = 114.05 \pm 45.94$). There was no statistically significant Group x Trial interaction effect, $F(1, 20) = 1.10$, $p = .330$, partial $\eta^2 = .047$. The set of planned comparisons yielded the following results: First, at short-delay recall there were no statistically significant between-group differences, $p = .094$, $d = 0.75$; second, at long-delay recall participants in the Stress group took a statistically significantly longer time to complete their drawing than did those in the Non-Stress group, $p = .017$, $d = 1.11$.

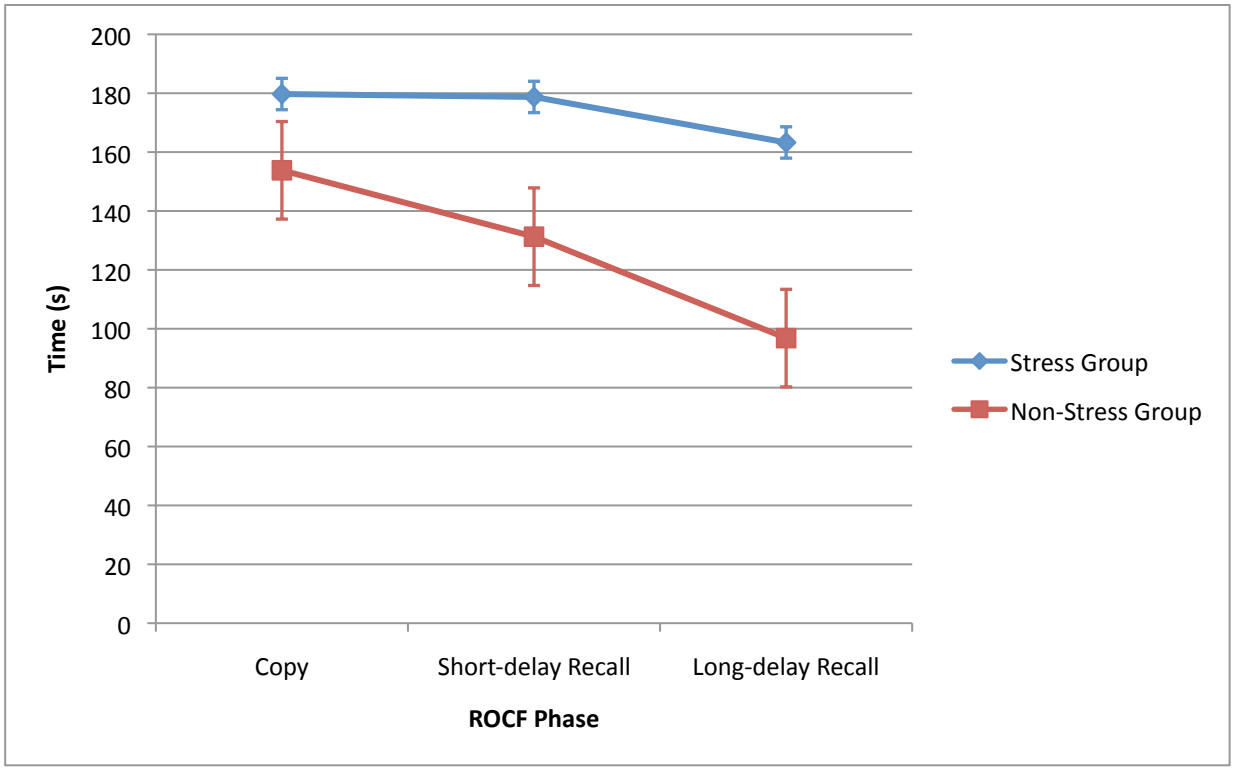

Figure 5. Time taken to complete the ROCF trials for the Stress and Non-Stress groups. Error bars indicate standard error of means.

Accuracy. As expected, there were no between-group differences on the copy trial, $t(1,20) = 0.40$, $p = .968$, $d = 0.02$. Regarding the mixed-design ANOVA conducted on accuracy scores on the two recall trials (see Figure 6), the assumption of homogeneity of variance was violated for the long-delay recall data at the between-group main effect,

Levene's test for equal variances, $F(1, 20) = 4.38$, $p = .049$. However, ANOVA is a robust enough test to handle such violations if there are equal group sizes. Hence, the analysis proceeded in conventional fashion.

