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The Temporal Nature of the Acute Stress Response and its Impact on Explicit Learning

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THE TEMPORAL NATURE OF THE ACUTE STRESS RESPONSE AND

ITS IMPACT ON EXPLICIT LEARNING

By

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DISSERTATION ACCEPTANCE STATEMENT

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An Abstract of the Dissertation Presented
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Acute stress is commonly experienced by many throughout their lives. Given the demanding lifestyle of many career paths, it's important to gauge the influence of these stressors upon cognitive performance. The present dissertation focus' upon explicit learning in attempts to explore one avenue of the stress-cognition relationship. The Trier Social Stress Test (TSST) was used as a lab stressor for Experiments 1 and 2, in which participants are asked to give a speech and complete a difficult math task in front of 2 evaluators trained to monitor non-verbal behavior. Experiment 1 investigates the dynamic stress response during the minutes following stress, and how changes in the physiological response influence cognitive task performance. Stress was measured cardiovascularly, hormonally and as a self-reported appraisal of the situation. Findings from Experiment 1 revealed a time point 55 min following stress in which participants' task performance was enhanced compared to a non-stressed comparison condition. These results suggest explicit task performance can be facilitated given a sufficient length of time following stress. Experiment 2 was designed in attempts to replicate the delayed RB task enhancement following the TSST, and given suggestions from the extant literature, explore if this task enhancement is attributed to enhanced working memory (WM). WM was assessed using an n-back task. Results confirmed the delayed RB task enhancement 55 min after

stress, however no effect was present for n-back task performance. Experiment 3 was designed to understand if the RB task enhancement extended for a number of hours following stress. Additionally, cold-pressor stress was used to assess if the delayed task enhancement was stressor specific. In this task, participants were asked to submerge their hand in ice-water for up to 3 min. Results revealed a marginal task enhancement following a similar delay as Experiments 1 & 2, however the enhanced task performance did not remain hours later. Taken together the present experiments suggest a time frame following a delay from stress in which explicit learning and more specifically RB category learning is enhanced, however it doesn't seem as if this effect is due to the impact of stress on WM.

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ABBREVIATIONS

APA	American Psychological Association
BLA	Basolateral Amygdala
BP	Blood Pressure
CO	Cardiac Output
CORT	Cortisol & Corticosterone
CPT	Cold-Pressor Task
CRF	Corticotropin Releasing Factor
DA	Dopamine
ECG	Electrocardiogram
GC	Glucocorticoid
GR	Glucocorticoid Receptor
HPA	Hypothalamic-Pituitary-Adrenal axis
HR	Heart Rate
ICG	Impedance Cardiography
LC	Locus Coeruleus
LD	Long-Delay
LTD	Long-Term Depression
LTP	Long-Term Potentiation
MAP	Mean Arterial Pressure
mPFC	Medial Prefrontal Cortex
MR	Mineralocorticoid Receptor
ND	No-Delay
NE	Norepinephrine
NS	No-Stress
OC	Oral Contraceptive
PFC	Prefrontal Cortex
RB	Rule Based
sAA	Salivary Alpha-Amylase
SAM	Sympathetic-Adrenal-Medullary axis
SD	Short-Delay
SGK	Serum- and Glucocorticoid-inducible kinase (SGK) processes
TPR	Total Peripheral Resistance
TSST	Trier Social Stress Test
WM	Working Memory
WPT	Warm-Pressor Task

CHAPTER 1

INTRODUCTION

In general, acute stress is thought to negatively impact everyday life, however this may not be entirely accurate. In regards to cognition, it has been argued that acute stress can have both adaptive and maladaptive aspects, depending on the type of memory recruitment (Ell et al., 2011; Schwabe et al., 2012; Schwabe & Wolf, 2013), and the time frame of the stress response (Diamond et al., 2007; Joels et al, 2011; Joels & Baram, 2009). Importantly, the consequences of stress may be, in part, characterized by the physiological response to stress and the dynamics of the post stress recovery. The initial physiological response to a stressful event tends to be strongest, and although this time frame has much significance, the lasting influence of stress has largely been ignored in cognitive research.

The present thesis takes a cognitive perspective, however the physiological response to stress will be extensively discussed as well. A major theme throughout this document will be the time-course beyond the initial response to a stressor, and I argue this topic has been mostly ignored. The primary research questions are designed to investigate physiological characteristics of stress over a period of min to hours following a stressor and the resulting cognitive ramifications.

The field of cognition is enormous and the present dissertation will target a specific sector, explicit learning. Explicit learning is broadly defined as a cognitive system enabling flexible control over actions in complex, unpredictable environments (Verneau et al., 2014). The actions reliant upon explicit learning are termed executive functions, which provide us with the ability to flexibly control our thoughts and actions (Miyake et al., 2000). Explicit learning is composed of attention, cognitive control and working memory (WM). Attention is operationally

defined as a set of processes that allow us to concentrate on one set of events in our environment while ignoring other events (Revlin, 2012). Cognitive control, can be further broken up into inhibition, or the ability to inhibit irrelevant information and selectively attend to goal-relevant information, and attentional-shifting, allowing people to flexibly shift between modes of thought (Miyake et. al., 2000). The overlap in definition between explicit learning and executive functioning clearly depicts the complementary nature of two processes. In addition explicit learning is reliant upon WM, defined as a limited capacity system involving both the on-line storage and updating of information (Baddeley 2012; Baier et al., 2010).

The extensive terminology may seem to increase the complexity of explicit learning, however sub-processes are not entirely exclusive from one another and should be considered together as an overall constitution of explicit learning. For example, WM requires a high level of attention and cognitive control as a prerequisite for successful performance. As many of the papers discussed will attempt to target selected aspects of explicit learning rather than the construct as a whole, each term is used throughout and will be operationally defined in the context of the described study.

Much of this document as well as the present experiments are focused upon rule-based (RB) category learning or the use of rules to best learn and classify information (Ashby et al., 1998). RB category learning relies heavily upon explicit learning, and indeed depends upon each of the sub-processes (cognitive control, attention, WM etc.). Present questions include: How is explicit learning influenced by stress? How will the time-course of the stress response play a role in this relationship? Does the type of stressful situation itself matter? The present dissertation will report on 3 experiments designed to answer these questions.

Before describing results from the present research, this document will introduce and discuss the pertinent literature surrounding stress and cognition. This section will discuss what stress is, how stress is measured, and how a stressor can be best designed for study in a research setting. Next, a section describing the influence of stress on cognition will be explored. Beginning broadly, this section will discuss notable findings in the field in general, before targeting the research most relevant to the present experiments. A final section will focus upon the time-course of stress and the important variability of this time frame in terms of effects on cognition.

What is Stress?

Stress can be defined in different ways depending on the context in which one is using the term. Originally the term stress was used by engineers to describe the amount of strain and force induced upon a structure. In the mid 1930's this term was borrowed by Hans Selye to describe situations provoking physiological changes in the body triggering what we now consider the stress response. Selye, a true pioneer, studied acute stress in rats following what he labeled as "nocuous agents" including cold exposure, surgical injury, spinal shock, and excessive exercise, as well as numerous drug induced responses (Selye, 1936). Commonly the term stress is used to represent the personal impact from situations one might experience throughout life. Given the excessive use of the word in many contexts, it is important to provide an operational definition of the term as it pertains to lab research. The present study will focus on acute stress, in which the time frame during and relatively soon after an isolated stressful event will be evaluated. An adequate acute stressor is powerful enough to trigger a physiological and psychological response and can affect cognitive performance.

Acute stress will be described in detail, however I must also give mention to a well developed literature on chronic aspects of stress, and although mechanisms are linked, the study and time-course of each are very different. Many of us experience levels of stress consistently over time given the high pressure environments of work and school. The experience of stress chronically can be described in terms of allostatic load defined as the wear and tear the body experiences from each stressful situation (McEwen, 1998). Allostasis, defined as the body's physiological adaptation to different situations, is different than homeostasis or the body's effort to keep at a constant static equilibrium (Juster et al., 2010). While considered adaptive during an acute time-frame, chronically elevated levels of allostatic load can have a diminishing effects on both the body's ability to perform cognitively, and recover from future stressful situations (Juster et al., 2010; Evans & Schamberg, 2009). The literature on chronic stress and allostatic load are very well established, and although it is not the focus of this dissertation, the extent to which this topic has been studied must be recognized.

The present dissertation will focus on the influence of acute stress. The study of acute stress is not novel, however the definition remains vague even within the field of psychology. Acute stress is defined by the American Psychological Association (APA) as:

> "The most common form of stress. It comes from demands and pressures of the recent past and anticipated demands and pressures of the near future. Acute stress is thrilling and exciting in small doses, but too much is exhausting. A fast run down a challenging ski slope, for example, is exhilarating early in the day. That same ski run late in the day is taxing and wearing. Skiing beyond your limits can lead to falls and broken bones. By the same token, overdoing on short-term stress can lead to psychological distress, tension headaches, upset stomach and other symptoms.
>
> Fortunately, acute stress symptoms are recognized by most people. It's a laundry list of what has gone awry in their lives: the auto accident that crumpled the car fender, the loss of an important contract, a deadline they're rushing to meet, their child's occasional problems at school and so on. Because it is short term, acute stress doesn't have enough time to do the extensive damage associated with long-term stress. The most common symptoms are:

- Emotional distress — some combination of anger or irritability, anxiety and depression, the three stress emotions.
- Muscular problems including tension headache, back pain, jaw pain and the muscular tensions that lead to pulled muscles and tendon and ligament problems.
- Stomach, gut and bowel problems such as heartburn, acid stomach, flatulence, diarrhea, constipation and irritable bowel syndrome.
- Transient over arousal leads to elevation in blood pressure, rapid heartbeat, sweaty palms, heart palpitations, dizziness, migraine headaches, cold hands or feet, shortness of breath and chest pain.

  Acute stress can crop up in anyone's life, and it is highly treatable and manageable.

This definition given by the APA is informative, however a more precise definition is needed for the present work. The framing of acute stress as more of a symptomatic psychological disorder rather than reaction to a situation will not suffice for much of the field's quantitative research thus I urge the reader to suppress both the APA's definition and any preconceived notion of the term. Rather for this context it is best to consider stress as both a physiological and psychological state that can have both harmful and beneficial qualities, often triggered by an acute event. The research on acute stress is extensive and well studied, and different disciplines view this subject differently. Within the psychology literature, the perspectives vary between sub-fields and I argue a combination of these perspectives will best explain the nature of the response and the effects of acute stress.

The number of catalysts capable of inducing stress is countless, but the stress response likely is not ubiquitous across species or even individuals. Originally Sonia Lupien and colleagues categorize a stressful situation as either "relative", described as a situation that will elicit a stress response in only a proportion of individuals and will vary depending on the particular individual, or "absolute", described as a real threat leading to a significant stress response in every person.

Lupien gives examples of absolute stressors such as confronting a dangerous animal, or being subject to extreme temperatures, and admits they are rare in everyday life (Lupien et al., 2007). The notion of an absolute stressor is arguable given the infeasibility of investigation, and it is possible all stressors are relative with a widely varying likelihood of inducing a stress response. For example although the presence of a wild animal may be stress inducing to almost all individuals, a small subset who are prone to this type of event may not yield the same response. Regardless of the distinction, the existence of relative stress suggests the reaction to stress may differ between individuals.

The concept of a relative stressor suggests the appraisal of a situation is subjective. Evidence provided from social psychology proposes the personal appraisal of any stressful situation precedes the stress response itself. Pioneers in attempts to unveil the process, Lazarus & Folkman provide a theory of appraisal suggesting the individual must first assess whether a situation is relevant to their own well being. If the situation is considered relevant, an assessment of one's individual resources to cope with the situations combined with the one's feasible options will be appraised (Lazarus & Folkman, 1984). If the assessment reveals one has the resources to cope with the situation, the resulting psychological state may be termed as a "challenge" (Blascovich, 2008; Dienstbier, 1989; Seery, 2011). Dienstbier argues a challenge response will not necessarily only have short term benefits, but over time will elicit long term "toughening", providing many beneficial outcomes and a gradual reduction of future physiological responses. The view that stress may be beneficial was initially fairly radical, however the notion that stress is not unitary was the basis for future psychophysiological research. It is important to note, in an original proposal of the "General Adaptation Syndrome", Selye suggests too that the response to a stressful situation can have beneficial actions in the short term, as he considers the response to be a defense to the present situation regardless of

the subjective appraisal. Selye however, in conflict with views of Dienstbier, argued that even if positive outcomes result from the stressors in the short term, large numbers of acute stressors over time will have a negative impact on the body eventually leading to disease (Selye, 1950).

