The Relationship Between Acute Stress, Chronic Stress and Spatial Performance

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Abstract

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Many theories differentiate the cognitive impacts of acute versus chronic stress. For example, acute stress has been suggested to alter visual attention (Williams & Anderson, 1997), and chronic stress has been implicated in hippocampal atrophy by means of hypercortsolism (Sapolsky, 1999). This study related the stress indicators blood pressure, heart rate, and self-report questionnaires, with performance in a spatial navigation task, the virtual Morris Water Maze. Fifty-four university students participated. Significant positive relationships found between the questionnaires and two of the physiological measurements lend physiologic support to the questionnaires. Some support was found for the relatedness of acute and chronic stress measurements (hypothesis one). This study shows that a coalescence of chronic and acute stress indicators relates more closely with performance than either chronic or acute stress exclusively (hypothesis two). This uniquely suggests that acute and chronic stress are both instrumental to the stress-performance relationship. A quadratic relationship found between the main performance variable, mean time latency, and a combination of chronic and acute stress variables indicates the lowest stress levels corresponded with the lowest performance, and highest stress levels with both the poorest and best performance (F (2, 46) = 3.57, $p < .05$). Also of interest is daily video-gamers' significantly higher blood pressure readings.

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The Relationship Between Acute Stress, Chronic Stress and Spatial Performance

The scientific study of the relationship between stress and performance dates back to the beginning of the twentieth century. The relationship between stress and performance remains an interesting topic of research for its complexity, involvement across psychobiological systems, and wide variety of applications, such as job performance and cognitively degenerate diseases. Research within the past decade has provided us with greater knowledge about the stress response and its relationship with performance, enabling further development of stress-performance theories. Prior to describing the methods, results and discussion of the present study, this paper will provide a general description of physiological stress, the generality model of stress response, the integrated-specificity model of stress response, chronic stress, the stress-performance relationship, and spatial navigation performance.

Physiological stress

French physiologist Claude Bernard founded the concept milieu interieur (now known as homeostasis) in the mid 1800s. In 1865, Bernard described the perturbation of homeostasis: "[T]here are protective functions of organic elements holding living materials in reserve and maintaining without interruption humidity, heat and other conditions indispensible to vital activity. Sickness and death are only a dislocation or perturbation of that mechanism (as cited in Weiner, 1992)". Bernard's description was a prelude to the present day definition of stress -- the perturbation of homeostasis (Palumbo, et al., 2010). The cause of perturbation is referred to as the stressor, and the state of being disturbed is termed stress. The body's defensive response to the stressor is referred to as the stress response (Selye, 1975). Stress

responses comprise inherent defense systems which the body uses to combat the stressor(s) and regain homeostasis.

In 1936, Hans Selye discovered that uniform physiological changes occur in an organism in response to a variety of stressors (as cited in Noble, 2007). This general physiological stress response is referred to as the general adaptive syndrome (GAS). The GAS recognizes three stages within the physiological stress response: the alarm stage, the resistance stage, and the exhaustion stage (Selye, 1956). During the alarm stage, the body prepares to deal with the stressor. In the resistance stage the body combats the stressor. The body cycles between these two stages until the stressor is defeated or until the body goes into the exhaustion stage, in which case the body is defeated by the stressor. If and when the body has fully entered the exhaustion stage, all of the body's resources are depleted, disabling the body from performance and normal function (Baumeister & Bushman, 2008). Of acute stress symptoms, the stressed individual may be most keenly aware of increased anxiety and hyper-focused attention. Depression and 'burn-out syndrome' are some of the most noticeable symptoms of chronic stress (Staal, 2004). Stress has the capacity to effect performance throughout these stress response stages, and long-term effects of stress can continue to affect performance after the stressor has passed.

The body enters the alarm stage within seconds of perceiving a stressor. The sympathetic nervous system (SNS) is immediately activated, and instigates the body's first stress responses. The SNS is part of the autonomic nervous system, which promotes the body's involuntary functions. The SNS optimizes the body's immediate functionality to fight the stressor. A major aspect of the SNS is the adrenal medulla's release of stress hormones epinephrine (adrenaline) and norepinephrine

(noradrenaline) into the bloodstream, which increase heart rate, vasoconstriction, and consequentially, blood pressure (Thompson, 2000).

The hypothalamic-pituitary-adrenal (HPA) axis is another stress response system that regulates hormone levels. Changes in the HPA axis are seen within 20-40 minutes from perception of the stressor. First, the corticotrophin-releasing hormone (CRH) is released from the hypothalamus. CRH initiates the release of adrenocorticotropic hormone (ACTH) from the pituitary gland. ACTH signals the outer layer of the adrenal gland (the adrenal cortex) to produce the stress hormone cortisol. The effects of cortisol are extensive. Cortisol increases blood glucose levels, which supplies energy for the body, and especially the brain. Cortisol also breaks down protein into amino acids, which aid in tissue damage repair. Peripherally, cortisol levels increase in blood, saliva and urine. Cortisol enhances endothelium sensitivity to the aforementioned vasoconstriction effects of epinephrine and norepinephrine. Centrally, cortisol increases (via the bloodstream) in the hippocampus, where it binds to glucocorticoid receptors (Kemeny, 2003). Once a certain number of hippocampal glucocorticoid receptors are bound with cortisol, the receptors signal the hypothalamus to stop ACTH production, which stops cortisol production. This negative feedback mechanism prevents over production of cortisol (Michaud, Kelly & Anisman, 2008). A dysfunction in this negative feedback mechanism has been consistently connected with chronic stress (Wahbeh, Kishiyama, Zajdel, & Oken, 2008).

Following the alarm stage is the resistance stage of the general adaptive syndrome. In the resistance stage, some of the outward stress symptoms of the alarm stage subside, and regress to a more normal state. In the resistance stage, most of the

body's non-vital resources are still allocated to combating the stressor, at the expense of other functions such as immunity to other threats. Therefore, the resistance stage is only sustainable for a finite length of time. The body cycles between the alarm stage and the resistance stage. If successful, the stress responses defeat the stressor and the body returns to its normal state. If unsuccessful, the body becomes depleted of resources, and enters the exhaustion stage. Without external intervention in the exhaustion stage, physiological damage, and ultimately, death can result (Thompson, 2000).