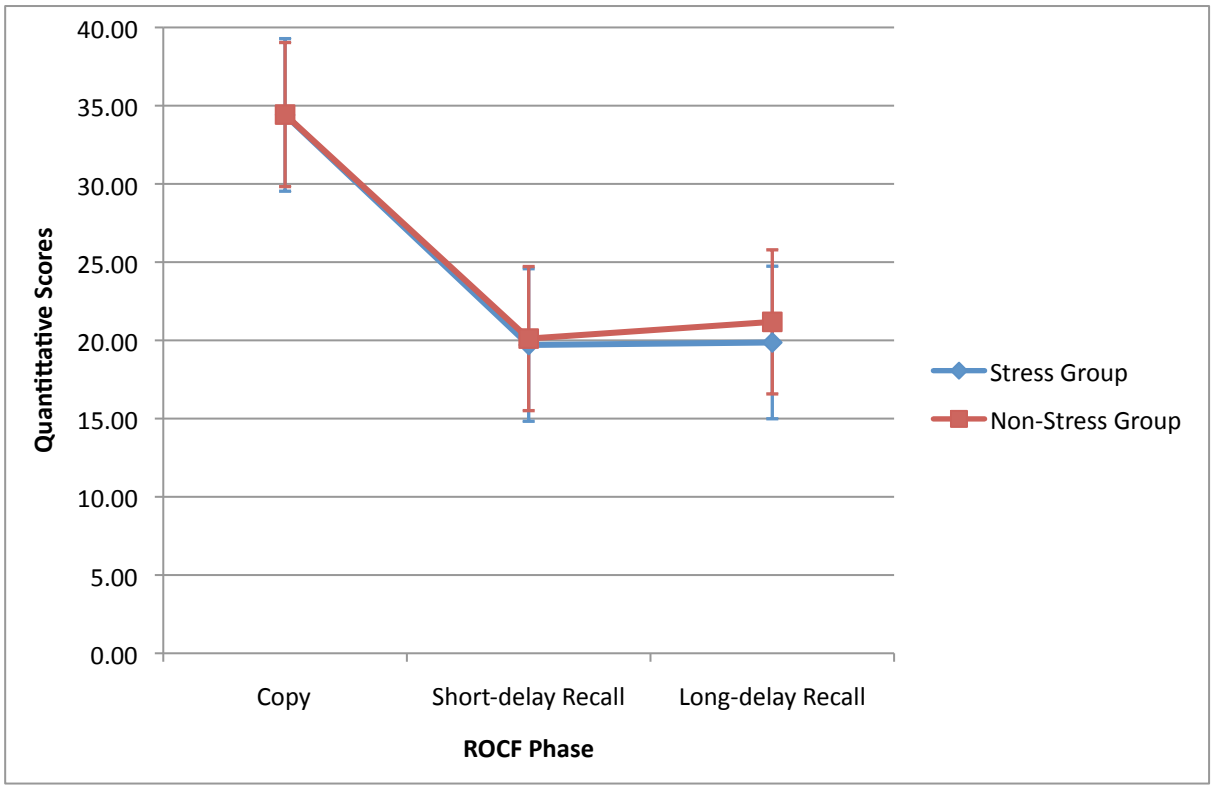

Figure 6. ROCF accuracy performance across trials for the Stress and Non-Stress groups. Error bars indicate standard error of means.

There was no statistically significant within-subjects main effect of Trial, $F(1, 20) = 0.94$, $p = .345$, partial $\eta^2 = .045$ (short-delay: $M = 19.91 \pm 5.32$; long-delay: $M = 20.52 \pm 4.58$), no statistically significant between-groups main effect, $F(1, 20) = 0.18$, $p = .680$, partial $\eta^2 = .009$ (Stress group: $M = 19.78 \pm 5.93$; Non-Stress group: $M = 20.65 \pm 3.72$), and no statistically significant Group x Trial interaction effect, $F(1, 20) = 0.51$, $p = .482$, partial $\eta^2 = .025$. The set of planned comparisons yielded the following results: At both short- and long-delay recall there were no statistically significant between-group differences, $p = .862$, $d = 0.08$ and $p = .513$ $d = 0.28$, respectively.

