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The impact of acute psychological stress on spatial cognition

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A minor dissertation submitted in partial fulfillment of the requirements for the award of the degree of Master of Social Science in Psychology

Faculty of the Humanities
University of Cape Town
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COMPULSORY DECLARATION

This work has not been previously submitted in whole, or in part, for the award of any degree. It is my own work. Each significant contribution to, and quotation in, this dissertation from the work, or works, of other people has been attributed, and has been cited and referenced.

Signature: ___________________________ Date: 12/2/2008
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ABSTRACT

Based on the premise that the hippocampus is both affected by cortisol and intimately involved in episodic memory and spatial cognition, the general aim of this study was to investigate the effects of psychosocial stress (and consequent cortisol increase) on spatial cognition and verbal memory in men and women. One group of 33 participants (16 males and 17 females) were exposed to the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), a procedure designed to induce mild psychosocial stress. I used 3 different means to check the effectiveness of this stress induction: salivary cortisol, self-report via the Spielberger State-Trait Anxiety Inventory, and heart-rate measurements. The measures all converged to suggest that the stress induction procedure was successful. A control group of 29 participants (15 males and 14 females) was exposed to a relaxation period rather than the TSST. Following this part of the experimental protocol, all participants completed a virtual environment spatial navigation task and a word-list learning and recall task. Results showed that, on the spatial navigation task, females and males who were not exposed to the stressor located and relocated a hidden target equally well (i.e., cognitive map-guided navigation was intact in unstressed participants). In addition, on the spatial navigation task the interaction effect of the gender and experimental condition approached statistical significance ($p = 0.085$), suggesting that females exposed to the stressor required more time to locate and relocate a hidden target than did the other participants (i.e., they were disrupted in their cognitive map-guided navigation). On the verbal memory task, participants who showed larger cortisol increases following exposure to the TSST tended to recall fewer words than did those with smaller cortisol increases, with a slightly stronger negative correlation in males than in females. The data therefore confirm that stress impacts different memory systems in different ways, and, perhaps more importantly, that sex differences play a role in moderating those effects. This is the first demonstration, within a single study, of a possible double dissociation of sex differences in cognitive performance following induction of mild psychosocial stress.
INTRODUCTION

Exposure to high levels of stress, either acutely or chronically, is associated with a range of behavioural, psychological, and physiological consequences. The most widely accepted sequelae of stress relate to hormonal and cardiovascular reactions that have a direct effect on the kidneys, pancreas, and heart (Caffo, Forresi, & Lievers, 2005; McEwen & Sapolsky, 1995; Sapolsky, 2004). Stress is also known to disrupt eating and sleeping habits, digestion, and reproduction (Kemeny, 2003). Furthermore, stress affects pain perception, influences the immune system, and increases risk of depression (McEwen, 2000). But most importantly here, stress affects memory.

Numerous studies have shown that exposure to high levels of stress can affect memory in both animals (e.g., Diamond, Park, Heman, & Rose, 1999; Sandi et al., 2005; Sapolsky, Krey, McEwen, 1986; Topic et al., 2007; Xiang, Hao, & Deng, 2006) and humans (e.g., Lupien et al., 1994; Newcomer, Craft, Hershey, Askins, & Bardgett, 1994; Newcomer et al., 1999). For instance, Lupien and colleagues (1997) subjected 14 healthy elderly subjects to a stressful task and found that, under these conditions, the participants showed significantly decreased declarative memory performance compared to participants in a non-stressful condition. Furthermore, this study successfully demonstrated that (a) stress affects memory functions that are dependent on hippocampal activity, and (b) the stress-induced release of glucocorticoids (GCs; corticosterone in rats, cortisol in humans) contributes to this effect.

The release of glucocorticoids is regulated by the action of the hypothalamic-pituitary-adrenocortical (HPA) axis. The HPA axis is a closed-loop neurocircuit controlled by a regulatory set of afferents, mostly the neurons in the paraventricular region of the hypothalamus. As the brain recognises the presence of a stressor, these neurons secrete corticotrophin-releasing hormone (CRF), which stimulates the anterior pituitary gland to release adrenocorticotropic hormone (ACTH), which in turn triggers glucocorticoid secretion from the adrenal gland. Glucocorticoid secretion then regulates the entire HPA axis by providing negative feedback to terminate subsequent CRF and ACTH release (Bowman, 2005). In humans, then, cortisol is the major mediator of the physiological effects of either psychological or environmental stress.
Neuroscientists and neuropsychologists are able to accurately describe the impact of the above-described physiological stress response on particular brain regions, and can therefore make predictions about the kinds of cognitive impairment that may develop following exposure to stress. Numerous studies make it clear that high levels of cortisol release disrupt the functioning of the hippocampus, a structure integral to the process of new learning and the formation of new memories (Squire, 1992). Intact hippocampal structure, and optimal hippocampal functioning, is required for the consolidation and the retrieval of memory (Payne & Nadel, 2004).

Current debates about whether impaired memory retrieval results directly from cortisol increase or whether it results from cortisol increase paired with hippocampal atrophy (see, e.g., see Conrad, 2006; Wright, Lightner, Harman, Meijer, & Conrad, 2006) do not concern us here because it is widely demonstrated that increase of cortisol on its own is sufficient to disrupt hippocampal processing (Het, Ramlow, & Wolf, 2005; Kim & Diamond, 2002; McEwen, 2000; Newcomer et al., 1999). This disruption likely occurs because the hippocampus (a) contains high concentrations of corticosteroid receptors, and (b) is a major influence on the HPA axis by way of negative feedback (McEwen & Sapolsky, 1995; Ruel & de Kloet, 1995; Wright et al., 2006).

Extensive research, primarily in rats, has shown that circulating glucocorticoids readily cross the blood-brain barrier and alter the functioning of the hippocampal neurons (Payne & Nadel, 2004; Squire, 1992). This effect is due to differential activation of two types of corticosteroid receptors, that is, mineralocorticoid receptors (MR) and glucocorticoid receptors (GR). Studies with humans have shown similar effects of cortisol on memory, also demonstrating that glucocorticoids affect hippocampally mediated learning, thus suggesting that GR activation in the hippocampus represents the underlying effects of cortisol on memory in humans (Abercombie, Kalin, Thurow, Rosenkranz, & Davidson, 2003).

In hormonal terms, then, cortisol is the main mediator of the physiological effects of stress; in neuronal terms, the hippocampus is the major structure involved in mediating the relation between physiological stress and cognition. Indeed, the hippocampus becomes initially involved in this process by sending neuronal projections to the hypothalamus to initiate the cascade of cortisol release; it also terminates stress responses via glucocorticoid-mediated negative feedback of the HPA axis (Corcoran et al., 2001). In the process, it runs the risk of
being affected in its function because it contains heavy concentrations of corticosteroid receptors and is therefore a major target organ for cortisol action in the brain.

In laboratory settings, stress is generally induced either directly, by exposing subjects to stress hormones (e.g., oral cortisol intake), or indirectly, by exposing subjects to a psychological manipulation (e.g., public speaking). Both techniques have been demonstrated to reliably increase cortisol levels, to be detrimental to the functioning of the hippocampus, and to therefore result in pronounced memory deficits (DeQuervain et al., 2003; DeQuervain, Roozendaal, Nitsch, McGaugh, & Hock, 2000; Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Newcomer et al., 1994, 1999; Payne, Nadel, Allen, Thomas, & Jacobs, 2002).

Not all memory systems are equally affected by increased cortisol, however. For example, semantic memory systems, which have no direct link to hippocampal functioning, seem to be unchanged by increase of cortisol (Battaglia, Sutherland, & McNaughton, 2004). In contrast, studies on the episodic form of declarative memory show evidence that acutely elevated glucocorticoid levels impair retrieval processes (DeQuervain et al., 2003; Newcomer et al., 1994, 1999). Furthermore, certain forms of spatial memory have underlying neural substrates that are profoundly affected by stress (Luine, Villages, Martinex, & McEwen, 1994).

Researchers who study spatial memory have identified the fact that, in their day-to-day experiences, humans use at least two kinds of navigation. One kind involves following a familiar route, where the person performs the task almost unconsciously (e.g., driving from work to home every day). This form of navigation is typically called route following or landmark-guided navigation. The other kind of navigation is a deliberate, consciously controlled process that may depend on knowing or inferring the global spatial relations among various locations in an environment (e.g., when a person has to find a new place or a new route to a destination). This form of navigation is typically called wayfinding or cognitive map-guided navigation (Maguire, Burgess, & O'Keefe, 1999; O'Keefe & Nadel, 1978).

Navigation based on route following is thought to rely on route knowledge (i.e., the knowledge of places or landmarks and the routes that connect them). Route knowledge, then, can be conceived of as a sequence of view-based (egocentric) visual images of landmarks.
together with directions. When exploring an environment from ground level without the help of a map, the type of navigation information that most people first acquire is route knowledge (Thorndyke & Hayes-Roth, 1982).

In contrast, navigation based on wayfinding or cognitive mapping is thought to rely on survey knowledge (i.e., an understanding of the spatial relationships between locations within an environment). Survey representations provide an overview of the spatial layout, based on an extrinsic frame of reference. In general, the acquisition of survey knowledge appears desirable for successful and flexible orientation in an environment (Cornell & Heth, 2000; Munzer, Zimmer, Schwalm, Baus, & Aslan, 2006).