The generality model of stress response

The generality model of stress response proposes that there is only one type of stress response (Kemeny, 2003). This model is consistent with Selye's general adaptive syndrome. The concurrences of stress responses support this model. Increases in psychological and physiological stress markers typically exhibit a direct correlation with one another (Malmo, 1957; Michaud, et al., 2008). The common antecedents of stress responses also support this model. Various types of stressors have been shown to create the same stress responses (Bedwell, Renk, Orem, Sims, 2009; McKay, Buen, Bohan, & Maye, 2010; Oldehinkel, Verhulst, & Ormel, 2008). For example, early research demonstrated increased muscle tension in response to the psychologically stressful task of solo flight training (Williams, Mcmillian & Jenkins, 1946). Physiological data collected from lie-detectors is used to infer whether a person is experiencing psychological stress. Increases in galvanic skin response have been induced by the administration of an academic exam (Bronzaft & Stuart, 1971).

Some factors seem to have an influence on the body's ability to overcome a stressor, and/or how cognition is affected by stress (McEwen, 2003). For example,

Virgin and Sapolsky observed that the behavioral styles of male olive baboons seem to moderate socially-induced chronic stress (1997). Oldenhinkel, Verhulst and Ormel (2008) found lower heart rate to be indicative of stress resilience in humans. Modern generality models recognize these factors as moderators of the stress response. As moderators, these factors do not qualitatively change the stress response, but can suppress or exacerbate the outcome.

Several classifications have been created for stress-response moderators. These include stressor severity, the strength of physiological orienting to stimuli (Zuckerman, 1990), individual stress sensitivity (Hennessy, 2000; Radu, 2003), cognitive appraisal of the circumstance demands relative to self-perceived resources (Kemeny, 2003), and emotion (Duffy, 1957). These moderators, in turn, may be shaped by the many factors believed to influence learning, memory and the brain, including genetic make-up, pre- and post-natal development events, and life experiences (McDonald, Devan & Hong, 2004). In modern generality models these factors are considered moderators, rather than modifiers of the stress response, because they are relevant only to the extent to which they buffer or exaggerate the effects of the stressor (Kemeny, 2003).

The integrated specificity model of stress response

A deviation from the generality model was prompted by compounding evidence of distinct neural systems linking stress, emotion, and performance. The integrated specificity model proposes that qualitatively different types of stress responses exist. This model attributes a greater significance to the factors that seem to influence the stress response, than do the generality model, and the modern generality models of stress response. Rather than moderators of the stress response, the integrated-specificity model views these factors as the determinants of which distinct emotional and psychoneuroimmunological path is taken.

For example, participants have produced different physiological responses to various types of cognitive appraisals (Kemeny, 2003). Kemeny (2003) describes three different cognitive appraisals: challenge versus threat, perceived control, and social cogntition. Whether a participant appraises a stressful situation as a threat or challenge is determined by how she or he perceives her or his resources (e.g. resources of intellectual, social and/or financial relevance) in relation to the demands of the situation. If the individual perceives her or his resources to exceed the demands of the situation, she or he would appraise the situation as a challenge, with the reverse perception resulting in the appraisal of the situation as a threat. Blascovich and Tomaka (1996) found that circumstances perceived as challenges were correlated with increased sympathetic arousal (higher heart rates), and reduced or maintained levels of blood pressure. Appraisal of the situation as a threat corresponded with high higher heart rate and blood pressure (Blascovich and Tomak, 1996, as cited in Kemeny, 2003). In another study, participants were given contrived information

about failure and personal reproach. The intent of the researchers was to influence the participants' perception of the task demands as beyond their abilities. This negative information resulted in poorer performance of participants who measured high in anxiety on the Taylor Manifest Anxiety Scale (Ishiguro, 1965).

Perceived control over a stressor has been observed to result in less severe immune system responses, regardless of whether the participant has real control over the stressor (Kemeny, 2003). Rodents with control over stressor exposure showed lower corticosterone (stress hormone) responses than those without control. In laboratory settings, humans have shown higher HPA activation when the stressors were not under their control (Dickerson, & Kemeny, 2004).

Emotional and physiological responses in humans have differed in the presence of a social evaluative threat (Dickerson, 2008). Instrumental but not emotional support was shown to elevate systolic blood pressure measurements during task performance (Hughes, 2005). Kemeny (2003) describes a study that demonstrated HPA activation when social status or social self-esteem was dependent on successful performance of a demanding task. HPA activation was diminished in the absence of this social threat, indicating the contributing role of social cognition to physiological stress response during performance. Effects of social-standing on stress have also been observed in primate communities. As mentioned above, Virgin and Sapolsky (1997) documented higher levels of stress hormones (glucocorticoids) in subordinate olive baboons, with behavioral style a moderating factor of the severity of stress response.

Chronic stress

Chronic stress can produce immediate and long-standing effects on the individual. Both exhaustion-stage stress and prolonged resistance-stage stress can result in physiological and psychological damage (McEwen & Dahar, 2002). Chronic stress can involve the exhaustion stage of the general adaptive syndrome, but often, chronic stress exclusively consists of an extended resistance stage. For example, posttraumatic stress disorder and chronic depression are both related with prolonged resistance-stage stress (Kemeny, 2003). Stress defenses utilized in the resistance stage are designed for short-term use, and extended activation of these defenses can pose permanent adverse effects on the individual (Bäckström, 2008; Rosch, 1999). McEwen (1998) termed allostatic load to refer to the aggregate penalty incurred by chronic activation of physiological stress-response systems. A few of the many areas susceptible to the affects of chronic stress are perceptual ability, cognitive performance, working memory, motor control (Staal, 2004), and increased vulnerability to upper respiratory infections (Kemeny, 2003).

The stress hormone cortisol is an exemplar of a stress response that can be beneficial in the short-term but detrimental when chronically employed. Cortisol increases vasoconstriction and consequentially, blood pressure. While raised blood pressure can help fuel muscles for a short-term, physical challenge, chronically high blood pressure seems to be maladaptive, and is linked with hypertension and cardiovascular disease (Carroll, Ring, Hunt, Ford, & Macintyre, 2003). Chronically high cortisol also may reduce brain glucose supply (Bäckström, 2008; Rosch, 1999).