As described, the definitions of stress and particularly acute stress vary between fields, and even within the field of psychology. To make the definition more tangible the next section will provide some background surrounding the stress response itself. The stress response will be discussed in terms of both a bodily psychological reaction, and transition to the measurement of stress in a research setting. The influence of stress on neural function will be described in detail in later sections.

Stress Response

During stress the body reacts in multiple ways, often allocating resources to best respond to the situation at hand. For example, activation of the cardiovascular system will increase blood flow to the brain and skeletal muscle, while activation of the respiratory system increases oxygen and prioritizes the lungs and brain. Concurrently the liver will work to release stored glucose utilizing energy when it is needed the most, and the adrenal glands will release hormones capable of binding to cells throughout the brain and body. Allocation of resources causes other systems to be suppressed such as the digestive and urinary systems which are less critical given the likely threatening nature of the current situation. This reaction to stress may be crucial depending on the nature of the current scenario, and the body's response should be considered adaptive as it aids the individual to endure and move past the situation.

The endocrine hormones released during stress are a distinguishing characteristic of the bodily physiological response. In reaction to a stressor, 2 separate axes are prompted. Activation of the primary axis, the Hypothalamic-Pituitary-Adrenal (HPA) axis, triggers a peripheral release

of cortisol from the adrenal gland. This system, although fairly quick to respond, is relatively slow in comparison to its counterpart, the Sympathetic-Adrenal-Medullary (SAM) axis. The SAM axis is extremely quick to respond, triggering a peripheral release of epinephrine into the blood, ultimately generating vagus nerve stimulation and initiating a central release of NE (Roozendaal et al., 2007; Roozendaal & McGaugh, 2011; Schwabe et al., 2012). This system is not only quick to act, but just as quick to recover after culmination of the stressful situation. Given the varying time-courses of these physiological systems, it is important to understand how resulting hormone release can affect cognition (Diamond et al., 2007; Joels & Baram, 2009). More on this topic will be discussed throughout.

The stress response itself has been well established and can be provoked by numerous situations, in particular social situations (Kirschbaum et. al., 1993), physical events (Schwabe et al., 2008b) and even exercise (Brenner et al., 1998). Early stress research suggested the stress response differs depending on the controllability a human has over the situation (Glass et al., 1971). A similar finding in rodents tested this by manipulating the capability of rats to escape a stressful shock. Rats in the stress group did not have the capability to escape the stressful shock, and hence did not have control over the situation. Results revealed decision making performance differences between the stressed rats and controls (Minor et al., 1984). These early studies illustrate a now well accepted view thanks to a vast meta-analysis authored by Dickerson & Kemeny (2004). This paper explores in depth the specific elements of a stressful situation that elicit an HPA response in humans. First, in line with the aforementioned research, findings reveal the strongest psychological stressors include a feeling of "uncontrollability" in which "participants are unable to avoid negative consequences or cannot succeed despite their best efforts". Second, the strongest psychological stressors include a feeling of "social evaluative threat" found to be present "when an important aspect of the self-identity is or could be

negatively judged by others", and it is proposed that "social-evaluative threat is most likely to occur when failure or poor performance could reveal a lack of a valued trait or ability" (Dickerson & Kemeny, 2004).

Results from the previously mentioned meta-analysis suggests a specific type of lab stressor will result in the strongest HPA response. In particular the Trier Social Stress Test (TSST; Kirshbaum et al., 1993) incorporates both the elements of uncontrollability and social evaluative threat in most individuals. In this manipulation, participants are directed to sit in a room alone and prepare a 5 minute speech they will be giving about themselves in front of a camera and panel of evaluators trained to monitor their non-verbal behavior. The evaluators are not actually assessing non-verbal behavior but instead are trained to hold a flat affect providing limited neutral feedback to the participant throughout the speech. Additionally, the evaluators are given a clipboard with notes, and instructed to give the impression they are evaluating the participant's unconscious body language. The speech often entails the participant explaining why they would fit for a position in their ideal job, discussing both strengths and weaknesses. After completion of the speech the participant is then instructed to complete a serial subtraction task for 5 min, starting over after every mistake. The evaluators again provide an impression of evaluation during the subtraction task. The TSST has been shown to induce both an HPA and SAM response (Kirschbaum et al., 1994; Rohleder et al., 2004), and has been widely used to induce stress throughout the field.

The questions posed in the present study encompass acute stress in general, and are likely not exclusive to the TSST. As described above social stressors encompassing each of the stress inducing elements are sufficient to elicit a stress response, and because of this they are most commonly used in psychological laboratories. The value of the TSST is undeniable,

however sustaining a level of deception can be difficult and the expense in terms of time and labor can be quite high. Schwabe and colleagues developed an alternative version of the very commonly used cold pressor test (Hines & Brown, 1936), to abate some of these issues while attempting to retain the physiological response to stress in a lab setting. This alternative CPT is similar to the original physical stressor with the addition of social evaluation (Schwabe et al., 2008b) and relabeled as the socially-evaluated CPT. In this stressor, participants must submerge their hand and arm in ice water for up to 3 min, while being recorded on video and evaluated by an experimenter with a white coat and clipboard. Results reveal a similar stress response to the standard TSST in terms of cardiovascular and endocrine reaction (Schwabe et al., 2008b). It is not fair to call this a purely physical stressor given the addition of social evaluation, however considering the experience itself is much different, using the CPT provides researchers with a legitimate lab stressor to extend findings associated with the TSST. The present work will take advantage of both the TSST and socially evaluated CPT as a way to induce laboratory stress.

Despite the differences in stress definitions, the response itself to particular situations has been well studied. The two stress axes discussed above provide a blueprint for the physiological response associated with acute stress. Despite the subjectivity of stress appraisal, lab stressors have been designed and empirically tested to include elements most often resulting in a significant HPA response in humans. The specifics of the lab stressor are imperative for comparison purposes across studies, and provide reliable endocrine responses important for the stressful influence of neural mechanisms which will be discussed in detail later. The next section will provide details about the psychological and physiological measurement of the stress response used within psychological research.

Stress Measurement in Research

The stress response can be measured and classified in numerous ways depending on the research question and the stance of the researcher. Stress can be measured as a physiological state in terms of bodily arousal, or in humans, the subjective appraisal of stress can be assessed. The latter is often measured as a self reported statistic in order to gauge a subjective psychological state. The research questions should be a large contributing factor in this decision of how to measure stress. The following section will discuss and critique different avenues of stress measurement and how they are applied to research in psychology.

The simplest option to assess stress is to gauge the appraisal by self-report. Simply asking the participant how stressed they are or were during a situation is easy and cost efficient, however assessing the relationships between the perception of one's stress and the physiological response to the situation have yielded inconsistent and often null results (Dickerson & Kemeny, 2004; Hellhammer et al., 2009; Kudielka et al., 2009; Schommer et al., 2003). These inconsistencies would suggest perceived stress and the physiological response to stress are not one to one, however it is unclear whether there is an explanation for this difference or if there is something inherently different about the two constructs. Given the contrasting definitions of stress between researchers and the lay person, it may be fair to question the harmony of self-reported and physiological stress measures. An experimental paper by Schlotz and colleagues (2008) suggests this difference may be due to methodological standards. Consistent with the literature, Schlotz et al. did not find a correlation between endocrine responses online with self-reported stress appraisals, however they did find a time-lagged association revealing appraisals online during stress correlated with endocrine response 10-20 min later (Schlotz et al., 2008). The authors urge stress researchers not to discount the

measurement of subjective psychological stress, but to instead keep the timing of measurement in mind during study design. Despite this novel suggestion, I choose to err on the side of caution in linking the appraisal of stress with the physiological response until additional data can further solve this puzzle. In the present research the self-appraisal of stress will be considered a separate psychological construct from the physiological stress response, however both may contribute to the overarching research question.

The cardiovascular response adds a new element of arousal beyond the psychological stress measured from self appraisal. First, with the correct equipment the cardiovascular response can be monitored during most experimental manipulations and tasks, and can be continually measured over time. This freedom of measurement can be extremely valuable as compared to self-reported and endocrine measures that can only be assessed periodically. With this technology researchers are able to monitor a physiological response before, throughout, and after the stressful experiences and can obtain a full time-course of the reactivity. Heart rate (HR) is a common cardiovascular measurement in psychological labs and is considered by many as a bi-product of an aroused state, and not to be associated with all definitions of a stress response (Blascovich et al., 2008). HR is often associated with sympathetic nervous system activity however, research suggests HR can be likewise influenced by the parasympathetic nervous system (Glick & Braunwald, 1965). The parasympathetic nervous system acts to slow HR consistently having a contribution that varies beat to beat (Akselrod et al., 1981). Given the influence from multiple sources, using HR as a measure to help define a particular stress response may be difficult. For example an increase in HR does not ensure activation of the HPA axis and in many cases no association between the HPA axis and HR are found (Schommer et al., 2003). Schommer and colleagues found elevations in endocrine as well as HR responses following the TSST, regardless of how many times participants had already experienced the TSST

previously. Importantly HR reactivity differences were absent when the data was split according to cortisol response, revealing many cases in which participants had significantly elevated HR without an HPA response. Similarly, the absence of an association between HR and a SAM endocrine response is commonly found (Nater et al., 2005; Schommer et al., 2003) revealing discrepant characteristics of the endocrine and cardiovascular systems.

Comparing different cardiovascular measures is not straightforward seeing as they are not mutually exclusive from one another, and this can be especially highlighted in BP collection. For example, increased HR indirectly influences BP via elevations in blood flow, however BP can also be influenced by levels of vascular and arterial resistance. The collection of BP as an indicator of SAM is controversial, however has been reported previously (Elzinga & Roelofs, 2005). The cardiovascular response to a stressor can vary depending upon one's appraisal of the situation (Blascovich et al., 2008, Dickerson & Kemeny, 2004; Lazarus & Folkman, 1984), and therefore some argue BP should be analyzed in combination with other vascular measures rather than defining a particular physiologically aroused state alone (Blascovich et al., 2008; Seery et al., 2011). Given the uncertainties in terms of a particular stress response, it may be best to remain hesitant in using BP reactivity definitively. BP however, like HR, is a reliable measure of arousal, thus in the present study HR and BP will be measured to gauge the cardiovascular responses as indication of physiological arousal.

In addition to arousal, cardiovascular measures have been used to determine individual differences in the stress response, as described previously. In particular the challenge response has been studied in depth in terms of cardiovascular reaction. According to the biopsychosocial model (BPS) introduced by Jim Blascovich, the psychological state of challenge results in a specific physiological response, in particular sympathetic nervous system activation.

Cardiovascularly, challenge is defined by increases in heart-rate (HR), and cardiac output (CO), or the volume of blood being released from the heart, with a decrease in total peripheral resistance (TPR), a measure of blood vessel constriction (decreases in TPR represent relatively dilated arteries; Blascovich, 2008). Antithetical to challenge is the "threat" response (Blascovich, 2008) provoked during a situation in which one feels they do not have the resources necessary to cope with the current situation (Lazarus & Folkman, 1984). A situation inducing threat may exemplify the more traditional or layman's definition of a stressful situation. During threat both SAM and HPA are activated and characterized by the BPS with a similar increase in HR but a decrease in CO and increase in TPR (Blascovich, 2008). Although challenge and threat have clearly defined profiles, they are not considered dichotomous, but instead a continuum anchored by each state (Seery, 2011). Viewing stress in this way allows researchers to compare the challenge and threat cardiovascular profiles in relative terms rather than defining any particular individual's response. Given that challenge and threat are considered in relative terms to one another, analyses most often assess relationships between the cardiovascular responses with other variables as a way to determine the influence of the cardiovascular response within a particular sample.

These measures provide researchers with another option beyond cardiovascular arousal. The profiles of challenge and threat are meant to help clarify a spectrum of cardiovascular response, and should always be considered in relation to one another. The cardiovascular response can be a useful online measurement tool and quite informative, however a stronger portrayal of the separate physiological stress axes can be measured with the endocrine response. Despite the advantages of measuring CO and TPR to assess the presence of individual differences, they cannot provide much for research questions surrounding the endocrine response. Given the present research questions are not designed to investigate

individual differences in the stress response, cardiovascular measurement will be restricted to assessment of arousal using HR and BP. Endocrine measures will help to decipher the nature of the response for the present experiments.