Wayfinding and route following are not only distinguishable in terms of the kinds of knowledge on which they are based; they are also neurally distinguishable. Studies involving rodents, and more recently humans, have shown that wayfinding and route following involve different forms of representation with corresponding distinct neural bases (Morris, Garrud, Rawlins, & O'Keefe, 1982; O'Keefe & Nadel, 1978). Arising from the discovery of location-specific firing of place cells in the rodent hippocampus (O'Keefe & Dostrovsky, 1971), cognitive mapping theory posits that a fundamental function of the hippocampus is the construction and maintenance of spatial maps of the environment (i.e., the hippocampus has a special role in wayfinding; Maguire et al., 1999; O'Keefe & Nadel, 1978). A recent neuroimaging study by Kumaran and Maguire (2005) replicated earlier findings (e.g., Astur, Taylor, Mamelak, Philpott, & Sutherland, 2002; Thomas, 2003; Worsley et al., 2001) suggesting that the right hemisphere hippocampus has a bias towards processing spatial relationships and has a special role in mapping large-scale space. Those authors also, however, argued that the hippocampus is not simply involved in amodal relational processing (e.g., Eichenbaum, 2000).

In their study, Kumaran and Maguire (2005) compared patterns of brain activation while their subjects (18 healthy right-handed individuals) performed two tasks placing similar demands on relational processing: navigation within either a spatial domain (their city) or a non-spatial domain (their social network). They showed that execution of these two complex tasks resulted in very different patterns of brain activation; specifically, the hippocampus was only engaged by relational processing in a spatial (city), but not in a non-spatial (social), domain. Their results were consistent with previous neuroimaging studies of spatial navigation (e.g.,
Roche, Mangaoang, Commins, & O'Mara, 2005) and provide support for the theory that the hippocampus is preferentially engaged during spatial mapping tasks.

Whereas wayfinding preferentially engages the hippocampus, route-following is more dependent on the caudate nucleus (McNamara & Shelton, 2001). A recent neuroimaging study by Bohbot, Iaria, and Petrides (2004) demonstrated that individuals who undertook a virtual environment navigation task showed hippocampal activity correlating positively with wayfinding accuracy. Furthermore, their results showed that adoption of a non-spatial strategy involved sustained activation of the caudate nucleus. Similarly, Hartley, Maguire, Spiers, and Burgess (2003) used functional MRI and virtual reality navigation tasks to confirm that humans show distinct patterns of neural activation when engaging in different forms of navigation. Specifically, their findings supported the notion that the hippocampus is especially involved in accurate navigation via new routes (i.e., cognitive mapping), whereas navigation that follows well-learned routes activates the head of the right caudate nucleus.

In summary, we know that stress leads to the release of cortisol, and that in turn cortisol targets the hippocampus. We also know that not all memory systems engage the hippocampus during their processing. For instance, in the spatial memory domain, wayfinding engages the hippocampus but route following does not. Therefore, under stressful conditions, hippocampal-dependent memory tasks such as cognitive map-guided navigation (wayfinding) are much more likely to be negatively affected than are tasks based on landmark-guided navigation (route following).

The Impact of Acute Stress on Hippocampal-Dependent Forms of Memory

Of interest in the current study is the impact of acute stress on hippocampal-dependent forms of memory. As noted above, many studies have established the impact of stress on memory via hippocampal mechanisms, and many studies have established the fact that the hippocampus is critical for spatial forms of memory, but we know of only two that have examined the impact of stress on spatial memory. First, Schwabe et al. (2007) designed a spatial learning task that allowed the differentiation of spatial from stimulus-response learning strategies during acquisition. Participants (88 male and female students) had to locate a ‘win card’ out of four placed at a fixed location in a 3D model of a room. Psychosocial stress was induced with the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993). The learning strategy used was derived from the actual performance
of the participants as well as their verbal report. Findings from this study suggest that, in completing this task, participants under stress will likely use dorsal striatum-based memory systems (i.e., 'habit' learning strategies) rather than medial temporal lobe-based memory systems (i.e., flexible cognitive map-based learning strategies).

Second, Thomas, Laurance, Nadel, & Jacobs (2007), using a small-scale desktop virtual reality navigation task, demonstrated that an acute social stressor (the TSST) disrupted wayfinding, but did not disrupt route following. More specifically, they found that (a) exposure to the stressor did not detectably affect the time participants required to navigate from a series of novel starting positions toward a target that was easily visible (i.e., landmark-guided navigation was unaffected by stress), and (b) participants not exposed to the stressor successfully navigated, over several trials, from a series of novel starting positions to a target that was hidden from view but always located in the same place (i.e., cognitive map-guided navigation was intact in unstressed participants). The latter researchers also found interesting sex differences with regard to the effects of stress on wayfinding. Specifically, females exposed to the stressor required more time to locate and relocate the hidden target than did the other participants (i.e., cognitive-map guided navigation was impaired by stress in females only). In summary, Thomas et al. (2007) found a marked sex difference, where the acute social stressor disrupted cognitive map-based spatial navigation in females but not in males; the same stressor had no detectable effect on landmark-guided navigation in either females or males.

Sex Differences in Cognitive Performance

Although there is much debate and uncertainty in the literature, empirical data suggest there are slight differences in mental ability across sexes, with men generally performing better on visuospatial tasks and women generally performing better on tests of verbal usage (Hyde, Fennema, & Lamon, 1990; Johnson & Bouchard, 2005, 2007). With regard to the biological mechanisms underlying these sex differences, a growing number of studies demonstrate that the sex-dependent effects in human cognitive and motor skills may be due, at least in part, to organisational or activational oestrogenic effects of sex hormones on the brain (Hampson, Finestone & Levy, 2005; Kimura, 2004; Kimura & Hampson, 1994).

In a study investigating hormone-mediated changes in cognitive performance, Hampson (1990) showed that in the menstrual phase of their cycle, women's performance on non-
verbal/spatial tests differed from that of women in the midluteal phase (when oestrogen is higher).\(^2\) Comparing the performances of 45 women, Hampson demonstrated that variations in gonadal steroid levels across the human menstrual cycle are sufficient to exert small but consistent effects on cognitive and motor performance. Specifically, she showed improved verbal-articulatory, verbal fluency, and fine motor skills in the midluteal phase. The opposing effect was found on the tests of spatial ability and abstract reasoning, where women at the midluteal phase performed significantly worse than women at the menstrual phase.

Most importantly for the purposes of the current research, it is becoming increasingly clear from animal and human studies that, on average, females and males respond differently to stressors. For instance, Bowman (2005) reviewed sex differences in response to stress on a variety of spatial tasks over the lifespan of rats. He found that, in general, female rats are more resistant to stress-induced impairment on spatial tasks than male. Studies using human participants show similar results. For example, exposure to a stressor increases cortisol levels in both sexes, but in men stress exposure facilitates fear conditioning whereas in women stress seems to inhibit fear conditioning (Jackson, Payne, Nadel, & Jacobs, 2005). Furthermore, in a study where students were exposed to the TSST, females showed more of a heart rate increase during the stress exposure than did males (Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004).

Looking more specifically at the sex-dependent impact of stress, recent studies have found marked inter- and intra-sex differences in hormonal response patterns to HPA axis activation. In general, the data show that premenopausal women have a significantly larger cortisol response in the luteal phase than in the follicular phase of the menstrual cycle (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999; Kudielka, Buske-Kirschbaum, et al., 2004). Furthermore, in a study examining the relationship between stress-induced cortisol levels and verbal memory in men and women, Wolf, Schommer, Hellhammer, McEwen, and Kirschbaum (2001) found that, for women in the luteal phase compared to men in general, performance on a word-recall task was less sensitive to the disruptive effects of a social stress-induced cortisol increase. The results of these studies and others like it imply that phase of the menstrual cycle may be an important control factor in studies of cognitive performance under stressful conditions.\(^3\)
Rationale for the Present Study and Specific Aims

Understanding that the hippocampus is both affected by cortisol and intimately involved in episodic memory and spatial cognition, the general aim of my study is to investigate the consequences of cortisol increase on verbal and spatial memory in men and women. To my knowledge, this is the first study to simultaneously examine both forms of cognitive performance under acute stress.

More specifically, I aim to systematically replicate the study of Thomas et al. (2007), while addressing several limitations of that study to allow for more clear interpretations of effects. For instance, the previous study had a relatively small sample size ($n = 29$) and did not assess physiological and hormonal levels of participants. I have therefore used a larger sample, controlled for hormonal differences across the menstrual cycle, and (following general procedures outlined by Schwabe and colleagues (2007)) measured heart rate, cortisol levels, and self-reported anxiety at each step of the experimental procedure.

Furthermore, Thomas et al. (2007) focused their study only on spatial cognition. With regards to the impact of stress on verbal learning and memory, Wolf and colleagues (2001) reported a sex-related difference of gender with respect to cortisol increase. That is, participants who showed a larger cortisol response following exposure to an acute stressor recalled fewer previously-learned words than did subjects who showed a small cortisol response. Importantly, this association was solely driven by the results of the strong association observed in men, while there was no such association in women. That is, women who showed a cortisol increase did not recall fewer words, but men who showed such an increase did recall fewer words. These findings are particularly interesting given that the pattern of stress-induced differences in performance across sex is diametrically opposite to those reported by Thomas et al. (2007). To provide clarification on this issue, I will replicate part of Wolf et al.'s (2001) design, using a similar stress-induction procedure and a similar verbal memory test.