As previously mentioned, the HPA-axis is responsible for regulating stress

hormones. HPA-axis dysfunction can create prolonged elevation of cortisol, and it occurs in conjunction with many mood disorders including anxiety disorder, depression, borderline personality disorder, post-traumatic stress disorder, attention deficit disorder as well as others such as alcoholism and Alzheimer's disease (King, Leichtman, Abelson, & Seng, 2008; Sheline, Wang, Gado, Csernansky, Vannier, 1996). One cause of abnormally high and prolonged production of cortisol seems to be a disruption of the previously mentioned hippocampus-based negative feedback mechanism of the HPA-axis (Wahbeh, et al., 2008). As glucocorticoid receptors in the hippocampus are occupied by cortisol, glucose is prevented from binding to these receptors and thereby from entering the hippocampus (Ginsberg, Pecoraro, Warne, Horneman, & Dallman, 2010). By blocking the influx of glucose to the hippocampus, the elevated cortisol levels lower hippocampal metabolism (Convit, et al., 1997). Chronically elevated cortisol unrelated to HPA axis function is believed to damage the hippocampus by the same means of reducing brain glucose supply (Magariños & McEwen, 1995; McEwen & Sapolsky, 1995; Quirin, Pruessner, & Kuhl, 2008; Sapolsky, 2000).

This link between cortisol and hippocampus atrophy is supported by data showing that individuals who have experienced chronic stress have smaller hippocampus structures and a lower hippocampal cell count than those who have not (Mondelli, et al., 2010; Sheline, Wang, Gado, Csernansky, Vannier, 1996). These individuals demonstrate hippocampal-related cognitive impairments in learning, memory, perceiving spatial relationships and temporal sequencing (Elgh, et al., 2005; Wolf, et al., 2002). Csernansky et al. (2006) found the rate of mental decline in Alzheimer's disease to be positively correlated with cortisol plasma levels and HPA-

axis dysfunction. In a separate study, individuals with mild cognitive impairment had higher levels of cortisol upon waking, indicating that HPA-axis dysfunction occurs in the beginning stages of cognitive decline. This supports the theory that HPA-axis dysfunction is a causal factor in cognitive impairment (Lind, Edman, Nordlund, Olsson, & Wallin, 2007).

Stress and Performance

In 1908, researchers Yerkes and Dodson found a curvilinear relationship between various intensities of electrical shocks and the time it took mice to demonstrate learning of a discrimination task. Subsequent literature extrapolated that the varying levels of shock intensity in the study induced respective levels of stress in the mice. The concept of a curvilinear stress-performance relationship, with a moderate level of stress producing optimal performance, became known as the Yerkes-Dodson law of performance (Staal, 2004). Possibly due to the widespread references to the Yerkes-Dodson law, the putative theory regarding the relationship between stress severity and performance is that a moderate degree of stress produces optimal performance (Staal, 2004).

However, upon re-examination of the original study, some critics have pointed out methodological problems in the original Yerkes and Dodson study, such as lack of accuracy in measuring stimuli intensity. These methodological problems have resurfaced doubts in the Yerkes-Dodson law, as have the failures of some attempts to replicate the results. A study of chicks by Cole in 1911, and another study by Dodson in 1915 of kittens both produced linear results between the stimulus and performance. However, some studies have since found curvilinear relationships between various types of stressors and performance. Two separate studies by Stauffacher (1937) and

Courts (1939) found a curvilinear relationship between human learning performance and muscle tension, with moderate tension producing optimal performance (Courts, 1942). A curvilinear relationship between physiologic stress and job performance was found for a group of student nurse anesthetists. Although like the Yerkes-Dodson law in its quadratic shape, this finding contrasts the Yerkes-Dodson law, as this study found moderate stress to be related with moderate performance, and high stress linked with both poor and excellent performance (McKay, et al., 2010).

As previously mentioned, the integrated-specificity model of stress response proposes that moderators can have differential impacts on stress outcomes (Kemeny, 2003). If stress impacts performance, then under the integrated-specificity model of stress response, stress-response moderators would also have the capacity to impact performance. Emotion has been indicated as stress-response moderator that affects performance. In a rodent study, stress-induced performance impairments were created, and then reversed using a drug that targets specific chemical receptors related to emotion (Metz, Jadavji & Smith, 2005). The administration of stress hormone corticosterone impaired rat performance in a motor and working memory task. The performance impairment was then reversed with administration of an anti-anxiety benzodiazepine drug, diazepam. Diazepam enhances the binding of GABA and benzodiazepine receptors (a subset of $GABA_A$ receptors), and the resulting neuron inhibition reduces corticosterone levels and anxiety.

Support for differential performance effects of cognitive, perceptual and social stress-response moderators have also been yielded. Hennessy (2000) found that higher measures of participant stress reactivity were linked with poorer driving performance. Results of a study by Petrac, Bedwell, Renk, Orem, and Sims (2009)

suggested that acute stress improves divided attention performance. A narrowing of peripheral vision has been found in individuals experiencing acute stress. This stress symptom was shown to impair athletic performance by reducing the player's awareness of peripheral visual cues. The effect was found to be more extreme with individuals who had a history of negative life events, low social support, and poor coping skills (Williams & Anderson, 1997).

Spatial navigation performance

The hippocampus has been consistently associated with spatial performance and cognitive mapping (O'Keefe & Nadel, 1978; Parslow, Fleminger, Brooks, et al., 2004; Sutherland, Kolb, & Whishaw, 1982). Rodent studies have implicated a network of other structures in spatial-learning in addition to the hippocampus, such as the fornix/fimbria and medial caudate putamen (Devan, Goad, & Petri, 1996; Devan, McDonald, & White, 1999; Sutherland & Rodriguez, 1989). Devan and White (1999) found that fimbria/fornix lesions were responsible for the complete impairment of cognitive-spatial information acquisition, and ability to learn place cues. Medial caudate putamen lesions and asymmetrical lesions of the hippocampus and medial caudate putamen were shown to partially impair the acquisition of cognitive-spatial information.