Cortisol, the hormone released from the adrenal gland during high levels of stress, is the ultimate product of HPA axis activation. Cortisol in humans is the homologue of corticosterone in rodents, and from this point forward both will be referred to as CORT. Traditionally, both free CORT, or CORT that is unbound to binding proteins, and total CORT, that is a combination of CORT bound to binding proteins and free or unbound CORT, have been measured from blood plasma. The debate between total CORT and free CORT is known as the free hormone hypothesis, and remains ongoing today (Ekins, 1992; Levine et al., 2007). Regardless of the of serum collection method, strong correlations with free salivary CORT have been found. The relationships between salivary free CORT with total CORT (between r = .71 and .96; Kirschbaum & Hellhammer, 1994) and free CORT in blood serum (Kirschbaum & Hellhammer, 1989) provides support for the collection of free CORT in saliva. Furthermore regression analyses from an extensive meta-analysis by Dickerson and Kemeny (2004) revealed the type of collection (plasma vs. saliva) did not predict an effect size of peak CORT response, inferring no quantitative advantages between one collection method over the other (Dickerson & Kemeny, 2004). The combination of sufficient strength and extreme ease of CORT collection within saliva has led researchers to embrace this collection method in the field (Hellhammer et al., 2009; Kirschbaum & Hellhammer, 1994).

Saliva must be collected at discrete time points which may be considered a disadvantage relative to cardiovascular assessment, however the nature of the CORT response has been well established. In the aforementioned meta-analysis looking at 208 lab studies of

salivary CORT collection following acute psychological stressors, authors Dickerson and Kemeny unveiled much about the relationship of the stressor and the CORT response. As discussed earlier, this study revealed the most important factors in triggering a CORT response include social-evaluative threat, and the feeling of uncontrollability. Analyses revealed stressors that involved both public speaking and a challenging cognitive task combination in the presence of social evaluation resulted in the strongest CORT response. Moreover, peak CORT levels were found between 21 and 40 min following the onset of the stressor. Notably there was no relationship between the duration of the stressor and the CORT response allowing authors to conclude "Shorter stressors, with the proper eliciting conditions, are equally as effective as longer tasks in increasing CORT levels" (Dickerson & Kemeny, 2004). It should also be noted that as comprehensive as this study is, the authors did not include any physical stressors (ex. Cold-Pressor task, exercise), so these results are restricted to social stress.

All cognitive processes rely upon brain functioning therefore it is important to acknowledge the relationship between the CORT release in the periphery, and how that compares to CORT levels in the brain. Droste et al. (2008) provided a comparison between CORT levels found in blood plasma and brain tissue, specifically the hippocampus and striatum following a glucocorticoid injection in male rats. A 20 minute delay between peak CORT increases in brain tissue and blood plasma was found, suggesting that although a bodily reaction to a stressor may be rapid, the process of reaching the brain is sluggish (Droste et al., 2008). Given that the results reveal a difference between the stress response and the time until CORT reaches the brain, using a lengthier stressor may be beneficial to allow CORT sufficient time to reach the brain if the goal is to assess performance directly following the stressor. These results propose the time-course of CORT's effects on the brain should not be directly compared to the

results found in saliva or blood and are important to consider even if the data being collected is exclusively behavioral.

With a great surge of CORT collection in stress research it is important to understand any factors that may influence the HPA response. A review from Kudielka and colleagues (2009) did just this in order to provide suggestions for improving experimental methodology throughout the field of stress. The authors reviewed the impacts of: age, gender, steroid supplements, pregnancy, lactation and breast-feeding, nicotine (acute & chronic), caffeine, alcohol, genetic factors, time of day, stressful early life experience, subjective stress, chronic stress, medication (psychoactive and anti-depressant drugs). Overall, the authors conclude that each of these factors play some role in the HPA response and if possible they should be controlled for or excluded, but they especially recommend that both time of day and gender be taken into strong consideration (Kudielka et al., 2009). The diurnal rhythm of CORT in humans presents a spike soon after awakening with a slow decay throughout the day (Nater et al., 2007; Roheleder et al., 2004), suggesting the absolute CORT levels will be higher following stress in the morning than the afternoon. Importantly, even with these absolute CORT differences throughout the day, the net salivary CORT response, or CORT reactivity, was not found to be different in the morning than the afternoon (Kudielka et al., 2004). All things considered authors recommend keeping time of testing consistent regardless what time of the day is chosen. In terms of gender, authors suggest restricting the sample to males is acceptable given differences driven by female hormone cycles and the use of oral contraceptives (OC), however they advocate females should be included whenever possible for stronger and more complete results. If including females, it is best to exclude women taking OCs and include only women in the Luteal phase of their menstrual cycle for the most consistent results (Kudielka et al., 2009).

Women in their luteal phase who are not taking OCs have shown equivalent CORT responses to males (Kirschbaum et al., 1999).

Given the advantages of measuring HPA activation via salivary CORT, one might expect it similarly advantageous to measure SAM activation via salivary norepinephrine (NE) or epinephrine. Unfortunately this turns out to be relatively difficult because the transfer from blood to saliva takes about an hour (Kennedy et al., 2001), making it hard to asses changes due to stress. Fortunately there is another option; The sympathetic nervous system that triggers SAM also results in salivary protein secretion, and specifically salivary alpha-amylase (sAA; Chatterton et al., 1996). sAA is responsible for 10-20% of the total salivary gland produced protein (Nater et al., 2005) and is responsible for the initiation of digestion in the oral cavity (Scannapieco et al., 1993). sAA increases have been shown following the TSST (Nater et al., 2005; Rohleder et al., 2004) and CPT (Smeets et al., 2008) and similar response patterns as well as positively correlated increases have been shown between plasma NE and sAA following the TSST (Rohleder et al., 2004). A primary study in the assessment of sAA, also found significant correlations between sAA and both plasma NE ($r = .64$) and epinephrine ($r = .49$) following exercise (Chatterton et al., 1996). Taken together, these results, as well as extensive reviews of the literature (Nater & Rohleder, 2009; Rohleder et al., 2004), provide strong support for the use of sAA as a measure of SAM activity in human research.

To conclude, multiple quality options are available for assessing stress in psychological research. There are advantages and disadvantages to each measurement, and the correct choice will depend on the research question. The different approaches to measuring the stress response do not always, and actually often don't depict one other. On the surface this may seem like a large issue, and indeed it complicates the integrality of research using each approach,

however it may also be argued that the breadth of techniques is advantageous in that numerous tools are available to best answer the particular research question. For example if a researcher is interested in measuring the subjective response to a particular stressor, they may choose to examine the deviation of a cardiovascular reaction within a group or subjective appraisal of the stressful experience. On the contrary, if the goal is to measure stress in terms of magnitude or a particular active stress axis then endocrine collection may make the most sense. For the present experiments I've chosen to include multiple stress response measures to gauge the psychological, cardiovascular, and endocrine responses to a stressor.

Stress Effects: Learning and Memory

The impact of stress on cognition is pertinent to the lives of many across all industrial and academic environments. Given the number of humans who experience stressful events in their everyday lives it is a bit surprising the literature is so young. Fortunately in recent years research in this area has increased and has begun to provide us with some insights into the topic. The following section reviews the relevant literature describing the effects of stress on memory as a general preface before explaining the research especially relevant to the present experiments.

It is well known that stress can have an impact on memory, but given that learning and memory involve multiple stages it is important to decipher which stages of the memory are affected. This section will discuss the impact of stress on three major stages of memory: memory encoding, or the initial formation of memories, memory consolidation, studied as the strengthening or at least persistence of a memory, and memory retrieval, or the recall of information previously stored in memory. Each stage of this process occurs separately, therefore the influence of stress can be studied by adjusting timing of the stressor relative to

cognitive assessment. These processes each contribute to the memory of information, yet they are all unique and thus will be discussed separately in terms of stress.

Memory Encoding

First, memory encoding, or the initial learning of information may be affected by stress. To study the influence of stress on memory encoding researchers induce stress prior to learning. The literature of stress and memory encoding has been inconclusive thus far, providing data to support both impairing (Diamond et al. 2006; Elzinga et al., 2005) and enhancing (Domes et al., 2002; Schwabe et al., 2008a, Smeets et al., 2007) effects of stress. One reason for this discrepancy may be the valence of the encoded information. It has been hypothesized that the encoding of emotional materials during stress may be more reliant upon the amygdala as compared to hippocampus for neutral materials (Payne et al., 2007), however this has yet to be tested. Schwabe and colleagues (2008) tested recall of positive, neutral, and negatively valenced words both 1 and 24 hrs following encoding. Participants were randomly assigned to either the CPT or a warm water control (WPT) and then provided a list of 18 words to remember. Neutral words were recalled significantly better both 1 and 24 hrs following the CPT than control, but recall of negative words was enhanced only at 1 hour post stressor, and no differences were found for positively valenced words. Overall this study suggests an enhancement in memory encoding following a stressor, however findings surrounding the valence of the encoded information may play a role in this effect. Similarly, Payne and colleagues designed a study incorporating the emotionality of information encoded following the TSST. In this study participants were shown a narrated slideshow depicting a story about a car accident including both emotional and neutral information. Participants returned to the lab 1 week later to complete a series of recall tests. Overall recall was enhanced for emotionally arousing materials,

and impaired for neutral materials following the TSST. Taken together, although methodologically different, the studies highlight the conflicting pattern of results present in the literature for memory encoding and particularly in terms of the valence of the stimuli. It is conceivable the difference in study design led to the conflicting results, however the role of valence in the stress and memory encoding relationship remains ambiguous.

If not valence, another possible factor in the stress and encoding literature is the relationship between the stressor itself and the encoded information. Originally proposed in an opinion piece from Joels and colleagues in 2006 (Joels et al., 2006), Smeets et al. (2007) set out to examine this relationship in humans. To associate the task and the stressor, the researchers modified the speech component of the TSST to include either a memory related speech or a personality related speech, and compared each group to a non-stressful control group. The authors predicted performance on a later verbal recall task would be improved if the participants had to remember words related to the topic of the speech during the stressor. Participants were assessed on recall of memory-related and personality-related words 24 hrs following the stressor and encoding sessions. Results revealed that personality-related words were recalled better for participants experiencing the personality version of the stressor as compared to both the memory version of the stressor and controls, and furthermore CORT responses to the stressor were significantly and positively related to performance in this group. No differences were found between groups in recall of the memory related words. Results assessing the relationship between the stressor and the encoded information were mixed providing some support for a link between recalled information and the context of the stressor, however more research is needed to fully understand this relationship.

Overall the impact of stress on encoding remains unclear. Findings are conflicting and although some support exists for potential factors mediating this relationship, conclusions should be taken with caution. A meta-analysis by Het et al. (2005) explored the influence of CORT on different types of human memory and similarly concluded that there are mixed results with memory encoding (Het et al., 2005). The authors suggest possible influential factors including the time of day, and dosing discrepancy issues, however with the limited literature they also state no overall conclusion. An area of greater agreement and stronger research is that of stress' influence on memory consolidation and retrieval.

Memory Consolidation

The influence of stress on memory consolidation is most commonly tested by inducing stress following the encoding of information and assessing later recall of the already learned information. In general stress has been shown to enhance memory consolidation (Barsegyan et al., 2010; Cahill et al., 2003; Roozendaal et al., 2006; Smeets et al., 2008). Similar to encoding, the consolidation of information under stress has been studied with stimuli of different valence. Results are more straightforward revealing enhanced memory consolidation of emotionally arousing stimuli following a CPT stressor than control (Cahill et al., 2003). Neuroscientific findings provide support for the function underlying this result, revealing enhanced memory consolidation following stress is reliant upon the amygdala (Roozendaal et al., 2009). Results from Roozendal et al. (2009) reveal that inhibition of noradrenergic basolateral amygdala (BLA) receptors negates a CORT driven memory consolidation enhancement in rats (Roozenaal et al., 2006). Additionally, the BLA has been shown to interact directly with the mPFC during this process (Roozendaal et al., 2009) and furthermore requires a combination of CORT and NE within the mPFC (Barsegyan et al., 2010). Research from Smeets et al. (2008) are in line with the

stress-consolidation enhancement finding revealing an association between enhanced performance and both salivary CORT and sAA following stress in humans (Smeets et al., 2008).