Hypotheses

My hypotheses are therefore as follows:

1. There will be no differences between stressed and non-stressed participants on a landmark-guided (non-hippocampally dependent) task.
2. Women exposed to an acute stressor will perform worse than all other participants (stressed males, non-stressed males and non-stressed females) on a task assessing cognitive map-guided navigational ability (i.e., female wayfinding abilities will be impaired by stress).

3. Males (specifically, a male subgroup showing highest levels of cortisol increase in response to an acute stressor) will perform more poorly on a free recall verbal memory task than will females exposed to the same stressor. Moreover, the female subgroup showing highest levels of cortisol increase will perform no differently than the female subgroup showing lowest levels of cortisol increase.
METHOD

Design
This study is cross-sectional in design. It compares specific cognitive abilities (spatial and verbal memory) in two groups of subjects, one of which is exposed to a social stressor and the other which is not. Additionally, within each group there are roughly equal numbers of males and females because, as noted in the Literature Review, sex differences are important to us both in terms of stressor effects and in terms of cognitive performance. In essence, there are two independent variables (sex and stress manipulation) and two major classes of dependent variables (spatial cognition measures and verbal memory measures).

Participants
Sixty-seven undergraduate students (34 female, 33 male) from the University of Cape Town were initially enrolled into the study. They were recruited from undergraduate psychology classes, and participated in exchange for course credit.

Women who put their names on the experiment sign-up sheets were contacted first. They were enrolled in the study if they were not taking any oral contraceptives and if they reported a regular (30 day) menstrual cycle. If the female participant remembered the exact dates of her previous menstrual cycle, she was given an appointment for 21-25 days from the end of her last period (i.e., so that she would be in the late luteal phase of the menstrual cycle when participating in the experimental procedures). The late luteal phase was chosen because, during this phase, stress-induced free cortisol levels do not differ between men and women (Kirschbaum, Pirke, & Hellhammer, 1995; Kirschbaum, Wust, & Hellhammer, 1993; Kirschbaum et al., 1999). If the female participant did not remember the exact dates of her menstrual cycle, she was asked to contact the experimenter on the first day of her next period, and an appointment was then set up in a similar way as described above. Menstrual cycle phase was checked post-experiment by participant self-report.

Each male participant in the study was yoked to a particular female participant. That is to say, when (following the procedure above) an appointment for the experiment had been set up with a female participant, a potential male participant was immediately contacted and asked to schedule an appointment for the same day. In this way we avoided any sex differences that
might have arisen from the delay between signing up for the study and actually being run through the experimental procedures.

Participants were pseudo-randomly assigned to either the Stress group (i.e., they were exposed to the TSST) or the Control group (i.e., they were not exposed to the TSST) to ensure there were approximately equal numbers of males and females in each group. For example, if the first pair of yoked male-female participants was assigned to the Stress group, the next pair would be assigned to the Control group, and so on. I excluded five participants from the final data analysis because their Beck Depression Inventory-II scores were ≥ 29. This left a final sample of 62 participants: Stress group \( n = 33 \) (17 females and 16 males); Control group \( n = 29 \) (14 females and 15 males).

Measures and Instruments

**Depression Screening Measure**

The Beck Depression Inventory-II (BDI-II; Beck, Steer, & Brown, 1996) is a 21-item test presented in multiple-choice format. It measures the presence, and degree, of depression in adolescents and adults. Each item consists of four statements that correspond to ratings from 0 to 3, with higher ratings indicating characteristics of more severe depression.

The instrument has strong psychometric properties. For instance, BDI-II scores correlate positively with Hamilton Depression Rating Scale scores (Pearson \( r = 0.71 \)). The BDI-II shows a high 1-week test-retest reliability (Pearson \( r = 0.93 \)), suggesting that it is not overly sensitive to daily variations in mood. Furthermore, it has high internal consistency (alpha = 0.91). The BDI-II is regularly used in South African clinical practice and research studies (see, e.g., Ward, Flisher, Zissis, Muller, & Lombard, 2001).

**Self-Reported Anxiety**

The Spielberger State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) consists of two 20-item self-report scales that, respectively, measure in-the-moment and characteristic anxiety. The 20-item State scale requires the respondent to describe the intensity of his/her feelings of anxiety at the current time. The scale is psychometrically sound in that it has a high degree of internal consistency (Spielberger & Vagg, 1984). The 20-item Trait scale requires the respondent to describe how he/she generally feels in terms of the frequency with which specific symptoms of anxiety are
experienced. Psychometric studies indicate that the scale has a high degree of internal consistency, as well as high test-retest reliability (Spielberger & Vagg, 1984). The STAI is also regularly used in South African clinical practice and research studies (see, e.g., Rieckert & Moller, 2000; Spangenberg & Campbell, 1999; Van Wijk, 1998).

**Physiological Measures**

Saliva samples from which we eventually derived cortisol measures were collected using Salimetrics Eyespear Sorbettes (Salimetrics LLC, Pennsylvania, USA). In a comparison study of three saliva collection methods (passive, salivettes and eyespears), the eyespear was shown to produce less reduction in concentration of cortisol. Additionally, eyespears offer methodological advantages for collecting saliva and have garnered positive ratings for their comfort and acceptability to research participants (Strazdins et al., 2005).

Participants in the current study were instructed to place the cotton-cellulose eyespear under their tongues for 1 minute. After removal, the sorbette was placed into an individual conical tube cap and immediately stored in our laboratory’s freezer until transport in insulated pack to an accredited laboratory for cortisol analysis.

A heart rate monitor (Polar S725x, Polar Electro Oy, Finland) was fitted onto the participant for the duration of the study. The device used is similar to those used by cyclists, long-distance runners, and other endurance athletes. Thus, it allowed participants to be mobile, which was an important consideration for this study given that the stressor manipulation took place in a different physical location from the cognitive measures.

**The Acute Social Stressor: The Trier Social Stress Test (TSST)**

I induced psychosocial stress in one group of participants by using the TSST. (For a complete description of the TSST and its development, see Kirschbaum et al., 1993). The TSST is widely applied in psychological research and has been found to produce levels of stress equal to that found after mild to moderate medical or surgical stress (Newcomer et al., 1999).

Although not all psychological stressors result in increased cortisol (Biondi & Picardi, 1999), a large number of studies have reported that laboratory tasks such as public speaking or mental arithmetic can increase cortisol levels (e.g., Het, Ramlow, & Wolf, 2005; Het & Wolf, 2007; Kudielka, Buske-Kirschbaum, et al., 2004; Kuhlman et al., 2005). Furthermore, a meta-
analysis reviewing laboratory studies of acute psychological stressors and tests of conditions capable of eliciting cortisol response concluded that tasks containing both uncontrollable and social-evaluative elements were associated with the largest cortisol changes (Dickerson & Kemeny, 2004). The same review found that the TSST produced an adequate increase of cortisol levels, probably because the task includes the three factors most closely associated with triggering those increased levels: (a) it is a motivated performance task, (b) there is relative uncontrollability of task outcome, and (c) there is the presence of social evaluation. Compared to other laboratory-based stress induction tasks, the TSST provoked the most robust physiological stress. Dickerson and Kemeny (2004) also found that the most effective psychosocial stressor combination was cognitive tasks (e.g., mental arithmetic) and verbal interaction tasks (e.g., public speaking), which the TSST possesses.

The version of the TSST used in this study was slightly modified from the original version described by Kirschbaum et al. (1993). For instance, I used only one room that had the desk of the researcher on one side and a two-way mirror with a stage set up on the other. The researcher, dressed in a white laboratory coat, faced the back of the participant. The participant, with his/her back to the researcher, faced a camera, a microphone, two lamps on tripods, and what he/she thought was a one-way mirror. Other than these two lamps the room was kept dark. The participant was told that in the room behind the “one-way mirror” was a psychologist who was a behavioural health expert; this person, the participant was told, would study verbal and nonverbal behaviour and, with the help of the video being produced, would later be able to produce a complete analysis.

In a manner similar to the original TSST, the participant was read a set of standard instructions designed to introduce him/her to the task of the TSST. He/she was asked to assume the role of a job candidate for the job of their choice and given 10 minutes to prepare a speech detailing his/her suitability for that job. After that 10-minute preparation period, the participant was told that the speech was to be delivered extemporaneously. Thereafter, each of the Stress group participants was given 5 minutes to present the speech. If the participant stopped speaking before time was up, the researcher said, “You still have time left, please continue.” If the participant was unable to continue delivering the speech, this set of standard questions was asked: 1. “Please tell us what are some of your weaknesses”; 2. “What is the most difficult experience that you have had that would help you on the job?”; 3: “For what reasons should we not take you?”
After completion of the entire 5-minute speech-delivery and questioning period, the participants were asked to perform a serial subtraction task (i.e., “Starting at 1022, keep subtracting 13 until I tell you to stop”). Each incorrect subtraction required the participant to start again at 1022. This mental arithmetic task lasted a full 5 minutes.

The Spatial Navigation Task: Computer-Generated Arena

The Computer-Generated Arena (CG Arena) is a desktop-based, non-immersive virtual environment (VE) spatial navigation task that is a human analogue of the Morris Water Maze (MWM; Morris, 1984). The CG Arena was developed in order to satisfy the need for a relatively ‘pure’ measure of hippocampal functioning and spatial cognition in humans (Jacobs, Laurance, & Thomas, 1997; Jacobs, Thomas, Laurance, & Nadel, 1998; Thomas et al., 2007).