Existing literature on humans has also produced substantial evidence for the use of the hippocampus in spatial tasks (Rains & Milner, 1994). Case studies of patients with structural hippocampus impairments from traumatic brain injuries (TBI) and surgical lesions performed to reduce epileptic symptoms have provided evidence of the reliance of spatial performance on the hippocampus in humans (Rains & Milner, 1994). Virtual environments are a useful method for studying spatial

performance in humans, as they allow for total experimental control over the environment. Utilizing blood-flow activation technology such as positron emission tomography (PET), and functional magnetic resonance imaging (fMRI), contemporary research using virtual environments have continued to indicate the essential role of the hippocampus in spatial learning and memory (Parslow, Fleminger, Brooks, et al., 2004; Skelton, Bukach, Laurance, Thomas, & Jacobs, 2000).

The Morris Water Maze task (MWM; Morris, 1981) is considered by many the 'gold standard' of spatial learning and memory in rodents (McDonald, Hong & Devan, 2004). The MWM task is performed in a circular pool of opaque water containing an escape platform. In each trial, the rat finds the platform using spatial cues. When the water line is below the top of the platform, it is visible, and can be located using the local visual cues of the platform itself. When the water line is above the top of the platform, the platform is not visible. This condition demonstrates ability to locate the platform using only distal visual cues in the environment. The distal visual cues are objects located outside of the pool but within the rat's visual field. Finding the hidden platform from different starting locations in the pool demonstrates allocentric spatial abilities. Allocentric refers to learning location based on the spatial relationships among the distal cues in the environment independent of the subject's relative position. Allocentric spatial learning contrasts with egocentric spatial learning, which depends on the distal cues in the environment in conjunction with the location of oneself. An automated computer system measures MWM performance with visual tracking and recording. Performance is quantified into several measures, including the accuracy of the subject's swim path trajectory, time to

reach the platform, and the percentage of time spent in each quadrant of the pool (Morris, 1981).

The virtual Morris Water Maze (vMWM) was created by Jacobs, Laurance & Thomas in 1997 as a comparable human version of the MWM. The vMWM has served as a useful tool in many studies to explore different facets of spatial memory and performance in humans (Hamilton, Driscoll, & Sutherland, 2002; Jacobs, Laurance & Thomas, 1997; Jacobs, Thomas, Laurance, & Nadel, 1998). Despite the physical navigation in the vMWM being virtual rather than physical, patients with chronic bilateral vestibular failure showed visual spatial impairments in the vMWM environment, indicating that vestibular cues may provide similar contributions to navigation in virtual environments as they do in physical environments (Schautzer, Brandt, Kalla, Strupp, & Hamilton, 2003). The comparability of human vMWM performance to rodent MWM performance has also been supported by fMRI studies. Hippocampal blood flow was notably increased on fMRIs during human performance in the vMWM (Hamilton, et al., 2002). The vMWM performance impairments of patients with axonic hippocampal damage correlated with their hippocampal loss (Morris, Parslow, Fleminger, Brooks & Giametro, 2005). N-methyl-D-aspartate receptor (NMDA) blockers are believed to disrupt long-term potentiation in the hippocampus, an important indicator of learning. When NMDA blockers were administered to human participants, visual spatial performance in the vMWM was impaired (Rowland, et al., 2005).

In cognitive spatial performance, younger age is related with better performance, and males typically outperform females (Conrad, 2010; Astur, Ortiz, & Sutherland, 1998; Burkitt, Widman, & Saucier, 2007). Hippocampus-based theories

are among the predominant explanations for both the age and gender differences. As previously mentioned, the hippocampus is believed to play a large role in cognitive spatial abilities, and it is one of the first brain regions to be negatively affected in the aging process (Newman & Kasniak, 2000). Because hippocampal atrophy progresses with age (Höschl & Hajek, 2001), it follows that consequential performance impairments would also increase as a factor of age.

Explanations for the gender performance gap range from differences in spatial task experience to the morphological effects of testosterone (Halpern, 2000; Prestopnik & RosKos-Ewoldsen, 2000). Providing visual task training to females to compensate for possibly more limited experience with computer and video games reduced the performance gender gap in one study (Waller & Hunt, 1999). A 2006 study by Tan, Czerwinski, and Robertson showed that the gender gap in virtual spatial performance could also be reduced by the use of larger computer screens.

In a study of humans in the vMWM, low testosterone in females was linked with poorer performance (Burkitt, et al., 2007). High testosterone females exhibited the same level of performance as the males. Male performance did not differ on accord of testosterone-level variations. Some studies have found significant relationships between found exogenously injected testosterone related variations and performance in male rodents (Burkitt et al., 2007). This testosterone and spatial performance relationship may be a product of testosterone-mediated enhanced morphology of the hippocampus. In 1992, Roof and Havens demonstrated that neonatal administration of testosterone in rodents produced a larger and more laterally asymmetrical granule cell layer of the hippocampus. A significant correlation between the size of granule cell layers and maze performance was found.

In humans, a similar cognitive enhancement effect has been found for females with post-menopause estrogen treatment. Estrogen seems to affect functional aspects of the hippocampus. In vivo, estrogen has enhanced synaptic transmission and long-term potentiation in the hippocampus (Thompson, 2000).

Present study

Overview

This study related spatial navigation performance with chronic and acute stress in humans. This study also examined preference for stimulus-cue versus place-cues spatial navigation strategies under stress. This study predicted that a combination of acute and chronic stress would depict a stronger relationship with performance than either acute or chronic stress alone, and investigated whether the stress-performance relationship in this study would support the inverted-U stress-performance theory.

It was not presumed that there is a clear causal relationship among any of the three factors being measured (chronic stress, acute stress, and performance). Instead, it was believed that a mutually-influencing relationship exists between these three factors and other moderating factors previously mentioned. It was predicted that acute stress would be positively related with chronic stress (hypothesis 1). It was predicted that performance would show a stronger relationship with a coalescence of chronic and acute stress indicators than it would with chronic or acute stress exclusively (hypothesis 2). It was predicted that stress levels would be predictive of time spent during the probe trial in the first platform location (hypothesis 3). A quadratic relationship between stress and performance depicting high and low extremes of

stress related with poor performance, and moderate levels of stress related with optimal performance would support the Yerkes-Dodson stress-performance theory.

Method

Participants

Fifty-four Towson University students participated in this study. All participants were between the ages of eighteen and thirty, with 92 percent of participants between the ages of eighteen and twenty-three. 65 percent of the participants were female, and 35 percent were males. 46 percent of the participants declared psychology as their major. All study sessions were conducted between the hours of 10:00 am and 4:00 pm.