Memory Retrieval

Resembling the impact of stress on consolidation, the relationship between stress and memory retrieval is also relatively conclusive. To study the effects on retrieval, researchers most often induce stress just prior to or during recall of previously learned information. The impact of stress on retrieval is related to situations common in everyday life, for example students trying to recall information during an exam, or a corporate professional presenting information to a large audience. In general stress impairs memory retrieval (Dominique et al., 1998; Roozendaal et al., 2003; Schwabe & Wolf, 2009; Smeets et al., 2008). In a compelling study published in the journal Nature (Dominique et al., 1998), Dominique and colleagues found both a foot shock stressor and CORT administration impaired retention of spatial information in rats, however this impairment was abolished when CORT synthesis was suppressed. To augment this behavioral finding Roozendaal and colleagues investigated the brain structures involved. Using a Morris water maze to study the retention of the spatial location of a learned platform, the researchers provide data suggesting the administration of a GR agonist within the hippocampus impairs platform location retention 24 hrs after platform location training as compared to a group administered a vehicle infusion. These results suggest CORT actions in the hippocampus play a role in the impairment of memory retrieval from stress. Importantly, a group receiving the exact same treatment infusion regimen within the BLA were not impaired relative to a vehicle infusion, however animals who received a lesion to the BLA 1 week before training followed by the previously described GR agonist administration within the hippocampus, were no longer impaired on the spatial location task (Roozendaal et al., 2003). These findings suggest the role of

the hippocampus in stress-mediated impairment of memory retrieval relies upon BLA functioning. A follow-up study from this group replicated the impairments found from the GR agonist, and further found administration of a β-adrenoceptor antagonist administered to either the Hippocampus or BLA was sufficient to attenuate the glucocorticoid (GC) induced impairment (Roozendaal et al., 2004) suggesting the necessity of both CORT and NE in this process. Taken together these results indicate the hippocampus as a location of interest in mediating the stress related effect on memory retrieval, however, the BLA seems to be indirectly involved in this process, and moreover the impact of this structural synergy seems to be mediated by a combination of CORT and NE influence.

The impairments in memory retrieval following stress have been found not just in rodents, but humans as well. As described previously, Smeets et al. (2008) also assessed the influence of a stressor on the retrieval of information 24 hrs after encoding. As opposed to their memory consolidation enhancements found following a stressor, and in line with the rodent literature, humans were also impaired on this task when memory retrieval of word lists was tested just after the CPT as compared to controls (Smeets et al., 2008). This finding suggests the impairments in memory retrieval following stress are present across species

The findings provided above reveal the nature of stress' influence on memory, and the factors and neural processes likely playing a role. To summarize, the impact stress plays on memory encoding is inconclusive, however more clearly demonstrated is the influence of stress on memory consolidation and retrieval. In particular, evidence suggests stress enhances memory consolidation and impairs memory retrieval. It should be noted that many of the rodent papers discussed in the present document and elsewhere, use fear driven manipulations as a way to induce stress. For example, researchers may induce stress by forcing a rodent to

swim in deep water with no assurance of finding safety. It is possible the physiological response to a stressful situation may differ depending on whether or not the situation causes fear for one's safety, and thus should be considered when comparing animal and human studies of stress. The review of these findings provides us with some important themes revealed within the stress cognition relationship. First, the timing of stress relative to task assessment is crucially important. Second, the relationship between stress and cognition is not unitary, as shown through the opposite effects of stress on consolidation and retrieval. These two themes will provide a basis for present hypotheses, and will be discussed throughout. The next section will shift to reviewing the relationship between stress and explicit learning in order to target processes underlying the tasks from the present experiments.

Stress Effects: Explicit Learning

Central to the cognitive literature is the type of learning impacted by stress. In general evidence suggests a declarative or explicit learning system, reliant upon processes such as attention, cognitive control, and WM, will be negatively impacted by stress (Arnsten, 2009; Barsegyan et al., 2010; Elzinga et al., 2005; Oei et al., 2006; Plessow et al., 2012a; Plessow et al., 2012b; Schoofs et al., 2008). This explicit system can be contrasted with an implicit system that uses procedural learning. The implicit system is slow as it takes time to learn the task, and given this, the explicit system is expected to be initially relied upon. Moreover, the theory assumes that both systems are in constant competition for control of cognitive resources with one another, and the implicit system may overtake control from the explicit system if it proves superior for the current task (Ashby et al., 1998). During stress it has been presumed that a premature or unnecessary shift to the implicit system may take place even if the task can be sufficiently achieved via explicit processing (Ell et al., 2011; Schwabe & Wolf, 2012). A premature

shift may ultimately diminish performance if the implicit system is sub-optimal or requires too much time to succeed. In order to understand more about the explicit system performance under stress, the following section will focus upon the influence of stress on sub-processes that construct explicit learning, and touch upon research suggesting the potential for a sub-optimal system shift. Much of the research discussed will be selected from the relatively abundant stress-WM literature. Exploring the effects of stress on WM will allow for a more thorough explanation of the biological mechanisms and time-course of the stress response.

Explicit learning is mediated by multiple cognitive processes, often explored separately in the context of stress. Two papers from Plessow et al. in 2012 targeted the relationship between stress and cognitive control processes. The authors define cognitive control as "a variety of processes that ensure successful goal attainment by incorporating both intentions and context conditions at all times". In the first of these studies, participants displayed larger error-related switch costs when shifting between relatively simple categorization tasks following the TSST as compared to non-stressed controls. In these tasks participants were either asked to categorize a single digit number as higher or lower than 5, or they were asked to categorize a number as odd or even. At the beginning of each trial a cue was given indicating which of the two responses should be made. When the current trial type was a repetition of the previous trial type results were equivalent between the stressed group and controls, however when the current trial type was a shift from the previous trial type performance was impaired under stress as compared to controls. Authors concluded that this detriment in shifting attentional resources towards the current goal indicates impairments in cognitive control processes following a stressor (Plessow et al., 2012a).

In a second study from Plessow and colleagues, cognitive control processes were again impaired under stress, this time examining "task shielding" by measuring the interference from a task that was not to be prioritized. In this experiment participants were asked to categorize two sets of stimuli appearing on the screen, one with a right hand key press and one with a left hand key press. They were instructed to prioritize one set of stimuli before categorizing the other, and because the stimuli from each set were displayed together, the ability to prioritize one set in the presence of another was defined as task shielding. Results revealed task shielding performance was worse following the TSST as compared to controls, again suggesting stress impairs cognitive control processing (Plessow et al., 2012b).

The present experiments explore the impact of acute stress on RB category learning. Commonly in RB category learning tasks, categories are learned progressively on a trial by trial basis and typically optimal performance strategies can be easily verbalized (Ashby et al., 1998; Ashby & Maddox, 2011). As previously mentioned, RB category learning tasks rely on selective attention, cognitive control, and WM resources (Ell et al., 2009; Waldron & Ashby, 2001; Zeithamova & Maddox, 2006). The category learning literature contributes a strong understanding of the underlying neural substrates (Ashby et al., 1998), providing reliable predictions in the context of stress. Furthermore, RB category learning tasks provide an opportunity to assess strategy via well established modeling techniques (Ashby, 1992). These models supply the researcher with tools revealing the likely strategy used during the task, thus providing a deeper level of analysis than task accuracy alone.

Even given the well established categorization literature, the study of stress and RB category learning is scarce. Recently in a study from McCoy et al. (2013), RB category learning was assessed for participants subjected to a high pressure situation. To induce pressure the

participants were told they and a partner were completing a categorization task (this partner was not real), and a monetary reward would be earned by both partners if they each reached a criterion level of accuracy on the task. Just prior to the final block of the task participants were informed their partner had completed and accomplished the task and reached the necessary performance goal, shifting all of the pressure on to them to earn the reward for both themselves and the partner. Notably, relative to the TSST, the cardiovascular response to pressure was much smaller in magnitude (see. Ell et al., 2011), yet RB task performance was significantly impaired for participants experiencing this high pressure situation. Additionally, a trend between a threat like cardiovascular response and RB category learning has been found following the TSST (Ell et al., 2011) suggesting the type of stress response may play a role in the impact of stress on RB category learning. These studies are in line with research suggesting a relationship between the stress response and RB category learning, however the role of the endocrine response remains elusive as physiological stress was measured using a cardiovascular response only.

RB category learning is most strongly equated with an explicit learning system, however just because one system may be optimal does not ensure its reliance. In category learning, it has become clear that in addition to an explicit system, the implicit system also exists (Ashby et al., 1998), and may be considered sub-optimal for RB learning. These two systems are believed to be in competition with one another until one system eventually prevails (Ashby et al., 1998). Given this system competition, a stress induced deficit with an explicit system dependent task may be explained by an impairment in the system itself, or a shift to a slower, and hence sub-optimal implicit learning system (Ashby et al., 1998; Poldrack et al., 2001).

In the category learning literature tasks have been developed to explore both the explicit and implicit learning systems. In 2012, Schwabe & Wolf employed a weather prediction categorization task (WPT) to explore this learning system competition during stress. In this task participants were given sets of 1-3 cards and they were asked to predict the weather based upon the cue patterns of cards. For each set of cards they responded whether the weather was "sun" or "rain", and unbeknownst to the participant, each set of cards had a different probability of weather outcome. A correct response was defined as choosing the outcome of the strongest probability. No accuracy differences were present between stressed and non-stressed participants, however authors also examined the likelihood of a particular strategy given the trial by trial responses of participants. To do this ideal responses were created that would result from consistently basing decision upon either single cue or multi cue strategies. Participants actual responses were then compared to the ideal response for each strategy for each to create a least-means squared estimate across trials representing the likelihood of using that particular strategy. Comparing the least-means estimates signify the strategy participants were most likely relying on throughout the task. Results revealed the likelihood of using single cue explicit strategies was decreased, and the likelihood of using more implicit multi-cue strategies was increased in stressed participants compared to controls. Furthermore, neural structures associated with explicit learning correlated with task performance in control participants, while neural structures associated with the implicit system correlated with task performance for stressed participants (Schwabe & Wolf, 2012). This finding suggests not only does stress impair explicit learning, but the impairment may be associated with a shift to a sub-optimal learning system. Similarly, data from Ell and colleagues suggests a more threatening stress response (as measured through a particular cardiovascular reaction) is associated with increased performance on an Information Integration category learning task, optimally performed using

implicit system learning strategies (Ell et al., 2011). Taken together these results demonstrate the possibility of stress induced shifts to the implicit learning system.

Even given the relatively young literature, it is becoming clear that stress can have an impact on explicit learning, however there is still much left to explore. It is well known that the explicit learning system and particularly RB category learning depends upon WM (Waldron & Ashby, 2001; Zeithamova & Maddox, 2006), and it is possible this relationship with stress is reliant upon the relationship with WM. The abundance of the stress and WM literature provides a deeper account of the neural functioning and behavioral relationship between stress and explicit learning, and thus the remainder of this section will explore this relationship. WM can be defined as a limited capacity system involving both the on-line storage and updating of information, dependent upon prefrontal cortex (PFC) functioning (Baddeley 2012; Baier et al., 2010). Most often, stress has been found to negatively impact WM (Barsegyan et al., 2010; Elzinga & Roelofs, 2005; Lupien et al., 1999; Oei et al., 2006; Schoofs et al., 2008; Schoofs et al. 2009; Wolf et al., 2001), however a minority of published research has shown no effect (Kuhlmann et al., 2005; Vedhara et al., 2000), even WM enhancements (Henckens et al., 2011; Oei et al., 2009; Yuen et al., 2009). It is worth noting that some results have been conflicting within the same research groups (Duncko, 2009; Schoofs et al., 2013).

Due to the inconsistencies in the literature it is difficult to come to a single conclusion, even within the same research groups. One study in particular from Schoofs and colleagues in 2008, explored the impact of psychosocial stress on an n-back WM task in men. In this task participants are presented with a list of random numbers one by one, and must respond "yes" or "no" to whether or not the current number is the same as "n" numbers prior. The number n can vary, and in this version alternated between 2 and 3 for each block of the task. In order to

succeed on this task, the participant must not only mentally store the previously seen stimuli, but constantly update and integrate the new stimuli. In this study, task performance and reaction time was significantly impaired for participants who experienced a stressor as compared to participants completing a control version of the stressor (Schoofs et al., 2008). Interestingly, another study from the same research group using the same manipulation, task and post-stress time-course, did not find the same results. No differences in task performance were found, and reaction time was enhanced between the stressed condition and controls in males (Schoofs et al., 2013). The lack of replication poses new questions about what variables may have been influencing the previous impaired performance.