In VE spatial navigation tasks such as the CG Arena, individuals use representations of distal cues, and the multiple spatial relations between them, to form a cognitive map of the virtual space. This map can then be used to relocate specific places within the space (Burgess, Maguire, & O’Keefe, 2002; Maguire et al., 1999; Sandstrom, Kaufman & Huettel, 1998). The use of VE spatial navigation tasks allows researchers to conduct empirical tests of spatial cognition theory without incurring the costs associated with the construction of real-world analogs of tasks such as the MWM. It has been found that (1) after learning in a VE, humans can make accurate judgements about metrics in real space, (2) there is a good transfer of spatial information from virtual to real environments, and (3) this technology can assist in investigating individual differences in spatial abilities (Astur et al., 2002; Loomis, DaSilva, Fujita, & Fukusima, 1992; Loomis, Lippa, Klatzky, & Golledge, 2002; Thomas, 2003; Worsley et al., 2001). In short, investigating human spatial cognition and behaviour has been made easier by the development of VE tasks such as the CG Arena. Furthermore, the data from virtual reality maze navigational tasks have contributed to our understanding of the neural pathways of these cognitive systems (Roche et al., 2005).

With specific regard to the CG Arena, it has been shown across numerous studies to be a reliable and valid measure of different forms of spatial navigation in humans (e.g., Jacobs et al., 1997, 1998; Thomas, Hsu, Laurance, Nadel, Jacobs, 2001). The task has been used in a variety of research studies featuring populations as diverse as older adults (Laurance et al.,
2002; Thomas, Laurance, Luczak, & Jacobs, 1999), children diagnosed with autism spectrum disorders (Daniels, Malcolm-Smith, & Thomas, 2007; Edgin & Pennington, 2005), traumatic brain injury patients (Skelton, Bukach, Laurance, Thomas, & Jacobs, 2000), and anterior temporal lobectomy patients (Frakey, Shrikisoon, Thomas, Jacobs, & Bauer, 2005; Thomas, 2003).

The CO Arena is presented on a desktop personal computer and monitor by custom-designed software. The screen display shows, from a first-person perspective, a multicoloured view of a circular arena contained within one of two square rooms (a “waiting room” and an “experimental room”).

Each participant received standard verbal instructions on how to move in the VE. These instructions prepared the participant for the VE display, directed him/her about how to move in the VE (e.g., “Pushing the joystick left or right will turn you in the corresponding direction, but will not move you sideways”), and told him/her how to move from one room to another (e.g., “While you are standing on the target, you can transport yourself to the waiting room by pressing the space bar”).

When the participant signalled his/her readiness to start the task, the experimenter introduced him/her to the waiting room. This large square room featured four textureless walls, each of a different solid colour (blue, green, red and yellow), surrounding an arena that was defined by a circular wall with a marble texture. The ceiling of the room was light gray and the floor dark gray. The purpose of the waiting room is, first, to allow the participant to practice using the joystick to move in VE, and, second, to offer the participant the opportunity to rest between experimental room trials.

When the participant was ready to leave the waiting room, he/she pressed the space bar. This changed the display from the waiting room to the experimental room. The dimensions and overall features of the experimental room were identical to those of the waiting room (e.g., the ceiling was light gray and the floor dark gray). Figure 1 shows four views within the experimental room. As can be seen, the background to each wall is gray, but each of the four walls displayed a different set of pictures. One wall showed photographs of a sleeping cat, a door, and a flowering cactus. Across from that wall was a wall showing photographs of a flower, a sunset, and a stone idol. A third wall was covered with black stripes and featured a
photograph of an inactive volcano at its centre. Across from the latter was a wall textured with small boxes and featuring a photograph of a group of hoodoos at its centre.

![Figure 1. The CG Experimental Room](image)

In the experimental room, the participant had the task of attempting to locate a target (a large blue square) on the floor of the room as quickly as possible before 120 seconds elapsed. To ensure that participants understood this task and could efficiently operate the joystick within the VE, a series of 4 experimental room trials, each featuring a visible target, was initially presented. The visible target could easily be seen by the participant following a rudimentary scan of the environment. The task then was simply to move to the target and to stand on it. The target was in a different location on each of these trials, and the participant began each trial from a different start point on the circumference of the arena.

When the participant reached the target, and as long as he/she stood on it, a computer-generated click sounded. While on the target, the participant could turn around and move toward its edges, but could not move outside its bounds. After approximately 8 seconds on the target, the trial ended and the participant was moved back into the waiting room.

Following the 4 visible target trials, the participant was told that he/she would now attempt 6 hidden target trials. These trials were formally identical to the visible target trials, with the crucial exceptions that (a) the target was initially invisible to the participant (i.e., its colour blended with that of the surrounding arena floor), and (b) the target was always in the same
place. Only when the participant moved onto the location of this target did the target icon (the blue square) become visible and the computer-generated clicking sound, signalling a successful search. Again, the participant began each of these trials at a different start point on the circumference of the arena.

Unbeknownst to the participant, the final (sixth) hidden target trial was a ‘probe’ trial on which the previously hidden target had been entirely removed from the experimental room. This trial was used to establish whether, and for how long, the participant would persist in searching for the target in its former location (see Morris, 1984, and Jacobs et al., 1997, for further explanation and illustrations of the value of probe trials).

After completion of the VE spatial navigation task, the participant was administered two pencil-and-paper companion tasks to the CG Arena: The Object Recognition Test (ORT) and the Arena Reconstitution Task (ART). These tasks provide measurements of spatial and non-spatial learning and memory that are independent of the CG Arena and the data collected from it.

**OBJECT RECOGNITION TASK (ORT)**

1  2  3  4
5  6  7  8
9 10 11 12
13 14 15 16

*Figure 2. ORT Stimulus Card*

The ORT tested the capacity of the participants to recognize the photographs that were on the walls of the experimental room. The participant was presented with an A4-size laminated sheet on which 16 numbered pictures were presented in a four-by-four array (see Figure 2).
Eight of the items were in the experimental room, and 8 were distractors. The participant was asked to indicate, by circling either 'yes' or 'no' on an answer sheet, whether each item was in the experimental room.

The ART, which was administered immediately following the ORT, required the participant to reconstruct the spatial layout of the experimental room. The participant was given a stimulus sheet similar to that shown in Figure 3, as well as 8 small pieces of laminated cardboard, each bearing a representation of a photograph from the experimental room. The participant was told that the stimulus sheet was a top-down representation of the experimental room, and was then asked to place each piece of cardboard in the appropriate space on the sheet. Finally, the participant was asked to indicate (by marking an X on the sheet) in which of the four squares the hidden target had been located.

![ARENA RECONSTITUTION TASK (ART)](image)

*Figure 3. ART Stimulus Sheet*

Whereas the ORT is a measure of pure (non-spatial) recognition memory, the ART is a measure of cognitive mapping ability. Previous studies have demonstrated that the ART provides data congruent with the data gathered from the CG Arena itself, whereas performance on the ORT is comparatively independent of that on spatial navigation task (Skelton et al., 2000; Thomas, 2003; Thomas et al., 2001).
The Verbal Memory Task

A list of 25 words (see Appendix A) was presented to the participants on a piece of paper, with the instruction to learn the words by reading them aloud at a speed of one word every 3 seconds. The words were selected from a pool of English words generated by software located at http://www.random.org/lists. Initially, a pool of 90 words was generated after setting criteria of high concreteness (more than 6 on the scale) and high meaningfulness (higher than 5 on the scale). An additional criterion was the word could have no more than 3 syllables. These criteria are identical to those used by Wolf et al. (2001) in their word-list construction.

After the learning phase of this task, a 25-second distractor task was presented (participants were instructed to read aloud the names of colours that were printed on a sheet of paper). This task abolished the possibility that the participant could use subvocal rehearsal strategies to remember the word list. Immediately after the distractor task, free recall of the word list was tested.

Procedure

All study procedures were approved by the Ethics Committee of the UCT Department of Psychology.

Following conventions established by recent studies (e.g., Schwabe et al., 2007), all participants were tested between 14h00 and 17h00. In studies featuring cortisol as a dependent measure, the time of day when participants are tested is a crucial variable: There is a large body of evidence showing that the magnitude of HPA axis response to pharmacological provocation varies according to time of day, with larger cortisol responses in the afternoon and evening compared to the morning hours (see, e.g., DeCherney et al., 1985). This variation mirrors normal HPA axis activity, which follows a pronounced circadian rhythm with highest hormone levels in the early morning hours and continuously decreasing levels over the course of the day (see, e.g., Kirschbaum & Hellhammer, 1994). However, although it is well accepted that the usually high levels of cortisol in the mornings will result in smaller endocrine response to different pharmacological provocations, a recent re-analysis of five independent studies showed evidence to the contrary with regards to stimulations following social stress (Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004).
In a reminder phone call the day before their appointments, participants were reminded to, during the 2 hours prior to the appointment, refrain from smoking, chewing gum, exercising, and drinking fizzy drinks, tea, or coffee. Upon arrival in the laboratory, participants were given a consent form (see Appendix B) to read and sign. At time of enrolment they had been given a brief explanation of the requirement of the study; the consent form provided more details of a procedural nature, and also listed their rights as research participants.

Once the participant had read and signed the consent form, he/she was fitted with the heart rate measurement device. The participant was then instructed to fill out the BDI-II and the STAI. A saliva sample and a heart rate measure were then taken. Participants in the Stress group were then administered the TSST, as described above. Participants in the Control group were not administered any part of these TSST procedures. Instead, they were seated in a comfortable chair, given a set of (non-political and non-factual) magazines to read, and relaxing music was played over the laboratory speakers. These Control group participants were told they should simply relax for the next 20 minutes. No audience was present in the room with them, and no video recordings were made during their relaxation period.