To further investigate between-group differences in accuracy of ROCF reproductions, I calculated the percentage recall from the copy trial to each of the short- and long-delay recall trials for each participant. There were no statistically significant between-group differences at either short-delay recall (Stress group: $M = 57.44\% \pm 18.93\%$; Non-Stress

group: $M = 58.54\% \pm 13.0\%$), $t(1, 20) = 0.16$, $p = .875$, $d = 0.07$, or at long-delay recall (Stress group: $M = 57.82\% \pm 17.60\%$; Non-Stress group: $M = 61.52\% \pm 8.25\%$), $t(1, 20) = 0.63$, $p = .538$, $d = 0.27$ (equal variances not assumed).

Planning and Organisation

As expected, there were no between-group differences on the copy trial, $t(1,20) = 1.35$, $p = .192$, $d = 0.57$. Regarding the mixed-design ANOVA conducted on planning scores on the two recall trials (see Figure 7), the assumption of homogeneity of variance was violated for the long-delay recall data at the between-group main effect, Levene's test for equal variances, $F(1, 20) = 7.10$, $p = .015$. However, ANOVA is a robust enough test to handle such violations if there are equal group sizes. Hence, the analysis proceeded in conventional fashion.

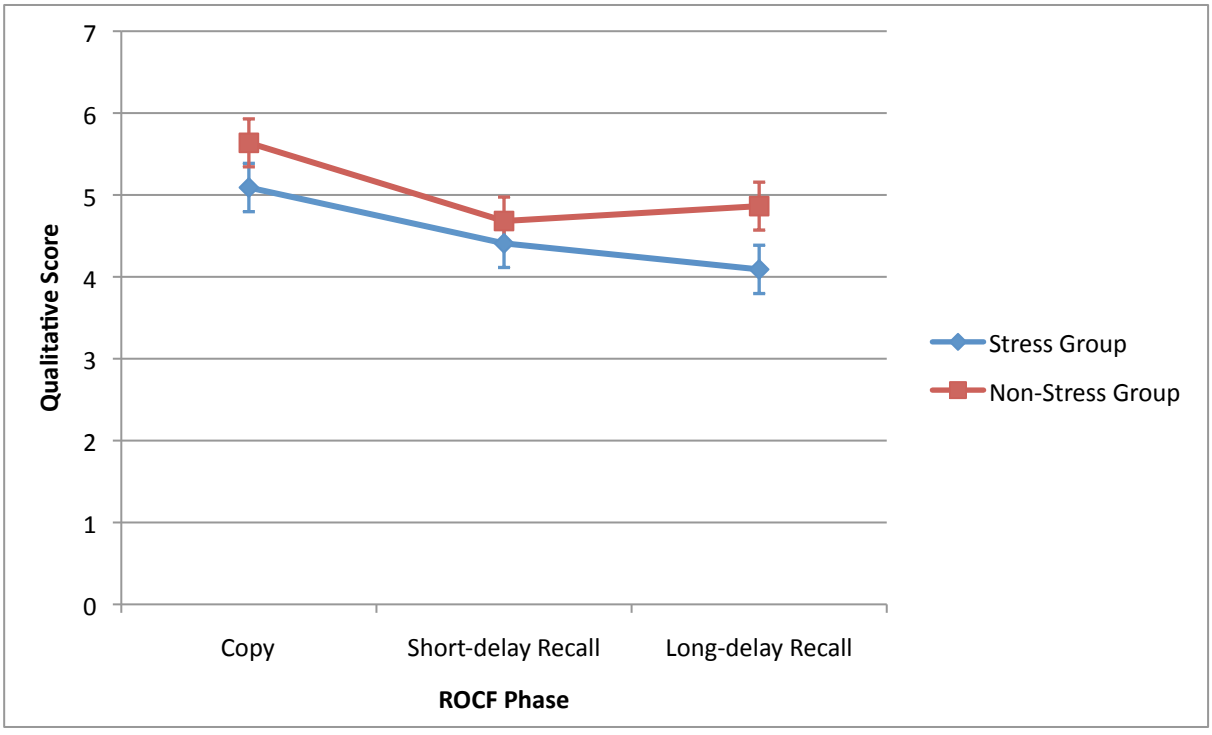

Figure 7. ROCF planning performance across trials for the Stress and Non-Stress groups. Error bars indicate standard error of means.