Importantly, Schoofs and colleagues are not the only group who failed to replicate WM findings following stress. Oei and colleagues similarly failed to replicate a WM impairment following stress (Oei et al., 2006, Oei et al., 2009). In the first of these studies male participants completed a classic item recognition task, designed to assess WM maintenance performance at varying WM loads (Sternberg, 1966), following the TSST. This task requires participants to hold a varying number of uppercase letter targets for 750 ms, until a new set of stimuli are presented, and to then respond whether or not the new set includes any of the previously stored targets. Data from fMRI analyses provides support for PFC functioning during this task (Narayanan et al. 2005), and previously impaired performance resulting from a hydrocortisone induced stress response has been found (Lupien et al. 1999). Reaction time was significantly slower for participants performing the task following the TSST than controls, revealing a WM impairment. Furthermore, in the TSST condition a negative correlation between salivary CORT reactivity and proportion correct was present, suggesting the stress response was responsible for task impairments (Oei, 2006). A follow-up study was conducted in 2009 using hydrocortisone to induce a human stress response. In this study WM was assessed again using the same WM

maintenance task, however a distracter was included by incorporating a display of either a neutral or emotional face during the maintenance period of the task (Oei et al., 2009). Overall, findings from this second study reveal enhanced task performance following hydrocortisone ingestion as measured by reaction time.

The discrepancy between the research from Oei and colleagues demonstrates the ambiguity in the literature even within the same lab. Important to consider however are the methodological differences between these two studies. Given the numerous differences it is impossible to conclude which may have played a role, however one difference the authors fail to acknowledge is the delay between the stress response and task. In the Oei et al. (2006) paper, participants completed the task 10 min after the TSST, whereas participants in the second study completed the task 115 min following hydrocortisone ingestion. Due to the design of Oei et al. (2009) it is unclear when the stress response began following drug administration, however other studies have shown increases in salivary CORT reactivity in as little as 30 min (Henckens, 2011) and plasma CORT reactivity as little as 25 min (Lupien, 1999) following hydrocortisone ingestion. Taken together these studies pose recovery time as a potentially mediating factor influencing WM task performance following stress. More on this topic will be discussed later.

The human literature is important presuming the research topic is ultimately geared towards helping humans, however much of the stress and cognition research in humans is either purely behavioral or indirectly assesses brain functioning following stressful situations via imaging techniques. The study of rodents, although relatively limited in terms of the complexity and variety of tasks, can provide insight into neuroscience that is presently not possible with human subjects offering a much more thorough examination of brain functioning. In terms of stress and WM, the rodent literature has provided us with reliable tasks and the likely neural

underpinnings of the cognitive process. Barsegyan and colleagues (2010) used a delayed alternation task with an elevated t-maze to assess behavior reliant on WM in rats. In this task, following habituation to a t-maze with food at each arm well, rats were placed in a start box, and once a gate was opened they were able to choose one of the two arm wells for a potential reward (chocolate treat). On the first trial a reward was given for either arm choice, and on all subsequent trials rats were rewarded only for entering the arm not chosen on the previous trial. Over time the inter-trial delays increased, forcing the animals to maintain information longer, and WM was assessed in comparison to a control version in which the inter-trial delay was held constant at 0 s. Increased delays have been found to require PFC activity for successful performance (Fuster, 1973), and given the influence of stress on PFC functioning (Arnsten, 2009) the authors hypothesized an impairment in task performance. All rats were reached 70-90% accuracy during training before continuing to the next phase of the experiment to ensure they were capable of completing the task. Results revealed significantly impaired WM performance when a glucocorticoid receptor (GR) agonist, mimicking the effects of CORT, was administered into the mPFC prior to WM assessment as compared to animals receiving saline administration. Importantly, the impairment was not present in the control groups who were tested without a delay between trials (Barsegyan et al., 2010). This finding suggests the WM is impaired under stress in rodents, and is in line with human studies. More on the mechanism underlying this finding will be discussed later.

Importance of Timing

An aspect receiving some much needed notoriety in the literature is the important role of the stress response time-course and its impact on explicit learning. This topic has often been ignored however, recent reviews have provided thorough proposals (Diamond et al., 2007;

Hermans et al., 2014; Joels et al., 2011; Joels et al., 2012; Schwabe et al., 2012). Explicit learning and the sub-processes involved are highly dependent upon PFC functioning (Miyake et al., 2000), therefore the focus of this section will mainly center on the stress-PFC literature. This section will begin by exploring the particular stress hormones that impact PFC functioning and propose that the interaction between these hormones is essential in the influence on cognitive processing. Next, a period further along the post-stress time-course will be explored in terms of neural function as well as explicit learning and the important role of the endocrine system. The goal of the section is to display the significance of the post-stress time-course, and review literature pertinent to the present hypotheses.

The PFC helps to coordinate a wide range of cognitive functions including selective attention and behavioral inhibition to make up cognitive control, WM, and RB learning (Miller & Cohen, 2001). The explicit learning system and similarly, RB category learning, rely upon afferent and efferent projections from the PFC (Ashby et al., 1998; Ashby & Ell, 2001). Given the role of the PFC and the behavioral impact of stress on explicit learning, it is not surprising the neurotransmitters released during stress are associated with PFC functioning (Arnsten, 2009; Roozendaal et al., 2007; van Stegeren et al., 2010). CORT and NE are important in determining the effects of stress on PFC functioning and will be discussed further below.

As mentioned previously, CORT released from the adrenal glands not only remains in the peripheral bloodstream and saliva, but is also able to freely cross the blood-brain barrier (Droste et al., 2008). After entering the brain, CORT freely binds to both mineralocorticoid receptors (MR) and GCs. Notably, CORT binds to MRs with 10 times higher affinity than GRs (de Kloet et al., 1999), and because of this only few GRs are bound until MRs are fully saturated (Lupien et al., 2007). Important for the present work, both MRs and GRs are highly expressed

within the human PFC, however it is worth noting a slight difference in rodents, in which MRs are not highly expressed in the PFC (Patel et al., 2007). This difference may not play much of a role in the present discussion as stress effects on explicit learning processes seem to be specific to GRs (Barsegyan et al., 2010). CORT ultimately affects the PFC in multiple ways including cellular activation, LTP via alterations in glutamate transmission (De Kloet et al., 2005) and even gene transcriptional changes (Barsegyan et al., 2010; Beato et al., 1995).

Arguably as relevant as the influence of CORT is the influence of catecholamines released during the stress response. During the initiation of the stress response, the locus coeruleus (LC), the brain's primary source of NE, shifts from a phasic to a tonic mode of activity (Aston-Jones et al., 1999). This shift is stimulated via the release of corticotropin releasing factor (CRF) (Valentino & Bockstaele, 2008). During the CRF induced NE release, the sympathetic nervous system activates the SAM axis triggering the peripheral release of epinephrine into the blood from the adrenal medulla. Once in the blood stream, epinephrine binds to the vagus nerve, which projects to the nucleus of the solitary tract in the brain stem (Schreurs et al. 1986). The brain stem stimulation triggers a further release of NE from the LC. Additionally, once the HPA is active, CORT binds to GRs in the brainstem again amplifying NE release. Throughout this process, NE binds to adrenoceptors in the BLA, and ultimately projects NE elsewhere to both cortical and sub-cortical structures (Roozendaal et al., 2007).

The paths triggered from the neurotransmitters released during stress may progress separately, however to understand the full impact of stress it is important to consider the synergy between CORT and NE. In particular, and discussed previously, research from Barsegyan and colleagues suggests glucocorticoid (GC) receptor stimulation in the mPFC is responsible for WM impairment in rats. Importantly, the simultaneous administration of a GR agonist and

noradrenergic antagonist within the PFC attenuated an impairment that was found in a group receiving the GC agonist alone. Also, no effects were revealed when a noradrenergic antagonist was administered alone suggesting the necessary combination of the two hormones (Barsegyan et al., 2010). The reliance upon this hormone combination is in line with previously reviewed studies on memory consolidation and retrieval, supporting the synergistic nature of the two hormones both for stress induced WM impairments and more general cognitive outcomes. Complimentary human research from van Stegeren and colleagues found a strong de-activation of the PFC measured with fMRI imaging data following hydrocortisone + yohimbine (alpha-2 antagonist that blocks inhibitory feedback of pre-synaptic auto-receptors ultimately facilitating the release of NE) treatment compared to groups receiving hydrocortisone + placebo, placebo + yohimbine and placebo + placebo (van Stegeren et al., 2010). Taken together these findings argue for the necessity of both CORT and NE for stress related PFC impairments across species.

Considering the focus of this section is to explore the impact of the stress time-course on WM and other explicit processes, the target structure has been the PFC, yet we must give credence to the role the amygdala plays in the development of neurotransmitter binding within the PFC. The majority of the direct LC projection sites include both the α (Birnbaum et al., 1999; Wang et al., 2007) and β receptors (Cole et al., 1981; Ellis & Kesner, 1981) found within the amygdala. Furthermore the amygdala may play a modulatory role in the projections from the LC to PFC (Bangasser & Shors, 2010; Roozendaal et al., 2009). In terms of the HPA's influence, a previously discussed study from Roozendaal and colleagues revealed both the administration of CORT and a GR agonist impaired WM performance as measured by a delayed-alternation task in rodents, however lesions to the BLA blocked both of these impairments. To be complete, this study also included a group of rodents given a systemic beta-adrenoceptor antagonist prior to the systemic CORT administration, and found that blocking the role of NE also abolished this

CORT induced impairment (Roozendaal et al., 2004). This finding indicates the CORT influenced WM impairments seem to be dependent upon the relationship between the BLA and PFC. Despite the authors conclusion that the BLA plays a direct role in the relationship between the PFC and WM, it remains a bit elusive whether or not the role of the amygdala is actually direct or just playing an intermediary role as a noradrenergic pathway to the PFC.

Most of the present cognitive influences from stress can be attributed to the roles of CORT and NE, however it is also important to acknowledge the breadth of neurotransmitter alterations occurring during stress (for review see: Joels & Baram, 2009). In addition to the previously discussed noradrenergic and glucocorticoid actions, numerous neurotransmitters are released during stress resulting in quick neural and cognitive changes. These hormones include CRH, vasopressin, oxytocin, serotonin, dopamine (DA), and more (Joels & Baram, 2009). For example, dopamine is centrally released shortly after a stressful event (Goto et al., 2007) and it is well known that DA release in the PFC is critical for cognitive functioning, particularly WM (Goldman-Rakic, 1995). DA has been typically studied in terms of its relationship with neural plasticity during learning and memory (Arbuthnott & Wickens, 2006) and it seems as if stress can impact this relationship. Prolonged stress has been known to trigger DA release causing long-term depression (LTD) during situations in which long-term potentiation (LTP) is the norm (Goto et al., 2007). Cognitively both DA (Zahrt et al., 1997) and NE (Arnsten et al., 1999) receptor stimulation within the PFC have been shown to mimic the effects of stress. This finding is in line with research revealing both DA (Arnsten & Goldman-Rakic, 1998) and NE (Birnbaum et al., 1999) receptor blockade following acute stress has been shown to reverse cognitive impairments. These findings suggest the NE mechanisms triggered via SAM activation do not reveal the entire stress and cognition story, and is just one example of the complexity surrounding neural physiology during stress. Similarly, mechanisms such as the release of GABA

and Opioids during stress have been shown to affect memory consolidation, however these mechanisms act through the modulation of NE release in the brain (Roozendaal & McGaugh, 2011), suggesting a large variability in the exclusivity of these processes. Given the immense number of possible mechanisms influencing cognition during stress, limiting the focus is essential to allow for a greater understanding and comparison between studies. In order to stay consistent and focused the present section has and will continue to target research reviewing HPA and SAM activation as well as the resulting influences specific to CORT and NE. Furthermore the time-course of these neurotransmitters will be of major consideration moving forward.

The research discussed thus far has focused on the endocrine mediated rapid effects of stress on cognition, that is the immediate influences of the stress response. This topic is important and well studied, however it doesn't include all aspects of the physiological time-course. We must also consider genomic effects driven through delayed intracellular mechanism. In addition to the rapid effects, we know the concurrent binding of CORT and NE within PFC cells also triggers a cascade of intracellular events, ultimately resulting in a delayed enhancement of behavioral WM performance (Henckens et al., 2011; Yuen et al., 2009). The best support for this mechanism comes from research by Yuen and colleagues who found a relationship between WM performance and amplified glutamate transmission driven by increases in AMPA and NMDA surface expression on PFC pyramidal neurons. This finding occurred in rats both 4 hrs and 24 hrs post GR stimulation, suggesting a delayed and extended duration of effects. The enhancement was abolished altogether with the co-induction of a GR antagonist, but not a MR antagonist within the PFC, demonstrating the specificity to GRs. Behaviorally, rats had increased performance in an alternating T-maze WM task, both 4 hrs and 24 hrs post stressor (forced swim stress) as compared to pre-stress performance, but the result was absent 2 days post stressor. The delayed behavioral enhancement was abolished following a combination of stress

and a GR antagonist, building continued support for an early endocrine mediated delayed enhancement of WM. (Yuen et al., 2009). These findings suggest the time-course of genomic WM effects extend well beyond the endocrine response itself, however will ultimately return back to baseline levels.