After the completion of the Stress group’s TSST exposure and the Control group’s relaxation period, saliva samples and heart rate measurements were again taken from all participants, and all were instructed to complete the STAI State scale for a second time. The CG Arena (and accompanying ORT and ART) and verbal memory tests were then administered. In order to guard against sequence effects of the tests, we counterbalanced the administration: half of the participants in the Stress group and half the participants in the Control group were administered the spatial tests followed by the verbal tests, while the remaining participants in both groups were administered the tests in the opposite order.

Finally, a third saliva sample and heart rate measurement was taken from each participant, and each was instructed to complete the STAI State scale for a third time. All participants were fully debriefed before they left the laboratory, and the experimenter specifically ensured that no participant in the Stress was experiencing any distress due to the experimental procedures. Table 1 presents a timeline for experimental events, and Figure 4 is a flowchart summarizing the experimental protocol.
<table>
<thead>
<tr>
<th>Time (mins) from experiment start</th>
<th>Event: Control group</th>
<th>Event: Stress group</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.00</td>
<td>Read and sign consent form; complete BDI, STAI</td>
<td>Read and sign consent form; complete BDI, STAI</td>
</tr>
<tr>
<td>10.00</td>
<td>First heart rate measure and saliva sample</td>
<td>First heart rate measure and saliva sample</td>
</tr>
<tr>
<td>20.00</td>
<td>Relaxation period instructions</td>
<td>TSST: Instructions</td>
</tr>
<tr>
<td>25.00</td>
<td>Relaxation</td>
<td>TSST: Begin 10-min speech preparation</td>
</tr>
<tr>
<td>35.00</td>
<td>Relaxation</td>
<td>TSST: Begin 5-min speech presentation</td>
</tr>
<tr>
<td>40.00</td>
<td>Relaxation</td>
<td>TSST: Begin mental arithmetic task</td>
</tr>
<tr>
<td>45.00</td>
<td>Relaxation</td>
<td>Relaxation</td>
</tr>
<tr>
<td>50.00</td>
<td>Second heart rate measure and saliva sample; complete STAI – State</td>
<td>Second heart rate measure and saliva sample; complete STAI – State</td>
</tr>
<tr>
<td>55.00</td>
<td>Begin cognitive testing</td>
<td>Begin cognitive testing</td>
</tr>
<tr>
<td>90.00</td>
<td>Begin short relaxation period</td>
<td>Begin short relaxation period</td>
</tr>
<tr>
<td>95.00</td>
<td>Third heart rate measure and saliva sample; complete STAI – State</td>
<td>Third heart rate measure and saliva sample; complete STAI – State</td>
</tr>
<tr>
<td>100.00</td>
<td>Debriefing</td>
<td>Debriefing</td>
</tr>
</tbody>
</table>
and Signal Absent distribution. Values of $d'$ that are near zero indicate chance performance. (See http://wise.cgu.edu/sdtmod/signal_applet.asp for more details.)

To score the ART, I counted the distance from the participant’s location of a particular picture icon to the actual location of that icon. For example, if the participant placed an icon directly east of the target location when its actual location was two spaces away (to either the north or the south), then a score of 2 was awarded. Each of the 8 picture icons was scored in that way, meaning that higher scores indicated poorer performance and a score of zero indicated perfect reconstitution of the spatial layout of the experimental room.

To score the verbal memory task, I simply counted the number of words the participant correctly remembered during free recall.

All statistical analyses were performed using the software packages Statistica version 7 (StatSoft, 2004) and SPSS version 15.0 (SPSS Inc., Chicago IL). I used $a = 0.05$ as the threshold for statistical significance. Details of each individual analysis are given within the Results section.
RESULTS

Measures of Stress

These measurements provided a check of the TSST stress induction procedure.

Table 2. Measures of Stress

<table>
<thead>
<tr>
<th></th>
<th>STRESS</th>
<th></th>
<th>CONTROL</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
<td>Female</td>
<td></td>
</tr>
<tr>
<td></td>
<td>n=16</td>
<td>n=17</td>
<td>n=15</td>
<td>n=14</td>
<td></td>
</tr>
<tr>
<td>STAI Trait</td>
<td>44.94 (8.70)</td>
<td>39.47 (9.65)</td>
<td>38.20 (9.92)</td>
<td>44.21 (11.33)</td>
<td></td>
</tr>
<tr>
<td>STAI State - baseline</td>
<td>44.94 (8.24)</td>
<td>39.71 (12.80)</td>
<td>34.27 (7.31)</td>
<td>37.93 (9.01)</td>
<td></td>
</tr>
<tr>
<td>STAI State - post-manipulation</td>
<td>48.06 (8.91)</td>
<td>44.05 (14.77)</td>
<td>27.93 (4.42)</td>
<td>32.21 (7.28)</td>
<td></td>
</tr>
<tr>
<td>STAI State - end</td>
<td>37.13 (6.73)</td>
<td>38.06 (13.64)</td>
<td>35.07 (9.40)</td>
<td>34.42 (7.73)</td>
<td></td>
</tr>
<tr>
<td>Heart Rate - baseline</td>
<td>73.23 (28.13)</td>
<td>80.00 (11.33)</td>
<td>83.47 (10.56)</td>
<td>86.69 (12.95)</td>
<td></td>
</tr>
<tr>
<td>Heart Rate - post-manipulation</td>
<td>85.15 (32.94)</td>
<td>82.36 (16.10)</td>
<td>74.47 (11.03)</td>
<td>78.85 (11.94)</td>
<td></td>
</tr>
<tr>
<td>Heart Rate - end</td>
<td>71.69 (26.98)</td>
<td>77.00 (11.17)</td>
<td>76.87 (13.77)</td>
<td>81.71 (10.70)</td>
<td></td>
</tr>
<tr>
<td>Cortisol - baseline</td>
<td>06.19 (2.96)</td>
<td>05.70 (2.64)</td>
<td>04.75 (4.21)</td>
<td>05.77 (4.19)</td>
<td></td>
</tr>
<tr>
<td>Cortisol - post-manipulation</td>
<td>09.38 (6.07)</td>
<td>08.36 (7.20)</td>
<td>03.16 (2.93)</td>
<td>03.77 (01.39)</td>
<td></td>
</tr>
<tr>
<td>Cortisol - end</td>
<td>08.01 (5.05)</td>
<td>07.18 (5.79)</td>
<td>03.56 (2.79)</td>
<td>03.77 (02.74)</td>
<td></td>
</tr>
</tbody>
</table>

Note. Means are presented with standard deviations in parentheses.
*Data based on 12 participants. bData based on 14 participants. cData based on 13 participants. dData based on 16 participants. eData based on 10 participants.

Self-Reported Measures of Anxiety

Trait Anxiety. With regard to self-reported trait anxiety, participants in the Control (M = 41.10, SD = 10.87) and Stress (M = 42.12, SD = 9.47) groups were not statistically significantly different, t(60) = .394, p = .695.

Self-reported trait anxiety data for male and female participants in each group are presented in Table 2. To confirm that the participants in our sample were representative of the general population with regard to trait anxiety, I compared their scores to normative data for undergraduate students presented in the STAI test manual (Spielberger, 1983) and derived standard scores from that comparison. All of these derived z-scores were within one standard deviation of the population mean (for males, Stress group z = 0.72, Control group z = -0.01;
for females, Stress group $z = -0.09$, Control group $z = 0.38$), suggesting that the current sample was a representative group from the population.

State Anxiety. With regard to self-reported state anxiety at the beginning of the experimental protocol (i.e., before the stress manipulation), participants in the Control ($M = 36.03, SD = 8.24$) and Stress ($M = 40.30, SD = 10.68$) groups were not statistically significantly different, $r(60) = 1.74, p = .086$. This result confirms that participants entered the experiment in the same state of mind and with no perceptible differences in expectation.

As shown in Table 2 and in Figure 5, participants in the Stress group showed an increase in self-reported state anxiety from pre-TSST to post-TSST, whereas participants in the Control group showed a decrease in self-reported state anxiety from pre-relaxation to post-relaxation. A set of repeated-measures ANOVAs confirmed that both these changes were statistically significant. For the Stress group, there was a significant main effect of the TSST, ($F(1, 31) = 10.53, p = .003$), in the absence of a main effect of gender ($p = .476$) or a gender x TSST interaction ($p = .439$). For the Control group, there was a significant main effect of the relaxation period, ($F(1, 27) = 27.13, p < .001$), in the absence of a main effect of gender ($p = .109$) or a gender x relaxation interaction ($p = .791$).

*Figure 5. Self-Reported State Anxiety during the Experiment*
From an ethical standpoint, it was important for us to know that the participants departed the laboratory in the approximately the same state of mind as when they arrived. As shown in Table 2 and in Figure 5, self-reported levels of state anxiety did not appear to be different from at the end of the experimental protocol compared to the start of the session. A set of repeated-measures ANOVAs, comparing state anxiety measured at baseline to state anxiety measured at the conclusion of the experimental procedures (i.e., immediately before the participants were debriefed) confirmed that impression. For the Stress group, there was no statistically significant main effect of the experimental procedures ($p = .121$), no main effect of gender ($p = .965$), and no gender x experimental procedures interaction ($p = .531$). Similarly, for the Control group, there was no statistically significant main effect of the experimental procedures ($p = .463$), no main effect of gender ($p = .557$), and no gender x experimental procedures interaction ($p = .246$).