There was no statistically significant within-subjects main effect of Trial, $F(1, 20) = 0.15$, $p = .699$, partial $\eta^2 = .008$ (short-delay: $M = 4.55 \pm 0.92$; long-delay: $M = 4.48 \pm 1.01$), no statistically significant between-groups main effect, $F(1, 20) = 2.08$, $p = .164$, partial $\eta^2 = .094$ (Stress group: $M = 4.25 \pm 1.07$; Non-Stress group: $M = 4.77 \pm 0.77$), and no

statistically significant Group x Trial interaction effect, $F(1, 20) = 2.07$, $p = .166$, partial $\eta^2 = .094$. The set of planned comparisons yielded the following results: At both short- and long-delay recall, there were no statistically significant between-group differences, $p = .503$, $d = 0.29$ and $p = .070$, $d = 0.81$ respectively.

Discussion

The purpose of this study was to investigate the effects of acute psychosocial stress on visuospatial information processing in males. The hypotheses tested were that participants exposed to the acute psychosocial stressor would, relative to those exposed to a non-stressful control condition, (a) take more time to complete the ROCF recall trials, (b) create less accurate reproductions on those recall trials, and (c) show poorer planning and less gestalt-based organizational strategies in creating those reproductions.

Experimental Manipulation Check

Self-report data from the STAI - State and PANAS questionnaires suggested that the stress manipulation was effective. Specifically, it appeared that exposure to the FFST increased levels of subjective anxiety and negative affect significantly for participants in the Stress group. In contrast, exposure to the control condition did not increase levels of subjective anxiety and negative affect for participants in the Non-Stress group.

Data from the physiological measures were not as clear, however. Specifically, there were no significant between-group differences during the manipulation in terms of both heart rate and SCR. Examination of the raw data suggested that the reason for this non-significance is that there were increases, in both heart rate and SCR, in *both* groups. Hence, the FFST did effectively increase levels of physiological markers of stress in the Stress-group participants, and thus served its purpose; the complicating factor here is that the control condition also increased those levels.

It is important to note that the physiological measures taken here (heart rate and SCR) are indicators of ANS activity; cortisol increases are an indicator of HPA axis activity. As noted in the Introduction, HPA axis and ANS responses to stress are not necessarily synonymous. The onset of the ANS response is more rapid, and the response itself resolves quickly: Physiological markers of this response typically return to baseline 35 minutes after the onset of the stressor (du Plooy et al., 2011). In contrast, cortisol levels only peak approximately 20-40 minutes after the onset of the stressor (Alderson & Novack, 2002;

Kemeny, 2003; Wolf, 2003). Furthermore, under the conditions of the current study the increase in ANS measures in the Non-Stress group are not necessarily attributable to a stress response. Increases in ANS activity can also be a result of physical activity. Participants in the Non-stress group were asked to complete activities that were matched in physical and mental type to those in the stress condition, but without the negative stress-inducing components. Thus, increases in heart rate and SCR observed in the Non-stress group could be an indication of the physical demands of those activities.

For the purposes of this study, it was most important that the stress induction procedure increased cortisol levels, given that that hormone crosses the blood-brain barrier and then has effects on brain regions critical to higher cognitive processing. Previous work by our group (e.g., du Plooy et al., 2011; Human et al., 2010) has established that the stress induction procedure used here raises cortisol levels reliably, and that physiological stress response centered on the HPA axis (measured by, for example, the cortisol response) that is activated by the psychosocial stressor is of a longer duration than the physiological stress response centered on the autonomic nervous system (measured by, for example, heart rate and SCR) that is activated by the stressor. Our work has also shown that self-report measures of changes in subjective anxiety following the stress induction procedure correlate positively with magnitude of the cortisol response. Hence, one can make the assumption that, in this study, the FFST raised cortisol levels of participants in the Stress group, and ensured that those levels remained high throughout completion of the ROCF recall trials. One can further assume, based on the careful neurobiological work done by others (see, e.g., Alderson & Novack, 2002; Wolf, 2003) that cortisol crossed the blood-brain barrier and bound with the specific receptors in the hippocampus and prefrontal cortex (Alderson & Novack, 2002; Putman & Roelofs, 2011; Wolf, 2003). The likely outcome, then, is that participants in the Stress group were experiencing differential activation of the hippocampus and PFC than were participants in the Non-Stress group when they were asked to complete the ROCF recall trials.