The increased glutamate transmission can be attributed to serum- and GC-inducible kinase (SGK) processes occurring within PFC pyramidal neurons. In a study complementary to the Yuen et al. (2009) paper, Yuen and colleagues (Yuen et al., 2011) provide evidence that glutamate signaling regulation requires SGK processes during stress. These processes can be attributed to CORT induced increases in Rab4 mediated AMPA and NMDA receptor recycling on the surface on PFC cells. The researchers showed mEPSC amplitude in PFC neurons was not increased in both SGK-knockdown rats and Rab4 deficient neurons. Furthermore this attenuation of glutamate transmission via reduced SGK and Rab4 processing was associated with a reduction in the previously shown WM enhancement following CORT treatment. Together the research from Yuen and colleagues provides a mechanism for the genomic affects of stress, and the ultimate WM ramifications. To be comprehensive the genomic effects of stress in humans will be discussed below.

Similar behavioral results have been shown in humans using hydrocortisone treatment to mimic the effects of acute stress. Henckens and colleagues (2011) found a delayed WM facilitation measured with n-back task performance 4 hrs after hydrocortisone treatment as compared to 30 min post hydrocortisone treatment and placebo treatment. Again, it is hypothesized that the early stress induction (hydrocortisone) initiated this belated WM effect (Henckens et al., 2011). Taken together, findings from Yuen and colleagues (2009, 2011) as well as Henckens et al. (2011) suggest WM performance is enhanced following stress provided a

lengthy enough delay from stress in both humans and rodents. Moreover, the delayed enhancements are triggered from rapid CORT and NE influences, but only indirectly via a genomic progression ultimately generating amplified glutamate transmission.

To my knowledge the only study utilizing the varying time-courses of SAM and HPA following a stressor and how they relate to WM in humans was published by Elzinga & Roelofs (2005). In this study participants experienced the TSST followed by two phases of WM assessment, one just following the TSST, and one 35 min after completion of the TSST. It is worth noting the duration of stress may have remained past the completion of the stressor as the evaluators from the TSST remained present in the room throughout the first task phase. Authors used a median split for a post-TSST CORT response to partition subjects into CORT "responders" vs. "non-responders". Results from this study revealed a rapid impairment on a digit span task for responders compared to non-responders, providing an agreement with the bulk of the literature. Pertinent to the present discussion, a WM effect was absent at a later time point (50 min after stressor onset) even though salivary CORT reactivity remained elevated in CORT responders (Elzinga & Roelofs, 2005). Given the time-course used and the design of this study, one may assume the absence of a delayed effect is due to a deactivation of SAM, even though a HPA elevation remains, however a measure of SAM was not included. The use of a data driven median split to define CORT response is useful to analyze current data, however creating these artificial groups disallows a comparison to other CORT response data because the definition of a significant response will vary across studies. The study was designed to explore the time-course of the stressful influence and supports a fleeting nature of stress' WM impairment, but given the absence of a SAM marker assumptions about the sympathetic stress response must be made. Together, these studies from Barsegyan et al. (2010) and Elzinga &

Roelofs (2005) suggest both HPA and SAM systems act in concert to inhibit PFC functioning and ultimately WM in both rodents and humans.

The progression of neural functioning following a stress response clearly affects WM, and it seems this finding can be broadened to explicit learning as a whole. Given the aforementioned inconsistencies surrounding the stress' influence on explicit learning it can be useful to aggregate the literature in attempts to step back and view the findings collectively. Shields et al. (2015) published a substantial meta-analysis in the spring of 2015 reviewing the findings of 30 studies exploring the influence of acute CORT administration on WM and other aspects of executive functioning. Perhaps unsurprisingly, given the inconsistencies mentioned thus far, Shields and his fellow researchers did not find overall relationships between CORT administration and either WM or inhibition (defined as "the ability to inhibit irrelevant information and selectively attend to goal-relevant information") task performance across studies. This data, discouraging on its own, becomes much more attractive when controlling for the length of delay between CORT administration and testing. WM impairments were found following a relatively short delay from CORT administration, described as a "non-genomic" or rapid effect, however enhanced performance was found when provided sufficient length of time post stress, described as a "genomic" effect. The association from the stress-delay relationship predicts the influence of WM will shift from non-genomic to genomic beginning 74 min following administration of CORT. Complicating this story, researchers report the effect to be in the opposite direction for inhibition. Enhancements in inhibition were found early on following the stress response, while impairments were present after greater delays (Shields 2015). Important to note is the very limited number of studies testing cognitive performance following a large delay from stress for both of these processes, and as the genomic time frame is explored further much more information may be unveiled. Also despite the relevance to the present

research questions, the time-course of CORT administration mustn't be considered equivalent to natural stress response time-course. Overall, these results strongly suggest the relationship between stress and recovery time will vastly impact executive functions and likely explicit learning as a whole.

In conclusion the nature of acute stress and its impact on cognition is an important topic for study and one that applies to a majority of the general public. A lot is known about stress and the bodily response, however assessment of the response is not straightforward. Numerous techniques can be applied to measure stress and help relate the type of response to other dependent factors, each with their own strengths and weaknesses. It has become clear that the physiological responses to stress can have an influence on cognitive performance, generating many questions about the mechanisms and outcomes. Overall stress has both harmful and beneficial impacts on cognition. Cognitive ramifications depend upon factors such as cognitive process, level and nature of physiological response, and time-course throughout and following stress. Despite a growing literature devoted to studying stress and cognition there is still much to learn, for example the specific time-courses of both rapid and genomic effects of the stress response, and furthermore how stress influences differ between separate cognitive processes. The overall picture remains elusive and the present research is designed to help answer some of the questions that still remain, in particular: What types of explicit learning are influenced by stress? How will the time-course of the stress response play a role in this relationship? Does the type of stressful situation matter? The 3 experiments presented in this thesis are designed to further explore these questions.

Experiment 1 aims to better understand how stress impacts RB category learning, and how the dynamic endocrine response plays a role. Since RB category learning has been shown to

depend upon WM processes (Waldron & Ashby, 2001; Zeithamova & Maddox, 2006), it is quite possible the influence of stress on RB category learning will be in line with the impact stress has been shown to have on WM. A between-subject design assessing RB task performance at three time points following stress will help to uncover the relationship between RB category learning and stress over time. The first time point (No Delay - ND) was chosen very soon following stress when both HPA and SAM should be active. The second time point (Short Delay - SD) was chosen during a period when HPA activity should still be elevated, but SAM activity should be fully recovered. Together the ND and SD conditions will help determine the rapid effects of a stressor on RB category learning, and assess whether they require activation of both SAM and HPA. The third and final time point (Long Delay - LD) is chosen during a greater delay following the stressor when both SAM and HPA should be fully recovered. The assessment of the RB task during this time point will target the potential genomic effects of stress on RB category learning. All conditions will be compared to a non-stressful comparison condition (No Stress - NS). I hypothesize the performance on the RB task will be impaired in the ND condition compared to the NS condition. This prediction is based upon the generally accepted rapid impairments of stress on explicit learning (McCoy et al., 2013; Oei et al., 2006; Plessow et al., 2012a; Schoofs et al., 2008). I hypothesize no performance differences in the SD condition as compared to the NS condition. This prediction is based upon the findings suggesting the rapid effects of stress on WM requires SAM activation (Barsegyan et al., 2010; Elzinga & Roelofs, 2005). Lastly I hypothesize performance in the LD condition will be enhanced as compared to the NS condition. This prediction is based upon the genomic enhancements found in WM following a sufficient delay from stress (Henckens et al., 2007; Yuen et al., 2009).

Experiment 2 is designed to build upon findings from the LD condition in Experiment 1. This between-subjects experiment will attempt to replicate the delayed enhancement from

Experiment 1, as well as assess whether RB category learning effects extend to a more traditional WM task. This experiment will help parse the relationship between RB category learning and WM following a delay from a stressor. I hypothesize both RB and WM task performance will be enhanced in the stress condition compared to the no-stress condition, given the reliance of the RB task upon WM.

Finally Experiment 3 was designed to replicate and extend RB category learning findings from the LD condition from Experiments 1 and the stress condition from 2. As a comparison to a previously studied stress-WM time-course the further delayed time point will be ~4 hrs post stress. Enhancements have been shown during this delay from stress in both rodents (Yuen et al., 2009) and humans (Henckens et al., 2011). Given the findings from Experiments 1 and 2 and the previously shown WM enhancements 4 hours post stress, I hypothesize the RB task will be enhanced at both time points following the stressor as compared to non-stressed controls. Additionally Experiment 3 will incorporate a new stressor in attempts to generalize this finding beyond stress related to the TSST.

CHAPTER 2

EXPERIMENT 1

Method

Participants & Design

Participants (N = 78undergraduate students from the University of Maine, 48 female; represents sample size after exclusions described below) arrived for a study on "Learning & Memory" and sensors to monitor cardiovascular and hemodynamic reactivity were applied (ECG, Impedance Cardiography (ICG), BP). Participants then relaxed for a 20 min baseline. Twenty-eight females were using OCs and 20 were naturally cycling (11 luteal phase, 9 follicular phase). None of the participants in this study reported any of the following: depression, bi-polar disorder, heart-disease, obesity, panic disorder, schizophrenia, psychosis, hypertension, alcohol or drug problems, neurological problems, anxiety, irregular menstrual cycle, heart problems, or pregnancy. All participants reported normal or corrected-to-normal vision. Participants received course credit or monetary reimbursement ($10/hr) for their participation. Seven participants were excluded from RB task analyses due to technical problems. Additionally the responses from participants who were in the normal range of cardiovascular measurement at baseline but were found to be statistical outliers (> 3 SD from the group mean) in terms of reactivity were winsorized to the closest non-outlier response. This included HR responses from three participants. Participants were excluded from saliva analyses for having greater than 50% of saliva samples missing. This was true for eight participants' CORT responses and eight participants' sAA responses. Additionally two participants were excluded due to greater than 50% of salivary responses revealed as statistical outliers (>3 SD from the group mean). All participants were randomly assigned to complete either a social stressor (n = 57) or a no-stress

comparison condition (NS, n = 21). Afterwards, all participants completed the categorization task. Participants in the stress condition were randomly assigned to complete the categorization task at one of three delay intervals relative to stressor offset (no delay - ND, n = 19; short delay - SD, n = 19; long delay - LD, n = 19). All participants in the no-stress comparison condition (NS, n = 21) performed the categorization task with no delay.

Social Stress Manipulation

Trier Social Stress Test. Participants in the ND, SD, and LD conditions performed a modified version of the TSST (Kirschbaum et al., 1993) in front of two evaluators (one female, one male) trained to display flat affect and neutral facial expression throughout the test. Participants met the evaluators, the test instructions were explained, and they were left alone to prepare for 5 min (anticipatory stress). The evaluators returned and guided the participant in the speech (5 min), the interview (5 min), and serial subtraction by 7s (5 min). All components were performed in the presence of the evaluators and participants were informed that their performance would be recorded on video for further evaluation at a later time.

No-Stress TSST. Participants in the NS condition performed a modified version of the "placebo" TSST (Het et al., 2009) designed to mimic the TSST while minimizing the possibility for social evaluation. The task instructions were explained and the participants were left alone to prepare for 5 min. Participants then gave a speech aloud about their favorite movie/novel (5 min), a speech aloud about their favorite (actual or desired) vacation (5 min), and serial addition by 15 s (5 min). All components were performed alone and participants were informed that their performance would not be seen, heard, or recorded.

Stress Markers

Stress Appraisal. To assess the efficacy of the social stress manipulation, participants were asked to rate (immediately after the TSST or no-stress TSST) the extent to which they found the experience to be stressful, challenging, and threatening (on a 1 "strongly disagree" to 7 "strongly agree" scale). Participants were also asked to rate the supportiveness of the evaluators (on a 1 "unsupportive" to 7 "unsupportive" scale). The latter three ratings were included for comparison to an ongoing project and, therefore, only the stressfulness ratings will be analyzed.

Cardiovascular Measures. Electrocardiogram (ECG) and BP were observed and recorded for the duration of the study using BioPac MP150 hardware and BioPac Acquire software. All cardiovascular recordings were ensemble averaged and cleaned using Mindware software. HR (in beats per minute) and BP measured as mean arterial pressure (MAP; in mmHg: [2(diastolic BP) + systolic BP] / 3) were calculated throughout the study. Reactivity scores for these latter time points were calculated by subtracting the last 2 min of the cardiovascular response during the baseline period from the cardiovascular response during the last 2 min of the social stress manipulation. Thus, positive reactivity scores indicate an increase in the physiological response relative to baseline. The cardiovascular response at the end of the social stress manipulation represents the extent to which the manipulation was cardiovascularly arousing.