The participants were recruited from two undergraduate psychology classes, and from the Towson University Psychology Department's research pool website (http://researchpool.towson.edu). This study excluded individuals who had previous experience with the vMWM, and who participated in a competitive sport at the time of the study, due to athletes' above average ability to perform under the pressure of competition (Strahler, Ehrlenspiel, Heene, & Brand, 2010). In order to prevent a confounding effect from a recent or atypical consumption of caffeine (Anderson, 1994), participants will be asked to consume the amount of caffeine they typically consume on a given day, but to abstain from caffeine the hour prior to their participation.

Procedure

Each session of this study consisted of one study administrator, one study assistant, and either three or four participants. The following procedure was carried out for each session. All participants were invited into the study room, asked to turn

all electronic devices on silent, place their belongings on a shelf and have a seat at any of the four desks in the room. Each of the four desks and chairs faced a wall and were separated from each other by translucent curtains. Participant identification numbers (IDs) were randomly assigned to the desks prior to the study session.

Participants were asked to remain seated with both feet on the floor, without talking, for four to five minutes to provide resting vital sign measurements. Participants were asked to fill out the consent form and the STICSA Trait Stress questionnaire on their desk and then turn them face-down. As each participant finished the questionnaire, the papers were collected from that participant, and a blood pressure/ heart rate monitor was placed on the participant's left wrist. Participants read an issue of the magazine *Hawaii* for the remainder of the five minutes. The magazines were rotated among the desks between sessions. After the participants had been seated for four to five minutes, the resting blood pressure and heart rate readings were taken. Throughout the study, the study administrator took readings for participant one and two, and the study assistant took readings for participants three and four. The order of readings was maintained within each session, and reversed between each session. For example, readings from participants one and three were taken first for all three readings, and readings from participants two and four were taken second. For the following session, readings from participants one and three were taken second, and readings from participants two and four were taken first.

Once the first readings had been taken, the participants followed the administrator and assistant to a room across the hall. As the participants entered the second room, they were directed to the computer they would be using. The four

computers were randomly assigned to participant IDs. Each session used a different pairing of computers and participant IDs. Once the participants were seated, the study administrator read aloud a scripted description of the computer task, which began as follows: "…This task measures cognitive abilities such as ability to navigate, spatial memory, and ability to recall under pressure. You will be competing against each other, with the goal of completing each round more quickly than the other participants. Your scores will be posted next to your names on the front board…along with your placement, which will be first, second, third, or last. Your job is to perform to the best of your ability." At the front of the room was a two foot by three foot white board, on which was written four mock names, scores, and placements, with the implication that the names and scores were from a previous session.

The scripted instructions proceeded to explain how to perform the task. All participants performed a practice trial. As the participants were performing in the practice trial, the study administrator wrote the participants' names on the board, in numerical order of the computers at which they were sitting. After writing each name, the administrator verified with the participant whether her or his name was spelled correctly. After all participants completed the practice trial, they were provided the opportunity to ask questions regarding the task. Participants were then told: "Now we will start the competitive trials. ...The computer automatically keeps track of your time and provides an average time in the end. This average time will be what use to determine the winning placements and what I post on the board at the front of the room next to your name. Because I will be posting this at the end of all of the trials, it is possible to make up time you spend in one trial during another trial…" The study administer counted to three, and participants began the trials on "go".

In the first segment of trials, the platform was hidden in a fixed location; the platform stayed in the same place for all twenty trials. As each trial was completed, the program automatically proceeded to the next trial. The second vital sign readings were taken after any one of the participants had completed the first trial. As each participant finished the twenty trials, instructions appeared on the screen instructing the participant to "wait for instructions from the experimenter". At this time, the participant was given the SUDs questionnaire on a folded piece of paper and asked to fill it out. The study administer waited in the back of the room while it was completed, and then collected it.

After all participants had completed the SUDs questionnaire, the study administrator read the scripted instructions for the second segment of eight trials, during which the platform was visible. The same protocol was followed for this segment of trials, and after all participants completed the eight visible trials, the study administrator read the scripted instructions for the last trial. The participants were told that the last platform would be submerged, and not visible. The participants were instructed to look for the platform in the location it was initially, during the hidden platform trials. Participants were also told that this last trial holds more weight than the others in calculating the participant's final average time. Because this was the probe trial, in which there was no platform, all participants reached the maximum allotted time, which was 60 seconds.

Participants were then invited to follow the administrator and assistant back to the initial study room. Participants were asked to sit at their original desks, and were asked to complete a final questionnaire, which inquired about typical caffeine intake (including coffee, soda, tea, energy drinks and chocolate), computer and video game

experience, major and year in school, age and gender. Each participant was given a debriefing statement, and the opportunity to read it and to ask questions. Four to five minutes after participants sat down, the third and final blood pressure and heart rate readings were taken. Participants were then thanked for their participation and dismissed.

Stress measurements

To measure acute stress, five acute stress measurements were analyzed: systolic, diastolic blood pressure, heart rate, an acute stress questionnaire (the Subjective Units of Distress scale; SUDs), and acute stress sum (the sum of the former acute stress variables). The SUDs asks participants to rate their current level of anxiety on a scale of 0 to 100, with 0 representing a state of absolute calm and relaxation and 100 representing the greatest level of anxiety that the individual has ever experienced; that number is the participant's SUD score (Kaplan & Smith, 1995).

To measure chronic stress, five chronic stress variables were measured: resting heart rate, resting diastolic and systolic blood pressure, the average difference of diastolic-systolic readings throughout the study, a chronic stress questionnaire (the State–Trait Inventory for Cognitive and Somatic Anxiety; STICSA), and chronic stress sum (the sum of the chronic stress variables). The chronic stress questionnaire score is a single number representing the participant's stress level in general, with a higher number indicating higher stress. The STICSA questionnaire has been shown to have as high or higher correlation with measures of anxiety and a lower correlation with measures of depression than another well-published measure of trait anxiety (Gro¨s, Antony, Simms, McCabe, 2007). The STICSA is a 21 item scale used to

assess the participants' general current state of arousal with statements such as 'my heart beats fast', and 'my muscles are tense'. The STICSA asks participants to rate on a four-point Likert scale how true each statement is of her or him, in general.