Visuospatial Memory Performance

There were no between-group differences in terms of time to completion or accuracy of reproduction on the copy trial of the ROCF. Hence, any differences at recall have to be attributed to the experimental manipulation.

Time. The a priori hypothesis here was that participants in the Stress group would, on the recall trials, take longer than those in the Non-Stress group to complete their ROCF

drawings. This prediction was derived from the assumption that raised cortisol levels would impair the ability of Stress-group participants to retrieve the elements of the figure efficiently, and that they would therefore take longer than Non-Stress participants to complete their drawings.

This hypothesis was only partially confirmed. Although there were no significant between-group differences at short-delay recall, at long-delay recall Stress-group participants took longer to complete their drawings than did those in the Non-Stress group. This pattern of results can, however, be accounted for by the effects of the psychosocial stressor on the cortisol response: Cortisol levels peak only 20-40 minutes after the onset of the stressor, and typically return to baseline 40-60 minutes after the termination of the stressor (Alderson & Novack, 2002; Kemeny, 2003; Wolf, 2003); the long-delay recall trial took place 20-30 minutes after the end of the stressor (i.e., during the window when cortisol levels might have been at their peak), whereas the short-delay trial took place immediately after the stressor (i.e., before cortisol levels reached a peak).

In summary, the significant between-group differences in terms of time to completion on long-delay recall of the ROCF might be attributed to the inhibitory effects of stress on figure recall.

Accuracy. Using data from the 36-point quantitative scoring system (Osterrieth, 1944; Taylor, 1991), analyses suggested there were no significant between-group differences at either short- or long-delay recall. Hence, it appears that exposure to the psychosocial stressor had no effect on visuospatial memory performance.

The current results are not consistent with earlier studies reporting that stress either enhances or impairs visuospatial memory performance (Luethi et al., 2009; Morgan et al., 2006; Traverniers et al., 2010). There are many possible reasons for the discrepancy between the current results and those from previous studies. One reason could relate to a difference in stress-induction procedures: The studies reporting that stress impaired visuospatial memory performance (Morgan et al., 2006; Traverniers et al., 2010) used high-intensity stressors that are probably outside the bounds of everyday human experience. Therefore, those studies measured the effects of stress on memory performance that occur on the far right hand side of the inverted-U curve. In contrast, this study used a less intense everyday psychosocial stressor, and therefore measured the effects of stress on memory performance that occur toward the left-hand side of the inverted-U curve.

Hoffman and al'Absi (2004) also used a less intense everyday psychosocial stressor, and they also reported that no effects of stress on visuospatial memory performance (as measured by ROCF). In contrast to most studies in this area, Hoffman and al'Absi tested a majority female sample (10 males, 15 females). Their non-significant result is therefore interesting because the effects of cortisol are influenced greatly by fluctuations in the menstrual cycle and by the use of oral contraceptives (Hausmann et al., 2009; Kirschbaum et al., 1995, 1999) Consequently, the reason that Hoffman and al'Absi (2004) found that stress has no effect on visuospatial memory could be due to the influence of the fluctuations in the menstrual cycle and the use of oral contraceptives in the majority female sample.

Regarding the data reported by Luethi et al. (2009) showing that stress enhanced visuospatial memory performance, an important note is that those researchers used a different visuospatial memory assessment tool to most other studies in this area. Specifically, Luethi and colleagues used a learned route on a map to measure visuospatial memory, whereas most others, including this one, used the ROCF. Clearly, these instruments are quite different, and require the use of different cognitive abilities and different brain regions for solution; it is therefore not surprising that stress might differentially affect their completion.