**Cortisol Levels**

Data from 12 participants (2 females and 2 males in the Stress group; 5 females and 3 males in the Control group) were omitted from the cortisol analysis due to insufficient saliva quantity. The cortisol data from one participant, a male in the Stress group, were lost due to experimenter error. Cortisol data for the remaining participants in each group are presented in Table 2 and in Figure 6.

With regard to free cortisol levels at the beginning of the experimental protocol (i.e., before the stress manipulation), participants in the Control ($M = 5.24 \text{ nmol/l, SD} = 4.16$) and Stress ($M = 5.93 \text{ nmol/l, SD} = 2.76$) groups were not statistically significantly different, $t(57) = 0.75, p = .454$. This result confirms that participants entered the experiment with a similar cortisol levels and with no measured differences in HPA axis activity.

In the Stress group, average free cortisol levels increased in response to the TSST from $5.93 \pm 2.76 \text{ nmol/l}$ to $8.83 \pm 3.44 \text{ nmol/l}$. For women in the Stress group, the average net cortisol increase was $1.10 \pm 3.53 \text{ nmol/1}$ (baseline: $5.70 \pm 2.64 \text{ nmol/1}$; post-stress: $8.38 \pm 7.20 \text{ nmol/1}$). For men in the Stress group, the average net cortisol increase was $3.17 \pm 4.80 \text{ nmol/1}$ (baseline: $6.19 \pm 2.97 \text{ nmol/1}$; post-stress: $9.38 \pm 6.07 \text{ nmol/1}$). Repeated-measures ANOVA showed that, for the Stress group overall, there was a significant main effect of the TSST, $(F(1, 26) = 7.33, p = .012)$, in the absence of a main effect of gender ($p = .380$) or a gender x TSST interaction ($p = .200$).
In the Control group, average free cortisol levels decreased in response to the relaxation period from $5.24 \pm 4.16$ nmol/l to $3.44 \pm 2.33$ nmol/l. For women in the Control group, the average net cortisol decrease was $2.10 \pm 2.49$ nmol/l (baseline: $5.77 \pm 4.19$ nmol/l; post-stress: $3.77 \pm 1.39$ nmol/l). For men in the Control group, the average net cortisol decrease was $0.73 \pm 2.29$ nmol/l (baseline: $4.75 \pm 4.21$ nmol/l; post-stress: $3.16 \pm 2.93$ nmol/l). A repeated-measures ANOVA showed that, for the Control group, there was a significant main effect of the relaxation period, $(F(1, 20) = 7.68, p = .012)$, in the absence of a main effect of gender $(p = .274)$ or a gender x relaxation interaction $(p = .194)$.

![Figure 6. Salivary Cortisol Levels during the Experiment](image)

These data confirm that the TSST worked as expected to raise cortisol levels in the Stress group, and that the relaxation period experienced by the Control group was effective in lowering cortisol levels.

**Cardiovascular Responses**

Due to hardware problems, no heart rate data were recorded for 6 participants (2 females and 4 males in the Stress group). Also due to hardware problems, baseline heart rate data were not recorded for 2 participants (1 female in the Stress group and 1 female in the Control group);
post-relaxation heart rate data were not recorded for 1 female in the Control group, and only baseline heart rate data were recorded for 1 female in the Stress group. Heart rate measurements for the remaining male and female participants in each group are presented in Table 2 and in Figures 7 and 8.

With regard to measured heart rate at the beginning of the experimental protocol (i.e., before the stress manipulation), participants in the Control ($M = 84.96$ beats per minute, $SD = 11.62$) and Stress ($M = 79.69$ bpm, $SD = 14.64$) groups were not statistically significantly different, $t(52) = -1.47, p = .147$. This result confirms that participants entered the experiment with no measured differences in heart rate.

![Figure 7. Participants' Heart Rates during the Experiment](image)

In the Control group, average heart rate decreased in response to the relaxation period from $84.96 \pm 11.62$ bpm to $76.50 \pm 11.47$ bpm. For women in the Control group, the average heart rate decrease was $6.92 \pm 8.16$ bpm (baseline: $86.69 \pm 12.95$ bpm; post-stress: $78.85 \pm 11.94$ bpm). For men in the Control group, the average heart rate decrease was $9.00 \pm 5.93$ bpm (baseline: $83.47 \pm 10.56$ bpm; post-stress: $74.47 \pm 11.03$ bpm). A repeated-measures ANOVA showed that, for the Control group, there was a significant main effect of the relaxation period, ($F(1, 25) = 34.47, p = .00000396$), in the absence of a main effect of gender ($p = .460$) or a gender x relaxation interaction ($p = .449$).
In the Stress group, average heart rate increased in response to the TSST from 79.69 ± 14.64 bpm to 86.92 ± 19.15 bpm. For women in the Stress group, the average heart rate increase was 2.23 ± 11.99 bpm (baseline: 80.00 ± 11.33 unit; post-stress: 82.36 ± 16.10 bpm). For men in the Stress group, the average heart rate increase was 12.92 ± 9.60 bpm (baseline: 79.33 ± 18.31 bpm; post-stress: 92.25 ± 21.68 bpm). Repeated-measures ANOVA showed that, for the Stress group overall, there was a significant main effect of the TSST, \( F(1, 25) = 12.03, p = .002 \) and a significant gender x TSST interaction, \( F(1, 25) = 5.99, p = .022 \), in the absence of a main effect of gender \( (p = .507) \). I interpret the interaction as indicating that the increase in male heart rate following the TSST was, on average, significantly higher than that in females.

**Figure 8.** Participants' Heart Rates during the Experiment, Female versus Male

**Correlations between Self-Reported and Objectively Measured Stress**

As shown in Table 3, there were statistically significant correlations between baseline and post-manipulation measures across all three indexes of stress. More meaningful, however (particularly because the salivary cortisol measurement is regarded within the literature as a reliable index of stress), is the fact that there were significant correlations between post-stress...
cortisol levels and post-stress state anxiety and post-stress heart rate. These data suggest good validity with respect to the way in which stress was measured in this study.
Table 3. Correlations between the Three Measures of Stress

<table>
<thead>
<tr>
<th>Correlations: Pearson $r$</th>
<th>STAI State - Baseline</th>
<th>STAI State - Post-Manipulation</th>
<th>Heart Rate - Baseline</th>
<th>Heart Rate - Post-Manipulation</th>
<th>Cortisol - Baseline</th>
<th>Cortisol - Post-Manipulation</th>
</tr>
</thead>
<tbody>
<tr>
<td>STAI State - Baseline</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>STAI State - Post-Manipulation</td>
<td>0.610* (62)</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.0000001386</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart Rate - Baseline</td>
<td>-0.0136 (55)</td>
<td>-0.0176 (55)</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.323</td>
<td>0.198</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart Rate - Post-Manipulation</td>
<td>0.48 (55)</td>
<td>0.250 (55)</td>
<td>0.793 * (53)</td>
<td>1.00</td>
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</tr>
<tr>
<td></td>
<td>0.726</td>
<td>0.065</td>
<td>0.00000000000014</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortisol - Baseline</td>
<td>0.88 (59)</td>
<td>0.116 (59)</td>
<td>-0.60 (52)</td>
<td>0.36 (52)</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.508</td>
<td>0.381</td>
<td>0.672</td>
<td>0.797</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortisol - Post-Manipulation</td>
<td>0.312 (51)</td>
<td>0.380* (51)</td>
<td>0.65 (46)</td>
<td>0.362* (46)</td>
<td>0.620* (50)</td>
<td>1.00</td>
</tr>
<tr>
<td></td>
<td>0.26</td>
<td>0.006</td>
<td>0.669</td>
<td>0.014</td>
<td>0.000000159</td>
<td></td>
</tr>
</tbody>
</table>

Note: Reported figures are Pearson's $r$ ($n$) $p$
*Correlation is significant at $p < .05$. 
The set of visible target trials is, as noted above, designed to ensure that all participants, regardless of prior computer gaming and other joystick experience, are well trained in the requirements of the task before moving onto the next, crucial, phase of the CO Arena task. If, as is the case with most mixed male-female samples, there is a great variation in prior computer gaming and other joystick experience, then one should expect to see more variance in visible target performance on the first couple of visible target trials, but a rapidly decreasing amount of variance toward the end of the set of trials. Table 4 shows that this is indeed the case: There is less variance in performance on later trials than on earlier trials, an indication that participants are becoming more proficient at moving within the VE and at locating the target.4

Statistical analyses, using repeated-measures ANOVA, support these impressions. Mauchly’s test indicated that the assumption of sphericity had been violated for the main effect of trials, \( \chi^2(5) = 107.08, p < .001 \). Therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity (\( e = .48 \)). There was a significant main effect of trials on path length to find the target, \( (F(1.44, 82.08) = 131.80, p < .0001) \), in the absence of a trials x gender interaction (\( p = .640 \)), a trials x experimental condition interaction (\( p = .652 \)), or a trials x gender x experimental condition interaction (\( p = .503 \)). With regard to between-subjects effects, the repeated-measures ANOVA showed there was a significant main effect of gender on path length to find the target, \( (F(1, 57) = 6.202, p = .016) \), in the absence of a main effect of experimental condition (\( p = .452 \)) or a gender x condition interaction (\( p = .870 \)). This statistically significant main effect is, as Figure 9 illustrates, driven by the relatively weaker performance of females compared to males, regardless of experimental condition.