The competition stress sum variable was created to reflect the collective contribution of stress variables that could uniquely contribute to the participant's stress level while she or he was performing in the competitive task. Competition stress sum is the sum of the chronic stress variables, average systolic and diastolic difference, chronic stress questionnaire, and the acute stress variables acute heart rate, acute systolic, and the acute stress questionnaire.

Performance measurements

Spatial navigation performance was measured with performance in the virtual Morris Water Maze (vMWM). The vMWM task is a computer task that displays a moving, computer-graphic visual of a water maze depicted from the perspective of a person submerged in the water. The participant navigated with the arrow keys to find the location of the submerged platform using spatial cues. Various virtual objects surrounding the pool served as spatial cues. The computer recorded quantitative measures of performance, including directional accuracy of navigating toward the platform and the time it takes participants to reach the platform.

Four performance variables were selected for analysis: mean time latency during hidden platform trials (MTLatHid), heading error average across hidden trials (HEavgHid), mean time in quadrant one across hidden trials (MTQuad1Hid), and mean percent of time spent in quadrant one during the probe trial (MTQuad1Probe). The objective of the task was to reach the platform in the shortest amount of time during each trial. Mean latency is the average amount of time it took the participant

to reach the platform during the hidden platform trials. Therefore, this variable is directly related to poor performance; the higher the mean latency, the poorer the participant's performance. Heading error is the average degree that the participant deviates from the direct pathway between the starting location and the hidden platform. In order to reach the platform in the shortest possible amount of time, the participant would need to navigate from his/her starting location directly to the hidden platform. Therefore, the degree of deviation from this direct pathway is considered to be directly related with poor performance. Performance in the probe trial was measured by the percentage of time the participant spent in the location of the pool they were told the platform was located. The mean time spent in quadrant one in hidden trials is directly related with good performance, as the platform was located in quadrant one during those trials. The mean percent of time spent in quadrant one during the probe trial is also directly related with good performance, as the participants were told that they should search for the platform in its location during the hidden trials, which was in quadrant one.

In summary, the variables mean time latency, and heading error are directly related with poor performance, whereas the variables mean time in quadrant one during hidden platform trials (MTQuad1Hid), mean time in quadrant one during the probe trial (MTQuad1Probe) are directly related with good performance.

Results

The two variables identified as positively related with poor performance (MTLatHid and HEavgHid) were found to have a significant, direct relationship with each other ($r = .738$, $p < .000$). Likewise, the variables identified as positively related with good performance (MTQuad1Hid, and PTQuad1Probe) were found to have a

significant direct relationship with one another (r = .466, p < .001). The good performance variables showed a significant negative correlation with both of the poor performance variables; MTLatHid and MTQuad1Hid (r = -.732, p < .000), and with PTQuad1Probe (r = -.374, p < .008); heading error and MTQuad1Hid (r = -.797, p < .000), and with PTQuad1Probe (r = -.367, p <.009). See Appendix B for performance variable correlations.

Hypothesis one stated that acute stress measurements will be positively related with chronic stress measurements. Two significant positive relationships were found to support this hypothesis. The acute stress questionnaire was positively related with the chronic stress questionnaire (r = .347, p < .05), and with resting heart rate (r = .371, p < .05).

The second hypothesis stated that chronic and acute stress combined will create a stronger relationship with performance than either chronic or acute stress alone. Neither the chronic nor acute stress sum variables produced significant relationships with performance. However, a combination of chronic and acute stress variables did produce a significant U-shaped, curvilinear relationship with the main performance variable, mean time latency. Mean time latency and competition stress sum had a negative linear relationship approaching significance (β= *-1.45*, p < .055), and a significant U-shaped relationship (F (2, 46) = 3.57, p < .05; Appendix A). With performance measured as mean time latency, and stress measured as competition stress sum, the best and worst performance is related with high stress, while moderate performance is related with low stress.

The third hypothesis stated that stress levels will be positively related with the time spent in the probe trial where the platform was most recently located (during the

visible platform trials). Acute systolic showed a positive linear relationship with percent time in quadrant one during the probe trial, that was approaching significance (β = .221, p < .057). Quadrant one was adjacent to the platform location during the visible trials, so (although only approaching significance), this finding contradicts hypothesis three.

Additional significant findings were the significant relationship between two physiological and questionnaire chronic stress variables, between two physiological and questionnaire acute stress variables, and higher blood pressure readings of video-gamers. The chronic stress questionnaire variable was positively related to resting heart rate (r = .302, p < .05), and the acute stress questionnaire score was positively related with acute systolic (r = .323, p < .05). Participants who reported to have played video games every day at some point in their lives had significantly higher resting systolic measurements (t (49) = 2.96, p < .005), third systolic measurements (t (49) = 3.58, p < .001), and a higher third diastolic measurement (t (49) = 2.82, p < .007). These participants also had a higher mean difference between systolic and diastolic measurements (t (49) = 2.76, p < .008).

Discussion

The positive relationships between the questionnaire and the physiological measurements provide support for the validity of self-report questionnaires. Because the chronic stress questionnaire (State-Trait Inventory for Cognitive Somatic Anxiety; STICSA) asks participants to report whether she or he frequently experiences fast heart rate, the positive relationship between the questionnaire and resting heart rate provides support for this questionnaire in particular.

The significantly higher blood pressure of video-game players is of interest, particularly because of the previously mentioned health implications of raised blood pressure, and because of the prevalence of video-game playing in our culture. One explanation may be that people who have higher stress levels choose to play video games in order to escape their real-world stresses. It may also be the case that higher stressed individuals are particularly attracted to the high intensity nature of video games. However, there are a myriad of alternative means to escape real-world stresses, such as music and art, and, likewise, many activities that are highly intense, such as sports. The physical activity of sports may reduce blood pressure (Perkins, et al., 1986) and provide protective neurotrophic factors that improve hippocampal function, and so the sedentary nature of video-game playing could account for all or part of these findings. It may also be that the continuous engagement in the high intensity mind-frame invoked during video game playing chronically increases stress levels. Given that many video games involve graphic warfare, simulating a flight-or-fight mindset, this seems quite plausible.