Salivary Endocrine Measures. To measure salivary CORT and sAA, saliva samples were collected at six time points (min) relative to stressor onset: -5, 5, 23, 47, 73, and just before leaving (approximately 88 for NS, ND, and SD conditions; approximately 109 for the LD condition). For each sample, participants were given 3-5 min to provide approximately 2 ml of saliva (unstimulated passive drool via straw into a polystyrene vial). Samples were stored at -20 °C until analyzed in batch at Dr. Nic Rohleder's laboratory at Brandeis University, Waltham MA.

Salivary CORT was measured using a commercial chemiluminescence immunoassay (CLIA; IBL-International, Toronto, ON, Canada). Inter- and intra-assay coefficients were 5.84% and 4.05%, respectively. sAA was measured by an enzyme kinetic assay using reagents from Roche Diagnostics (Indianapolis, IN, USA) as described in Rohleder et al. (2006). Inter- and intra-assay coefficients were 5.98% and 3.12%, respectively.

Rule-Based Categorization Task

Next, participants completed a RB categorization task. The stimuli were sine-wave gratings that varied across trials in spatial frequency and orientation (counterclockwise from

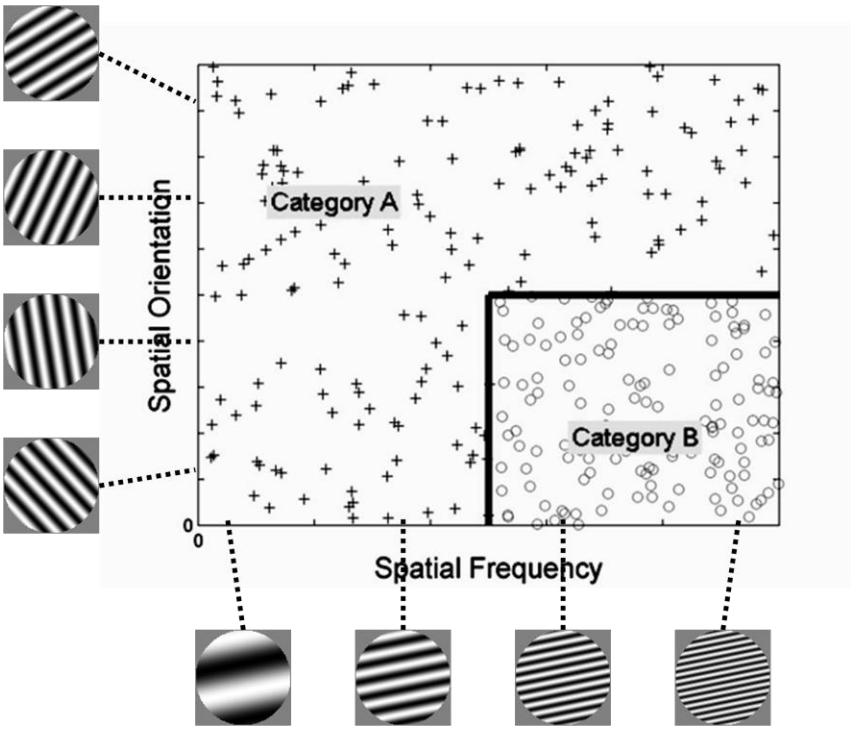

Figure 1: Stimuli used in Experiment 1. Stimuli vary on Spatial Orientation (angle of the stimuli) and Spatial Frequency (number of lines a stimulus has). The example stimuli shown represent the dimensional range along each respective stimulus axis. Each point on the scatter plot represents a single stimulus the participant will see during the RB task. Pluses represent stimuli belonging to Category A, and circles represent stimuli belonging to category B. The bold line represents the optimal decision boundary.

horizontal). Eighty-four stimuli were used with 42 assigned to each of the two response categories. To create these category structures, a variation of the randomization technique (Ashby & Gott, 1988) was used. Each cluster of stimuli was defined as a bivariate uniform distribution with a minimum and maximum on each dimension and was assigned to either category A or category B (see Table 1 for the category parameters and Figure 1 for the category structure).

Each stimulus was generated (offline) by taking a random sample (*x, y*) from one of the uniform distributions described in Table 1. Each random sample was converted to a stimulus by deriving the frequency, $f = .1 + \frac{x}{80}$ cycles/degree of visual angle, and orientation, $o = \frac{180y}{800}$ degrees. The stimuli were scaled in an attempt to equate the salience of spatial frequency and orientation as is common with these stimuli (e.g., Ell et al., 2009). Each stimulus was presented on a gray background and subtended a visual angle of 4.35° at a viewing distance of approximately 51 cm. The stimuli were generated and presented on a 17-in. LCD with 1,680 x 1,050 resolution using the Psychophysics Toolbox extensions (Brainard, 1997; Pelli, 1997) for MATLAB. Participants were instructed that their goal was to learn the categories by trial-and-error. Participants were informed that there were two equally likely categories and that the best possible accuracy was 100% (i.e., optimal accuracy). On each trial, a single stimulus was presented and the participant was instructed to make a category assignment by pressing one of two response keys (labeled ‘A’ or ‘B’) with either the index or middle finger of their dominant hand. A standard keyboard was used to collect responses. The keyboard characters ‘n’ and ‘j’ ('v' and 'f' for left-handed participants) were assigned to categories ‘A’ and ‘B’, respectively. Participants were instructed to make their response during a 2 sec interval that coincided with stimulus presentation. If the participant failed to respond during the interval, the participant

was instructed: "Too slow please try to respond within 2 seconds". Otherwise, the screen was blanked for the remainder of the interval. Using a fixed response interval ensured that the task

Table 1: Parameters Used to Generate the Figure 1 Categories

Category Cluster	Spatial Frequency		Orientation		Number Per Cluster
	Min	*Max*	*Min*	*Max*	
Category A	10	350	290	630	14
Category A	350	690	290	630	14
Category A	10	350	-50	290	14
Category B	350	690	-50	290	42

Note. Spatial frequency and orientation values are in arbitrary units. The number of stimuli per cluster was chosen in order to ensure equal category base rates.

duration was identical for all participants. After responding, feedback was provided. When the response was correct, the word “CORRECT” appeared in green and was accompanied by a 1 s, 500 Hz tone; when incorrect, the word “WRONG” appeared in red and was accompanied by a 1 s, 200 Hz tone. The screen was then blanked for 500 ms prior to the appearance of the next stimulus. In addition to trial-by-trial feedback, summary feedback was given at the end of each 84-trial block, indicating overall accuracy for that block. The presentation order of the 84 stimuli was randomized within each block (four total), separately for each participant.

Filler Task

During the filler task, participants were asked to partake in an online grocery shopping experience (peapod.com), where they were free to buy whatever items they wanted, and directed to shop for items they currently felt like buying. The data from the filler task are part of

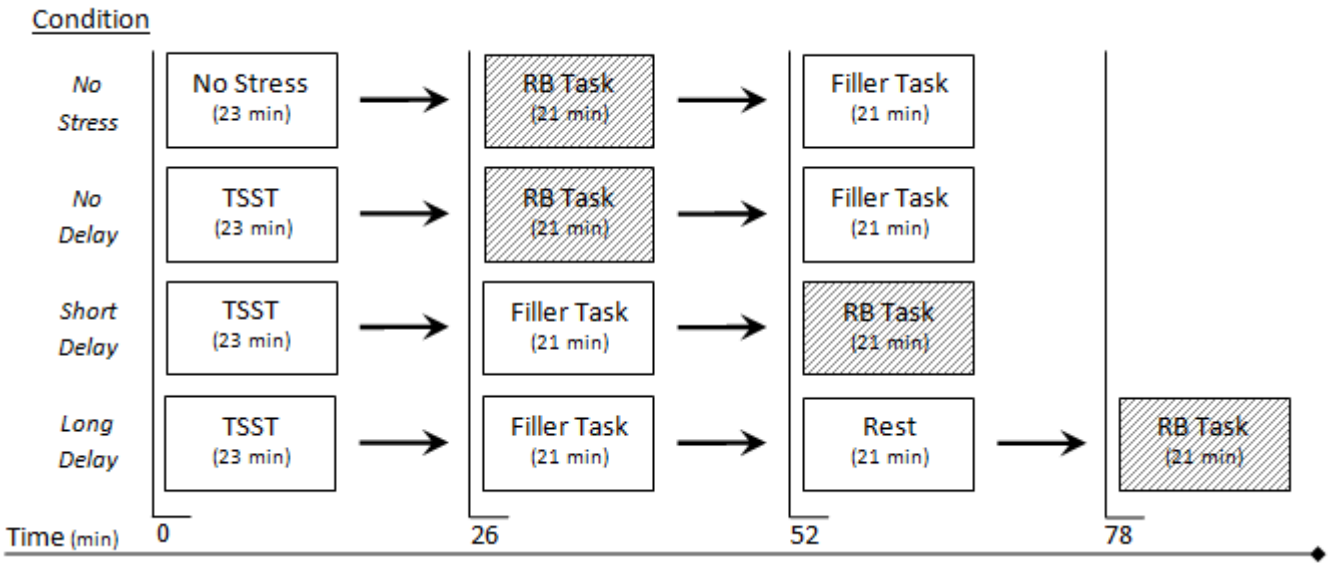

Figure 2: Procedure and timeline of Experiment 1.

an ongoing study and are not central to the present work, thus these data will not be discussed further.

Procedure

A graphical overview of the procedure is shown in Figure 2. After providing informed consent, participants were randomly assigned to one of the four conditions (NS, ND, SD, or LD). Following cardiovascular equipment preparation, participants were seated in a comfortable chair and asked to relax in the presence of calming music for 20 min for baseline cardiovascular response collection. Participants in the ND, SD, and LD conditions completed the TSST and then the RB task at varying delays post stressor. Participants in the NS condition completed the no-stress TSST followed by the RB task. At any time point where the participant was not completing the RB task, they were completing the filler task (data not provided), producing a saliva sample, or resting. Once the experiment was finished, all cardiovascular equipment was removed, the participant was de-briefed and thanked for their participation.

Results

Analyses of appraisal, cardiovascular [1], and salivary endocrine data as well as cognitive task performance are provided below.[2] Appraisal data from self-report is designed to assess the self-perception of stress, and the cardiovascular response is meant to measure physiological arousal. Endocrine responses are provided to assess SAM and HPA axis activation during the

[1] Note that the degrees of freedom for our cardiovascular and salivary measures fluctuated slightly as a result of missing or unscorable data due to equipment issues. This is true for Experiments 1 & 2 for cardiovascular data, and only Experiment 1 for salivary data.

[2] Post-hoc comparisons for within-subject factors were statistically adjusted using a Sidak correction to minimize type 1 error. Post-hoc comparisons for between subjects factors were compared using the Student Newman Keuls procedure, with the exception of ANCOVA analyses, which were adjusted using a Sidak correction. Violations of sphericity (for within-subjects analyses) were corrected using a Greenhouse-Geisser procedure. This procedure will continue for all experiments.

TSST. Group comparisons for the RB task will provide an assessment of cognitive performance during different time points following the stressor, and follow up regression analyses will assess the relationship between the stress measures and RB task performance.

Stress Markers

Stress Appraisal. After completion of the TSST, all participants were asked to rate how stressful their experience was on a likert scale from 1-7 (Figure 3). A stress appraisal x timing condition ANOVA revealed a main effect of timing condition ($F(3,72) = 16.88$, $p < .001$, $MSE = 1.96$, $\eta_p^2 = .413$), and follow up post hoc analyses revealed the main effect was driven by significantly higher appraisal of stress in the ND, SD, and LD conditions relative to the NS condition (all p's < .05). None of the remaining pairwise comparisons were significant (all p's > .05). Thus the TSST was perceived as being equivalently stressful across the ND, SD, and LD conditions and more stressful than the no-stress comparison condition.