To further illustrate the fact that, regardless of group assignment, participants were performing with relatively equal efficiency by the end of the set of visible target trials, we analysed performance on the final visible target trial only. Those data are presented graphically in Figure 9, which appears to indicate that males, regardless of group assignment, performed equally, and that females, regardless of group assignment, performed relatively equally, but that males took shorter path lengths to move to the target than did females.
Results of a one-way ANOVA confirmed these impressions. Levene's test of equality of error variances was significant, indicating that the assumption of homogenous variances across groups was not met. Thus, the Welch F-value is reported here. There was a statistically significant effect of group (either Stress-female, Stress-male, Control-female, or Control-male) on path length to the target, $F(3, 23.78) = 4.14, p = .017$. A set of planned contrasts revealed that the effect of group status was not significant (i.e., that the mean path length to the target of the Stress group participants was not statistically significantly different than that of the Control group participants), $t(57) = 1.69, p = .103$ (two-tailed). Another set of planned contrasts revealed that the effect of gender, regardless of group status was significant (i.e., that the mean path length to the target of the female participants was statistically significantly different than that of males), $t(57) = 3.09, p = .005$ (two-tailed).
**CG Arena: Hidden Target Trials**

Descriptive statistics for all participants on these trials are presented in Table 4. Figure 10 shows these data graphically. As can be seen, there is the suggestion that females in the Stress group are, on average, the poorest performers.

Statistical analyses of these data were again completed using repeated-measures ANOVA. Mauchly’s test indicated that the assumption of sphericity had been violated for the main effect of trials, χ²(9) = 25.958, p = .002. Therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity (ε = .82). With regard to within-subjects effects, there was no significant main effect of trials on path length to find the target (p = .304), no trials x gender interaction (p = .478), and no trials x experimental condition interaction (p = .800). The interaction effect of trials x gender x experimental condition was not significant (F(3.27, 186.69) = 2.193, p = .085).

With regard to between-subjects effects, there was no significant main effect of gender on path length to find the target (p = .339), no significant main effect of experimental condition (p = .668), and no gender x condition interaction (p = .123).

**CG Arena: Probe Trial**

As noted earlier, in the five ‘hidden target’ trials preceding this probe trial, the target was hidden in a fixed location in the northwest quadrant of the arena. The dependent variable of interest here, then, is how long participants in each group spent in that quadrant during this trial. Values of that dependent variable, for males and females in each experimental condition, are presented in Table 4, and are presented graphically in Figure 11. As can be seen, and contrary to expectations, males in the Stress group tended to spend slightly more time searching the appropriate quadrant than did participants in the other groups.
A two-way independent ANOVA confirmed the impression, however, that participants, regardless of group assignment, performed reasonably equivalently on this measure of cognitive mapping ability. Levene’s test of equality of error variances was non-significant, indicating that the assumption of homogeneous variances across groups was met. There was no statistically significant main effect of either gender or experimental condition on dwell time in the appropriate quadrant ($p = .309$ and .213, respectively), and no statistically significant interaction between gender and experimental condition on that dwell time ($p = .097$).
**Figure 11.** Probe Trial: Time Spent in Appropriate Quadrant

(Units are seconds. Error bars represent 95% confidence intervals.)

**Object Recognition Task (ORT)**

The $d'$ data for the ORT are presented in both Table 4 and Figure 12. I conducted a two-way independent ANOVA on these data. Levene's test of equality of error variances was non-significant, indicating that the assumption of homogeneous variances across groups was met. There was no statistically significant main effect of gender on recognition memory for pictures in the CG Arena experimental room, $F(1, 58) = .323, p = .572$, and there was no statistically significant interaction between gender and experimental condition on recognition memory for these pictures, $F(1, 58) = .501, p = .482$. There was, however, a statistically significant main effect of experimental condition on recognition memory for pictures in the CG Arena, $F(1, 58) = 13.55, p = .001$. In this case, participants in the Stress group outperformed participants in the Control group.
Figure 12. ORT Performance across the Two Groups
(Error bars represent 95% confidence intervals.)

Arena Reconstitution Task (ART)
Participant data for the ART are presented in both Table 4 and Figure 13. I conducted a two-way independent ANOVA on these data. Levene’s test of equality of error variances was non-significant, indicating that the assumption of homogeneous variances across groups was met. There was no statistically significant main effect of gender or of experimental condition on ability to reconstruct the spatial relationships of the pictures in the CG Arena experimental room ($p = .102$ and $.879$, respectively). There was also no statistically significant interaction effect between gender and experimental condition on ability to reconstruct the spatial relationships of the pictures in the CG Arena ($p = .928$).
Figure 13. ART Performance across the Two Groups.
(Error bars represent 95% confidence intervals.)

Verbal Learning and Memory

Between-Group Comparisons

Participants in the Stress group did not show poorer verbal memory performance than participants in the Control group (Stress group, males: 7.69 ± 3.54 words recalled; Stress group, females: 7.71 ± 2.82; Control group, males: 6.00 ± 1.85; Control group, females: 6.79 ± 2.72). I conducted a two-way independent ANOVA on these data. Levene's test of equality of error variances was non-significant, indicating that the assumption of homogeneous variances across groups was met. There was no statistically significant main effect of gender or of experimental condition on free recall of the word list, \( (F(1, 58) = .315, p = .577) \), and \( (F(1, 58) = 3.31, p = .074) \), respectively. There was also no statistically significant interaction between gender and experimental condition on free recall of the word list, \( F(1, 58) = .287, p = .594 \).
Within-Group Analysis: Stress group

Following Wolf et al. (2001), I conducted correlational analyses of the relationship between stress-induced cortisol increase and verbal memory. Within the Stress group, cortisol increase following the TSST was negatively correlated with free recall of the word list ($r = -.22, p = .133$, one-tailed). That is, participants who showed larger cortisol increases tended to recall fewer words than did those with smaller cortisol increases. Additional analysis revealed that there was a slightly stronger negative correlation in males ($r = -.24, p = .214$, one-tailed) than in females ($r = -.18, p = .256$, one-tailed).

I also investigated the association of verbal memory performance with baseline and post-TSST cortisol levels. Again, the correlations were relatively small and did not reach statistical significance. For baseline cortisol levels, I found the following: total group: $r = -.24, p = .212$; males: $r = -.24, p = .212$; females: $r = -.24, p = .212$. For post-stress cortisol levels ($n = 28$), I found the following: total group: $r = -.24, p = .212$; males: $r = -.24, p = .212$, females: $r = -.24, p = .212$. 
DISCUSSION

Based on several related strands of empirical evidence gathered by independent laboratories, I predicted that the physiological effects of an acute psychosocial stressor would, particularly in females, selectively disrupt a hippocampally-based neural system underlying cognitive map-guided navigation, while leaving intact neural systems underlying landmark-guided navigation. Additionally, following findings by, for example, Wolf et al. (2001), I predicted that verbal memory in a particular subgroup of males (cortisol responders) would be relatively impaired following exposure to the same acute psychosocial stressor. In essence, I systematically replicated elements of the designs used by Thomas et al. (2007) and Wolf et al. (2001), amalgamating them into one efficient design, in order to provide clarification on what appear to be conflicting findings regarding the effects of stress of verbal and spatial memory.

Results pertaining to a check of the experimental manipulation indicated that I was successful in administering the TSST and significantly raising cortisol levels, heart rate, and self-reported anxiety (and, by inference, subjectively experienced stress) in Stress group participants. Furthermore, participants in the Control group showed significantly lowered cortisol levels and heart rate, and reported decreased levels of anxiety, following a period of relaxation. Thus, as the participants entered the cognitive testing phase of the experiment, the TSST and relaxation procedures had effectively ensured that participants in the Control group were in a different physiological state to those in the Stress group, with the latter more likely to have temporarily impaired hippocampal function.

With regard to the visible target trials of the spatial navigation task, the findings here partially confirmed the a priori hypothesis (based on the trends reported by Thomas et al., 2007) that there would be no between-group difference in performance of a landmark-guided navigation task: There was no statistically significant difference between the Stress and Control groups taken as a whole. There was, however, a sex difference in that, regardless of group assignment, females performed more poorly than did males. This latter piece of data is consistent with numerous previous findings in the VE spatial navigation literature (e.g., Sandstrom et al., 1998). Nonetheless, our initial hypothesis of no between-group differences on this aspect of the spatial navigation task is confirmed.
With regard to the hidden target trials of the spatial navigation task, although the findings here tended toward statistical significance, they did not entirely confirm the hypothesis that women exposed to an acute stressor would perform more poorly than all other participants on the task assessing cognitive map-guided navigational ability. However, their study found that females and males who were not exposed to the stressor located and relocated a hidden target equally well (i.e., cognitive map-guided navigation was intact in unstressed participants), and that was also supported by this study.

These results are encouraging, as they tend towards the prediction of Jacobs and Nadel (1985) that exposure to an acute psychosocial stressor selectively disrupts cognitive map-based spatial navigation. Furthermore, although they do not identically replicate the findings of Thomas et al. (2007), they confirm a tendency toward a sex difference in performance of the cognitive map-guided spatial navigation task under conditions of acute psychosocial stress.