Hypothesis three was based on the theory that stress may impair performance by interfering with the development or access to a visual spatial map in short term memory, and/or that stress promotes a reliance on more recent visual memory. Higher stress did not appear to be related with time spent during the probe trial incorrectly searching for the platform in the more recent, visible platform trial location. Therefore, these results do not suggest that stress increases the influence of recent visual memory. However, this does not discount the possibility of a causal relationship between stress and visual memory. Stress may interfere with the

developing of a visual spatial map or accessing it from short term memory, but a relationship was not revealed in this study.

These findings provide support for hypothesis one, which stated that acute stress measurements will be positively related with chronic stress measurements. The positive relationships found between the acute stress questionnaire and the chronic stress questionnaire, and between the acute stress questionnaire and resting heart rate support hypothesis one, demonstrating that acute and chronic stress are not entirely independent.

Results supported hypothesis two; performance did not show a significant relationship with either the acute nor chronic stress variables exclusively, but performance did show a significant quadratic relationship with a combination of acute and chronic stress variables. These results suggest that both acute and chronic stress both make requisite contributions to the stress-performance relationship. The quadratic shape of the relationship suggests that there is a mediating factor involved in the stress-performance relationship. This study did not reveal a single variable that may influence high-stress individuals to perform either very well or very poorly. However, the absence of manipulation in this study suggests that the mediating factor in is related to a chronic stress characteristic of the individual.

One interpretation of this finding can be made within the context of the generality model of stress mentioned above. In this model, there is only one type of stress (Kemeny, 2003). Individual characteristics might determine whether stress is experienced, as well as the effect of stress on performance. It is possible that higher stress invariably aides a person in meeting a cognitive challenge. Whether stress is experienced may be determined by whether an individual perceives her or himself as

needing this performance aid. Using this premise to explain the stress-performance relationship in this study, the individuals who perceive themselves as needing this performance aid would comprise the high-stress individuals found in the best and worst extremes of performance. Whether high-stress results in very good or very poor performance might be determined by the ability of the individual in performing the task. A person who is incapable of performing well, would by definition, not perform well, regardless of whether she or he was aided by stress. A person who is capable of performing well, would exhibit enhanced performance with the aid of stress. The quadratic shape of the stress-performance relationship observed in this study fits this explanation. This interpretation aligns with Hans Selye's (1956) generalized stress response. This interpretation generates the question of whether stress levels change depending on whether the task is within the individual's ability, and in turn, how performance is affected. Subsequent studies could compare stress and performance for tasks of different difficulty levels.

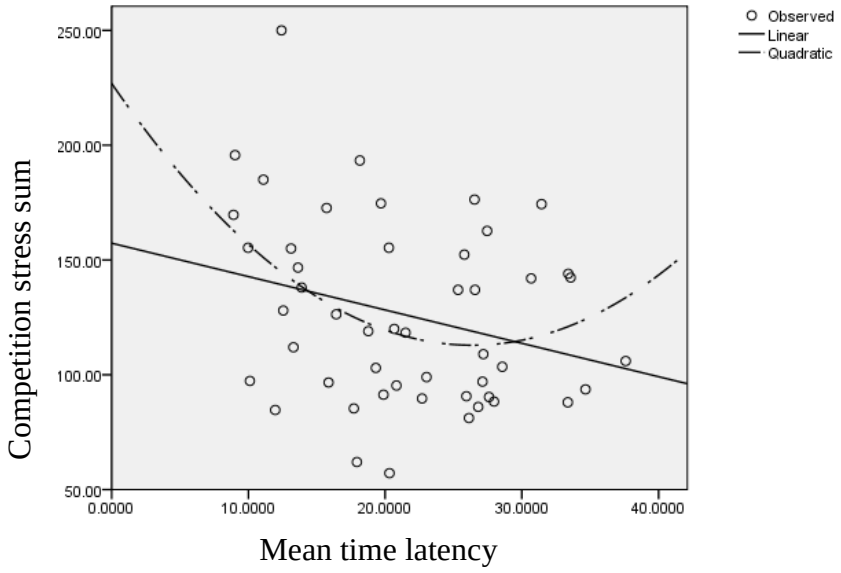
The quadratic stress-performance relationship found in this study can also be interpreted in the context of the modern generality model of stress, in which there is only one type of stress, but the effect of stress on performance is determined by a mediating individual factor (Kemeny, 2003). For example, an acute stressor may have the opposite effect on high chronic-stress individuals' performance than on low chronic-stress individuals' performance. This interpretation aligns with Ishiguro's study (1965) in which information of failure and personal reproach served as an acute stressor, and resulted in poor performance of individuals with high anxiety and good performance of individuals with low anxiety. While this study did not identify a chronic stress variable that determined whether stress would be a benefit or detriment

to performance, chronic stress measurements did provide an instrumental contribution to the significance of the relationship. Or, it may be that rather than a single determining individual factor, the relationship may depend on the interplay of acute and chronic stress. A subsequent study could measure additional individual characteristics, as well as other stress measurements to explore whether there is a single variable that is capable of explaining the relationship.

A third explanation of the stress-performance relationship found can be made in the context of the integrated specificity model of stress (Kemeny, 2003). This model proposes different types of stress, with individual characteristics determining which type of stress is invoked. In this interpretation, the type of stress invoked is determined by an individual characteristic, and performance is determined by the type of stress invoked. As previously mentioned, self-perception of task demands relative to self-perceived ability is an example of an individual determinant of stress type (Kemeny, 2003). Two types of stress may be highly anxious and fearing failure, versus highly motivated and anticipatory of success. To illustrate the stress-performance relationship in this study with these examples, individuals with highly anxious and fearful states of stress would constitute the poor performers and individuals with highly motivated and optimistic states of stress would constitute the very good performers. Subsequent studies could explore whether high-stressed, high-performance individuals experience a qualitatively different type of stress than the highly stressed individuals who perform very poorly. This could be investigated by including more qualitative psychological measurements, such as a questionnaire with scales related to fear of failure versus anticipation of success.