Planning and Organisation

There were no between-group differences in terms of approach to the reproduction on the copy trial of the ROCF. Hence, any differences at recall have to be attributed to the experimental manipulation.

Using data from the RCF-OSS 7-point qualitative scoring system (Anderson et al., 2001), analyses suggested there were no significant between-group differences at either short- or long-delay recall. Of note, however, is the trend toward significantly better performance (p = .070, d = 0.81) by the Non-Stress group at long-delay recall. As noted above, this group difference is consistent with the fact that cortisol levels are at their peak during administration of the long-delay trial, thus making disruption of PFC functioning much more likely at that point than earlier. These results are consistent with previous studies showing that stress impairs various aspects of PFC functioning (Human et al., 2010; Luethi et al., 2009; Oei et al., 2006; Schoofs et al., 2008, 2009; van den Bos et al., 2009).

Overall, the results of the current study indicate that the acute psychosocial stressor had no effect on the accuracy of participants' reproduction of the complex figure at short- and long-delay recall. It appears, therefore, that, contrary to predictions, the induced level of

stress did not result in alteration of hippocampal functioning. However, the results did suggest that participants' planning of their reproduction of the complex figure was impaired by the induced stress at the long-delay recall trial. The fact that Stress-group participants also took significantly longer than Non-Stress participants to complete their reproductions at long-delay recall might be consistent with a lack of efficient planning and organisation during retrieval of the figure's elements. Taken together, these data suggest that the induced stress resulted in impairments in PFC functioning, but not in hippocampal functioning.

Limitations and Directions for Future Research

A major limitation of this study is its small sample size. Time constraints, and the stringently-applied eligibility criteria, prevented the recruitment of more participants. With a larger sample, the promising trends toward uncovering impaired effects of stress on ROCF performance at long-delay recall might have been clearer. A power analysis indicated that, given the effect sizes observed here and an alpha level of .05, a sample size of approximately $n = 20$ in each group would be required to deliver statistically significant results.

Another limitation of this study is that the sample was entirely male. This recruitment strategy was intentional; females are frequently excluded from stress research because of the confounding effects contraceptive use and menstrual cycle stage have on baseline cortisol levels and on magnitude of cortisol response in reaction to a stressor (Hausmann et al., 2009; Kirschbaum et al., 1995, 1999). Although previous research into the effects of the stress on visuospatial memory used either majority male or all-male samples (for instance, Morgan et al. (2006) included 166 men in total sample of 184 participants, Luethi et al. (2009) used 35 males only, and Traverniers et al. (2010) used 27 males only), future research might explore possible sex differences in visuospatial information processing following exposure to an acute stressor.

A wealth of literature shows that, in absence of stress, men perform better on tasks requiring visuospatial abilities and women perform better on tasks that require verbal abilities (see, e.g., Hausmann et al., 2009; Lewin, Wolgers, & Herlitz, 2001). Under stressful conditions, however, there are sex differences in other cognitive functions (Kirschbaum et al., 1996; Thomas, Laurance, Nadel, & Jacobs, 2010; van den Bos et al., 2009; Wolf, Schommer, Hellhammer, McEwen, & Kirschbaum, 2001) at least some of those functions tapping into the same neural regions that support planning and organisation.

Summary and Conclusion

Overall, this study indicates that there are impairing effects of stress on planning and organization, but only when the relevant cognitive tasks are administered at least 20 minutes post-stressor. This observation is consistent with the peak in cortisol levels that occurs 20-40 minutes after the onset of the stressor, and with presumed altered PFC functioning in response to those raised cortisol levels. Continued research into the effects of stress on visuospatial information processing is important because this cognitive ability underpins effective navigation through environmental space and effective memory for spatial layouts. If these cognitive processes are impaired by acute psychosocial stress, a critical aspect of human adaptive functioning is compromised.

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Appendix A

Consent Form

Informed Consent to Participate in Research and Authorization for Collection, Use, and Disclosure of Protected Health Information

This form provides you with information about the study and seeks your authorization for the collection, use and disclosure of your protected health information necessary for the study. The Principal Investigator (the person in charge of this research) or a representative of the Principal Investigator will also describe this study to you and answer all of your questions. Your participation is entirely voluntary. Before you decide whether or not to take part, read the information below and ask questions about anything you do not understand. By participating in this study you will not be penalized or lose any benefits to which you would otherwise be entitled.