Results from the CG Arena probe trial and the ART, the post-CG Arena task designed to tap cognitive mapping ability, were similar in that they suggested there were no between-group or sex differences, and no interaction effects. That is, there were no differences between males and females, or between participants in the Stress group and the Control group, on tasks that required them to (a) persist in searching for the (now-absent) target in the place it had previously been hidden, and (b) reconstruct the spatial layout of the virtual environment using cardboard icons. These data, then, disconfirm the hypothesis that females in the Stress group would show impaired cognitive mapping ability relative to males generally and to females in the Control group. One possible explanation for the discontinuity between the these data and others from the CG Arena is that, particularly in non-clinical populations, these dependent measures may not be sensitive enough to detect subtle between-group differences in performance.

The results on of the recognition memory test for pictures in the experimental room of the CG Arena (the ORT) delivered some surprises. Consistent with expectations, there were no male-female differences in performance on this task; contrary to expectations, however, the data analyses demonstrated that there was a statistically significant performance difference between participants in the Stress group and those in the Control group. Even more surprisingly, those in the former group outperformed those in the latter group.
This result is inconsistent with well-documented research showing deficiencies in human episodic memory performance associated with acute glucocorticoid elevations (see Lupien & McEwen, 1997, for a review). A large animal literature, however (see Roozendaal, 2000, for a review) has established that, in aversive conditioning paradigms, corticosteroids can enhance memory performance. Several relatively recent studies of human memory performance have built on the latter findings and on the well-established notion that emotional information tends to be remembered better than neutral information (Heuer & Reisberg, 1990), and have demonstrated that mild to moderate cortisol elevations are associated with improved memory for material featuring emotional content.

For instance, Buchanan & Lovallo (2001) exposed 48 participants (who had received either oral administration of cortisol or placebo) to pictures varying in emotional arousal. Their results showed that elevated cortisol levels during memory encoding enhanced 1-week delayed recall of emotionally arousing pictures relative to neutral pictures. Similarly, Payne et al. (2007) showed that inducing psychosocial stress via the TSST prior to encoding impaired encoding of neutral information, but enhanced encoding of episodes containing emotional information.

As can be seen in Figures 1 and 2, the pictures featured in the CG experimental room and subsequently presented in the ORT were not emotionally arousing (indeed, they were chosen because of their emotionally neutral tones). Why, then, did the stress manipulation enhance recognition memory for these photographs, particularly as cortisol levels were reaching a peak during encoding of the material (there is recent animal literature showing that stress can have an enhancing effect on recognition memory if administered during consolidation phases; see Maroun & Akiray, 2008)? This question is left to future studies, where one might explore whether particular aspects of the CG Arena and ORT stimuli lend themselves to an improvement in recognition memory following stress, or whether this is a spurious result within the current dataset.

With regard to the verbal memory test, I predicted (on the basis of data reported by Wolf et al., 2001) that (a) male subgroup showing highest levels of cortisol increase in response to the acute stressor would perform more poorly on free recall than would females exposed to the same stressor, and (b) the female subgroup showing highest levels of cortisol increase would
perform no differently than the female subgroup showing lowest levels of cortisol increase. The second part of that prediction was confirmed, in that there were no significant between-group differences on the task. Within the Stress group, however, cortisol increase following the TSST was negatively correlated with free recall of the word list, with a slightly stronger negative correlation in males than in females.

The sex differences observed in the current data were not as striking as those reported by Wolf et al. (2001), however: They reported a correlation observed in men of $r = 0.82$ ($p < 0.05$), and in women of $r = -0.05$ ($p = 0.87$). A possible explanation for this is that the within-group analysis in both studies included very few participants (Wolf et al. (2001) had 8 men and 14 women in their Stress group, whereas 13 men and 15 women were included in the current Stress group analysis). Thus, effects may be difficult to replicate across studies.

Another possible reason for the failure to replicate the earlier finding is that the cortisol increases in both male and female participants in the Stress group were larger in the Wolf et al. (2001) study than in this study.

It should be noted, however, that in the current study I observed no sex differences in cortisol increase in response to acute social stress. Many recent most studies have, however, reported that there are major sex differences in sensitivity to acute social stressors (Kudielka, Buske-Kirschbaum, et al., 2004; Kudielka & Kirschbaum, 2005; Uhart, Chong, Oswald, Lin, & Wand, 2006), with males typically having a greater HPA axis response than females.

An explanation for this result may be that I carefully controlled for the impact of the menstrual cycle. Females have been found to show different reactions to stressors depending on the time of their menstrual cycle (Kudielka, Buske-Kirschbaum, et al., 2004; Wolf et al., 2006) and on whether they use oral contraception (Kirschbaum et al., 1995). In the current study, none of the female participants used oral contraception, and all were tested during their luteal phase, which may have aligned their cortisol response to the same levels as males. A similar finding was reported by Kirschbaum et al. (1999) in a study that looked at the impact of gender, menstrual cycle phase and oral contraceptives on HPA axis activity. They found that although men showed significantly higher ACTH responses to the TSST, their salivary cortisol responses were at a comparable level to a group of women tested in their luteal phase.
With regards to the lack of difference in verbal memory performance between the Stress and Control groups (a result that replicated data reported by Wolf and colleagues (2001) but that is in contrast to findings from several prior studies; see, e.g., Newcomer et al., 1999), there may be several reasons for this disparity. First, the levels of cortisol increase following the TSST are lower than those observed in pharmaceutical studies (see, e.g., Kirschbaum et al., 1996), and are even lower than those observed by Wolf et al. (2001). Second, the memory test employed (list learning, a brief delay, and then free recall) may be less sensitive to cortisol-induced effects than the working memory or declarative memory tests used in other studies (e.g., Lupien et al., 1994). Third, in the current study, as in the Wolf et al. (2001) study, stress exposure occurred prior to learning; in other studies (e.g., Lupien et al., 1997) stress exposure occurred between learning and recall.

Limitations and Directions for Future Research

The current study set out to show that there may be contrasting effects of acute psychosocial stress on men and women performing verbal and spatial memory tasks. Although the a priori hypotheses were, by and large, not confirmed, the results tended toward statistical significance in the predicted direction, which indicates that there is continued promise in the study of the impairing effects of stress on cognitive function, and that sex may moderate that relationship.

Several limitations of the current study should be addressed by future researchers who wish to more clearly delineate the relationships of interest. First, although the sample size used here was of the same order as (or larger than) those used in previous published studies in this field, the effects being studied may require a larger group of participants. Particularly given the inherent fragility of salivary cortisol samples (as shown by the number of participants in the current study whose cortisol data could not be analysed), collecting larger numbers of participants is imperative.

Second, the current study relied on self-report of menstrual cycle phase. Although I suspect that the female participants were accurate in their reporting (as borne out by the fact that females in the Stress group had cortisol increases were of similar magnitude to those of males in that group), future studies might add physiological measures of menstrual cycle to ensure improved accuracy. In the same vein, future research might investigate the effects of time of menstrual cycle on cognitive performance following stress; in particular, following rat
studies, investigations into the protective effects of estradiol in young females might be of interest (Galea et al., 1999).

Finally, both basal cortisol levels and cortisol increases in the current study were significantly lower than those reported in previous TSST studies (e.g., Kirschbaum et al., 1999; Wolf et al., 2001). Although the differences in magnitude of increase might be explained by small differences in the current administration of the stress induction procedure, the differences in basal cortisol levels are not so easily explained, particularly given that all participants in the current study were run at the same time of day as those in previous studies, and were within the same age range. This question, then, remains open to further exploration.
REFERENCES


APPENDIX A

Verbal Memory Task: Word List

Nutmeg
Factory
Nursery
Ankle
Cigar
Caravan
Boulder
Clock
Temple
Peach
Potato
Corn
Hotel
Village
Toast
Market
Fabric
Beast
Chair
Pepper
Cattle
Shoes
Python
Engine
Vest
APPENDIX B

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
The impact of acute psychological stress on spatial cognition

3. Principal Investigator and Telephone Number(s)
Kevin G. F. Thomas, Ph.D.
Department of Psychology
University of Cape Town
021-650-4608 and 021-650-3430

4. What is the purpose of this research study?

The purpose of this research study is to understand better how people process spatial information, how they learn to find their way around new environments, and how exposure to acute psychological stress affects this type of cognitive functioning. The main goal of the research is to try to understand how the brain processes information which requires memory for spatial detail, and to understand aspects of gender variables and brain activity that distinguish individuals who have been under acute psychological stress and from those who have not.
5. **What will be done if you take part in this research study?**

This study requires you to complete a 20 minutes presentation, followed by a 20 minutes virtual reality task on a computer based experiment. Another 10 minutes will be spent completing a Verbal Memory test. Throughout the study (over 3 steps), we will assess the level of your stress by collecting saliva sample with the aid of a cotton swab.

6. **What are the possible discomforts and risks?**

None.

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

With the result of this study, society will benefit from a better understanding of the effects of acute psychological stress on cognitive functioning. This research can also be applied to people who have experienced stressors or increased cortisol levels, in that it may help improve their medical/psychological treatment in the future.

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.
10. 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:
FOOTNOTES

1For instance, Spelke (2005) provides evidence that mathematical and scientific reasoning develop from a set of biologically-based cognitive capacities that males and females share. She argues that, therefore, these capacities can lead men and women to develop equal talent for mathematics and science.

2See Gordon & Lee (1993) for contradictory findings, however.

3Some studies have, however, found that cortisol and memory retrieval in women are not influenced by menstrual cycles but by the use of oral contraceptives that render women who use them insensitive to acute increase of cortisol (see, e.g., Kuhlman, Piel, & Wolf, 2005).

4This increasingly efficient performance cannot, however, be characterized as a learning curve for the spatial layout of the CG experimental room (i.e., the development of a cognitive map): The visible target is in a different location on each trial.