Appendix

Appendix A. Competition stress sum and mean time latency



Model Summary and Parameter Estimates

Dependent variable: Competition stress sum
Independent variable: Mean time latency (hidden trials)

Equation	Model Summary					Parameter Estimates		
	R Square	F	df1	df2	Sig.	Constant	b1	b2
Linear	.076	3.883	1	47	.055	157.304	-1.454	
Quadratic	.134	3.565	2	46	.036	226.886	-8.652	.164

Appendix B. Performance variable correlations

		Heading error average (hidden trials)	Mean time in quadrant one (hidden trials)	Percent time in quadrant one (probe trial)
Mean time latency (hidden trials)	Pearson Correlation	.738**	-.732**	-.374**
	Sig. (2-tailed)	.000	.000	.008
	N	50	50	49
Heading error average (hidden trials)	Pearson Correlation		-.797**	-.367**
	Sig. (2-tailed)		.000	.009
	N		50	49
Mean time in quadrant one (hidden trials)	Pearson Correlation			.466**
	Sig. (2-tailed)			.001
	N			49

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).

Appendix C. Stress variable correlations

		Average systolic and diastolic difference (chronic stress)	Resting systolic	Resting heart rate	Acute stress questionnaire	Acute systolic (systolic 2 – systolic 1)	Acute heart rate (heart rate 2 – heart rate 1)
Chronic stress questionnaire	Pearson r	.000	.125	**.302***	**.347***	.000	-.141
	Sig. (2-tailed)	.999	.387	**.033**	**.013**	.995	.329
	N	50	50	**50**	**50**	50	50
Average systolic diastolic difference	Pearson r			.049	.020		.020
	Sig. (2-tailed)			.732	.888		.887
	N			51	51		51
Resting systolic	Pearson r			.062	-.081		-.066
	Sig. (2-tailed)			.667	.573		.647
	N			51	51		51
Resting heart rate	Pearson r				**.371****	.127	
	Sig. (2-tailed)				**.007**	.373	
	N				**51**	51	
Acute stress questionnaire	Pearson r					.323*	-.232
	Sig. (2-tailed)					.021	.102
	N					51	51
Acute systolic (systolic 2 – systolic 1)	Pearson r						.115
	Sig. (2-tailed)						.420
	N						51

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).

Appendix D. Institutional Review Board approval letter

Date: Wednesday, January 05, 2011

NOTICE OF APPROVAL

TO: Margo Mitchell

DEPT: PSYC

PROJECT TITLE: The Relationship Between Acute Stress, Chronic Stress and Spatial Performance

SPONSORING AGENCY:

APPROVAL NUMB ER: 11-a044

The Institutional Review Board for the Protection of Human Participants has approved the project described above. Approval was based on the descriptive material and procedures you submitted for review. Should any changes be made in your procedures, or if you should encounter any new risks, reactions, injuries, or deaths of persons as participants, you must notify the Board.

A consent form:	[x] is	[ ] is not	required of each participant.
Assent:	[ ] is	[ ] is not	required of each participant.

This protocol was first approved on: 05-Jan-2011
This research will be reviewed every year from the date of first approval.

_____

Deborah Gartland, Chair
Towson University Institutional Review Board

Appendix E. Informed consent form

The aim of this study is to measure psychological affects, biological factors, and performance in the virtual Morris Water Maze. Your role in this project will consist of performing in a virtual navigation program on a computer screen. Your job is to perform at the best of your ability.
A person academically associated with Towson University will take readings of your blood pressure and heart rate before, during and after you perform in the computer program. These readings are non-invasive, and there are no known associated risks or discomforts. You will be asked to complete three questionnaires throughout the study.
Participation in this study is voluntary. You may decide to discontinue participation at any time, and you will not experience any consequences of your decision; you will still receive any academic credit(s) agreed upon when signing up for the study.
All information you provide will be anonymous. Although the descriptions and findings may be published, at no time will your name be used. All information obtained during the study will be kept separate from your name. You are at liberty to withdraw your consent to the experiment and discontinue participation at any time without prejudice. If you have any questions after today, please feel free to email Ms. Mitchell, mmitch6@students.towson.edu, or contact Dr. Debi Gartland, Chair, Institutional Review Board for the Protection of Human Participants at Towson University, 410-704-2236.

I, _____, affirm that I have read and understood the above statement and have had all of my questions answered.
Date: _____

Signature: _____

Witness: _____

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Education

Towson University, Towson, MD
Master of Arts, Experimental Psychology May 2011
Focus: Neuropsychology, Society for Neuroscience Member, 2009, 2011

Frostburg State University, Frostburg, MD
Bachelor of Science, Psychology; Minor, Fine Arts May 2007
Honors: Cum Laude, Psi Chi National Honor Society, Phi Eta Sigma
National Honor Society Chapter President and Scholarship Recipient, 2006,
Cultural Education Abroad Scholarship Recipient, 2005
Study abroad: Universidad Veritas, San José, Costa Rica, Spring 2006

Research experience

Towson University, Towson, MD
Masters thesis, Spring 2011
"The relationship between acute stress, chronic stress and spatial performance"
Blood pressure, heart rate, psychological stress, and spatial-navigation performance

Graduate researcher, Spring 2011
Cortisol hair analysis: competitive enzyme immunoassay using cortisol acetylcholinesterase conjugate

Graduate researcher, Spring 2009
"State and trait stress on virtual Morris Water Maze performance"
Galvanic skin response, blood pressure, heart rate and spatial navigation performance

Laboratory of Comparative Neuropsychology, *Laboratory Assistant,* Spring 2008
"Cognitive improving effect of the Phosphodiesterase Type 10 (PDE-10A) Inhibitor Papaverine in the Water Maze"
Cared for animals, maintained facilities, administered saline and drug injections, implemented water maze protocols

National Aquarium in Baltimore, Baltimore, MD
Marine Mammal Observer, Fall 2008—Summer 2009, Present
Took video, palm pilot, and written recordings of vocalizations, biological functions, social and maternal behavior of *Tursiops truncatus* (Atlantic bottlenose dolphins)

Marine Mammal Intern, Summer 2008
Prepared diets, facilities, observed and implemented select operant behavioral conditioning, monitored pregnancy behaviors of *Tursiops truncates*

Aviary Assistant Volunteer, Fall 2007
Upland Tropical Rainforest for bird species, *Choloepus didactylus* (two-toed sloths), and *Leontopithecus rosalia* (golden lion tamarins)

Work experience
Therapy Assistant to stroke survivor, Owings Mills, MD, Winter 2008—Present
Facilitated occupational and physical therapy, speech-language therapy, regaining control over daily activities

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