1. **Name of Participant ("Study Subject")**

_____

2. **Title of Research Study**

   Effects of Acute Psychosocial Stress on Visuospatial Memory Performance in Healthy Humans

3. **Principal Investigators, Ethics Committee, and Telephone Numbers**

Kevin G. F. Thomas, Ph.D.	Robyn Human, MA	Anna Dreyer
	PhD Candidate	Honours student
Department of Psychology	Department of Psychology	
University of Cape Town	University of Cape Town	
021-650-4608	021-788-5536	

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Email: lamees.emjedi@uct.ac.za

4. **What is the purpose of this research study?**

   The purpose of this research study is to better understand how exposure to acute psychological stress affects cognitive performance. More specifically, we are interested in how the acute psychosocial stressor affects visuo-spatial memory performance.

5. **What will be done if you take part in this research study?**

   During this study, you will be required to complete a number of memory based tasks and may be required to complete a 20-minute presentation. Your levels of stress will be assessed through the collection of self-report data, heart rate measurements and skin conductance measurements.

   What are the possible discomforts and risks?

   If you are one of the participants selected to complete the 20-minute presentation, you may be placed in a mildly stressful situation involving public speaking. Furthermore, you may be asked to place your hand in very cold water. There are no other discomforts and risks associated with participation in the study.

7. **What are the possible benefits of this study?**

   One major benefit of this study is that scientists and society in general, will have better understanding of the effects of acute psychological stress on cognitive performance, and what variables moderate this relationship. This knowledge can then be applied to many different individuals and situations, including students who are taking exams, business managers who have to present to their boards, and so on.

8. **Can you withdraw from this research study and if you withdraw, can information about you still be used and/or collected?**

   You may withdraw your consent and stop participation in this study at any time. Information already collected may be used.

9. **Once personal information is collected, how will it be kept confidential in order to protect your privacy and what protected health information about you may be collected, used and shared with others?**

Information collected will be stored in locked filing cabinets or in computers with security passwords. Only certain people - the researchers for this study and certain University of Cape Town officials - have the legal right to review these research records. Your research records will not be released without your permission unless required by law or a court order.

If you agree to be in this research study, it is possible that some of the information collected might be copied into a "limited data set" to be used for other research purposes. If so, the limited data set may only include information that does not directly identify you.

Signatures

As a representative of this study, I have explained to the participant the purpose, the procedures, the possible benefits, and the risks of this research study; the alternatives to being in the study; and how the participant's protected health information will be collected, used, and shared with others:

_____ _____

Signature of Person Obtaining Consent and Authorization Date

You have been informed about this study's purpose, procedures, and risks; how your protected health information will be collected, used and shared with others. You have received a copy of this form. You have been given the opportunity to ask questions before you sign, and you have been told that you can ask other questions at any time.

You voluntarily agree to participate in this study. You hereby authorize the collection, use and sharing of your protected health information. By signing this form, you are not waiving any of your legal rights.

_____ _____

Signature of Person Consenting and Authorizing Date

Please indicate below if you would like to be notified of future research projects conducted by our research group:

_____ (initial) Yes, I would like to be added to your research participation pool and be notified of research projects in which I might participate in the future.

Method of contact:

Phone number: _____

E-mail address: _____

Mailing address: _____

PLAGIARISM DECLARATION

1. I know that plagiarism is wrong. Plagiarism is using another's work and to pretend that it is ones own.

2. I have used the American Psychological Association (APA) as the convention for citation and referencing. Each significant contribution to, and quotation in, this essay/report/project/… from the work, or works of other people has been attributed and
has cited and referenced.

3. This essay/report/project… is my own work.

4. I have not allowed, and will not allow, anyone to copy my work with the intention of passing it off as his or her own work.
5. I acknowledge that copying someone else's assignment or essay, or part of it, is wrong, and declare that this is my own work

SIGNATURE: _____

DATE: _____