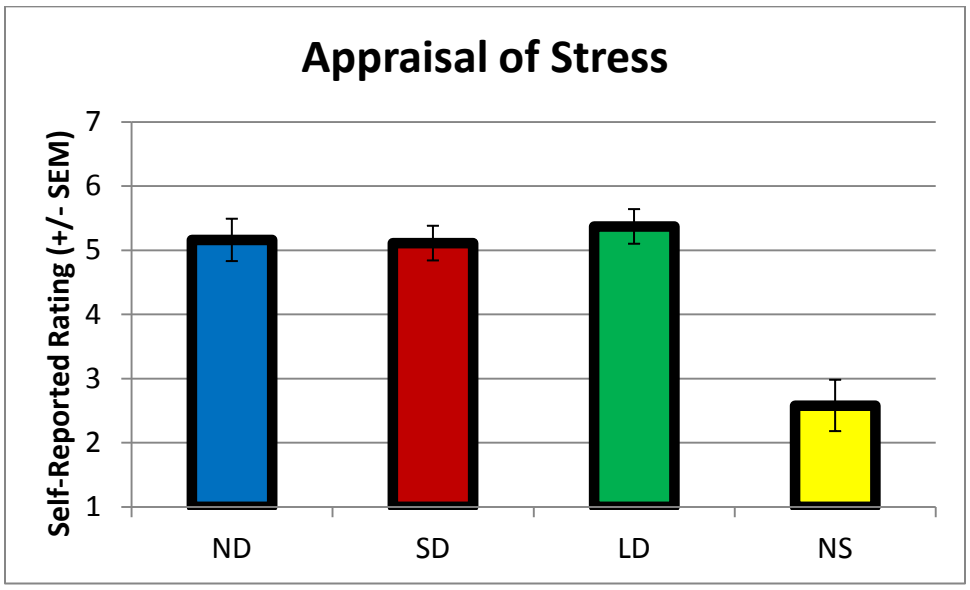

Figure 3: Stress appraisal data from Experiment 1

Baseline Cardiovascular Response. Cardiovascular measures x timing condition ANOVAs conducted for baseline cardiovascular data suggested participants in the four conditions did not differ in HR ($F(3,67) = .74$, $p = .53$, $MSE = 136.1$, $\eta_p^2 = .03$). There was, however, a main effect of timing condition for MAP ($F(3,66) = 3.75$, $p = .02$, $MSE = 105.97$, $\eta_p^2 = .15$) driven by a significant difference between the NS and LD conditions ($p < .05$), possibly reflecting a failure of random assignment. To correct for these differences, follow up reactivity analyses for MAP will use baseline cardiovascular response as a covariate. For ease of interpretation, data reported in figures will not be covariate-adjusted.

Cardiovascular Reactivity. Reactivity scores were calculated by subtracting the average cardiovascular response during the last 2 min of baseline from the last 2 min of the stress manipulation (higher scores indicate higher reactivity). A series of one sample t-tests comparing the reactivity scores to 0, indicated that all four conditions had significant HR reactivity (t's > 4.20, p's < .002) suggesting HR was consistently elevated relative to baseline (Figure 4).

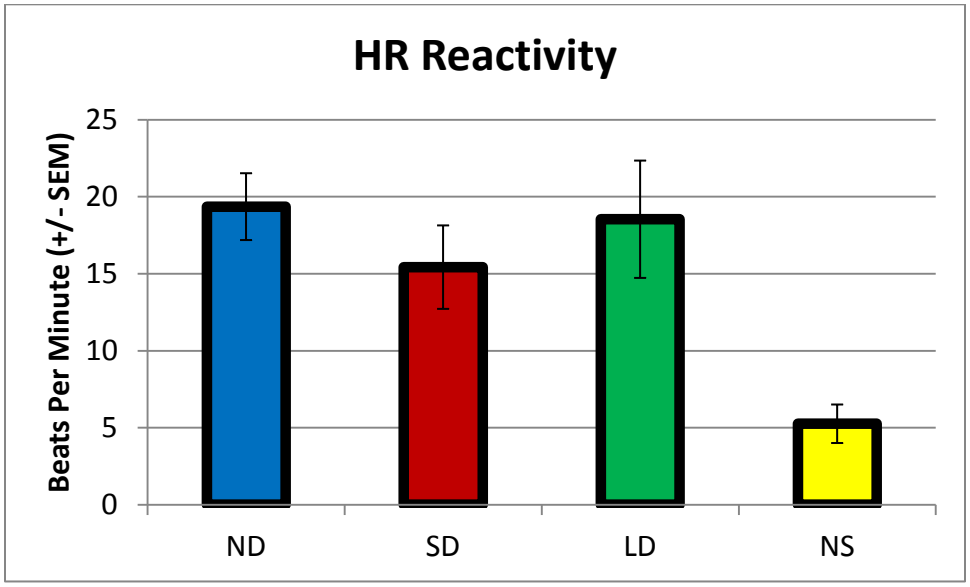

Figure 4: HR reactivity data from Experiment 1

However, a HR reactivity x timing condition ANOVA revealed a main effect of timing condition ($F(3,67) = 6.46$, $p = .001$, $MSE = 116.64$, $\eta_p^2 = .22$) driven by significantly higher HR reactivity in the ND, SD, and LD conditions relative to the NS condition ($p's < .05$). None of the remaining pairwise comparisons were significant (p's > .05).

A series of one sample t-tests indicated all four conditions had significant increase in MAP reactivity (t's > 3.29, p's < .005) suggesting that MAP was consistently elevated relative to baseline (Figure 5). A MAP reactivity x timing condition ANCOVA revealed a main effect of timing condition ($F(3,61) = 5.21$, $p = .003$, $MSE = 134.26$, $\eta_p^2 = .2$) and follow up post-hoc analyses indicate this effect is driven by a significantly higher MAP reactivity in the SD ($p = .03$), and LD ($p = .002$) conditions as compared to the NS condition. Reactivity in the NS condition and the ND condition ($p = .25$) were not significantly different. None of the remaining pairwise comparisons

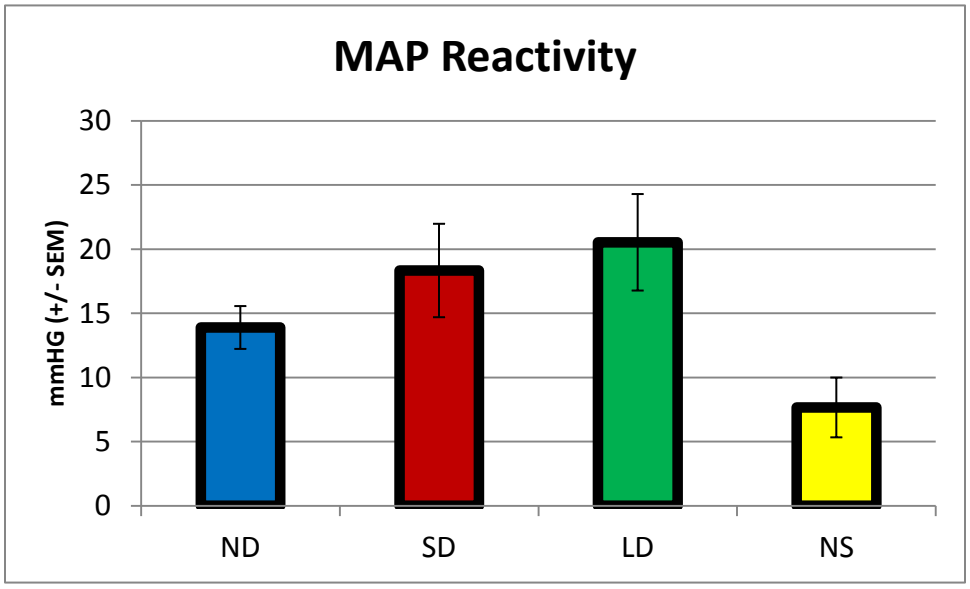

Figure 5: MAP reactivity data from Experiment 1

were significant (p's > .28) [3]. Taken together, the HR and MAP reactivity results indicate participants were cardiovascularly stressed during the TSST.

Baseline Salivary Endocrine Response. Given the differences in endocrine response time-courses, baseline will be chosen separately for each salivary measure in an attempt to gauge the trough of the response (Dickerson & Kemeny, 2004; Rohleder et al. 2004). The HPA axis is sluggish, both during onset and recovery, thus to provide as much time as possible following any potential stress inducing elements of equipment preparation, a measure of baseline CORT was measured during the anticipatory period. CORT collected during the anticipatory period will not yet be influenced from the TSST, as salivary CORT elevation requires a significant length of time following the onset of a stressor (Kirschbaum & Hellhammer, 1994). Given the brief nature of both SAM activation and recovery, baseline sAA levels were assessed from samples at the end of the relaxation period. Endocrine measure x timing condition ANOVAs conducted for baseline salivary measures suggest no main effect of timing condition for both CORT ($F(3,70) = .83$, $p = .48$, $MSE = 17.28$, $\eta_p^2 = .03$) and sAA($F(3,69) = .07$, $p = .97$, $MSE = 2879.95$, $\eta_p^2 = .003$). The absence of a difference between conditions in baseline salivary measures allow for subsequent salivary analyses to be assessed in terms of reactivity, or a change from baseline.

Salivary Endocrine Reactivity. Reactivity scores were calculated by subtracting the salivary sample at baseline from the sample immediately following the stress manipulation (higher scores indicate higher reactivity; see Figures 6 and 7 for CORT and sAA data at all time points, respectively). A series of one sample t-tests indicated significant increase in CORT reactivity in the ND ($t(16) = 3.47$, $p = .003$), SD ($t(16) = 2.23$, $p = .04$) and LD ($t(17) = 3.73$, $p = .002$)

[3] Baseline MAP was a marginally significant covariate ($F(1,67) = 3.76$, $p = .057$, $MSE = 140.22$, $\eta_p^2 = .053$). Note that the results of a one-way ANOVA are consistent with the ANCOVA results [main effect of timing condition $F(3,68) = 5.13$, $MSE = 145.92$, $p = .003$]; follow up post-hoc analyses reveal this main effect to be driven by significant differences between the NS condition with both SD and LD conditions (p's < .05).

conditions. As expected no increase in CORT reactivity was found in the NS (t(21) = -.56, p = .58) condition. A CORT reactivity x timing condition ANOVA reveals a main effect of timing condition (F(3,70) = 7.31, p <.001, MSE = 14.63, η_p^2 = .24), and post-hoc pairwise comparisons indicate the main effect is driven by a significantly greater CORT reactivity in LD condition than the NS condition (p's <.05) and a marginally greater CORT reactivity in the ND condition than the NS condition (p = .065), but no differences between the SD condition and NS condition (p > .05). CORT reactivity was also greater in the LD condition than both the ND and SD conditions (p's < .05).

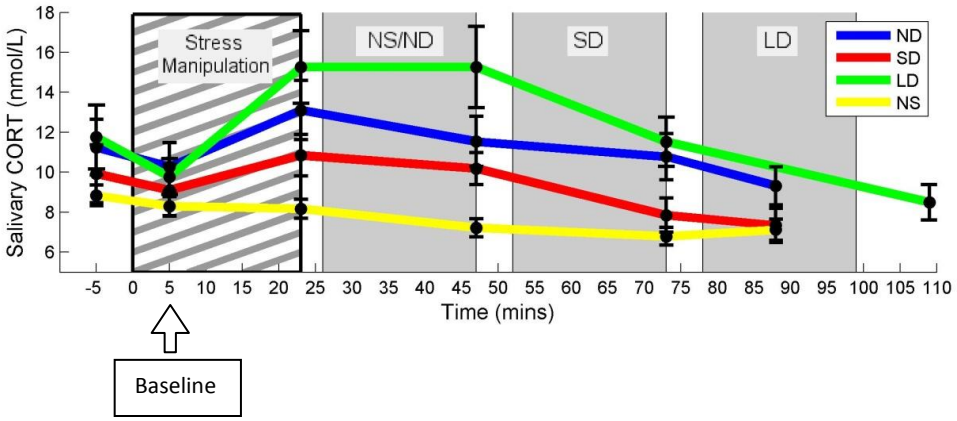

Figure 6: Salivary CORT responses. Each black point represents the beginning of collection of a saliva sample. The striped box represents the time during which the stress manipulation was being completed. Each of the gray boxes represent the time during which the RB task was performed for each condition.

A series of one sample t-tests indicated a significant increase in sAA reactivity in the SD (t(16) = 3.23, p = .005) and LD (t(17) = 2.82, p = .01) conditions, but not the ND (t(14) = 1.86, p = .08) condition. A one sample t-test also reveals a significant negative reactivity in the NS (t(20) = -2.79, p = .01) condition, indicating a reduction in sAA from baseline. A sAA reactivity x timing condition ANOVA reveals a main effect of timing condition (F(3,67) = 7.06, p < .001, MSE = 2251.66, η_p^2 = .24). Post-hoc pairwise comparisons suggest the main effect is driven by a

significantly greater sAA reactivity in the SD and LD conditions compared to the NS (p's < .05) condition, and a marginal elevation in the ND condition compared to the NS (p = .056) condition. sAA reactivity was also marginally elevated in the SD condition compared to the ND (p = .056) condition.

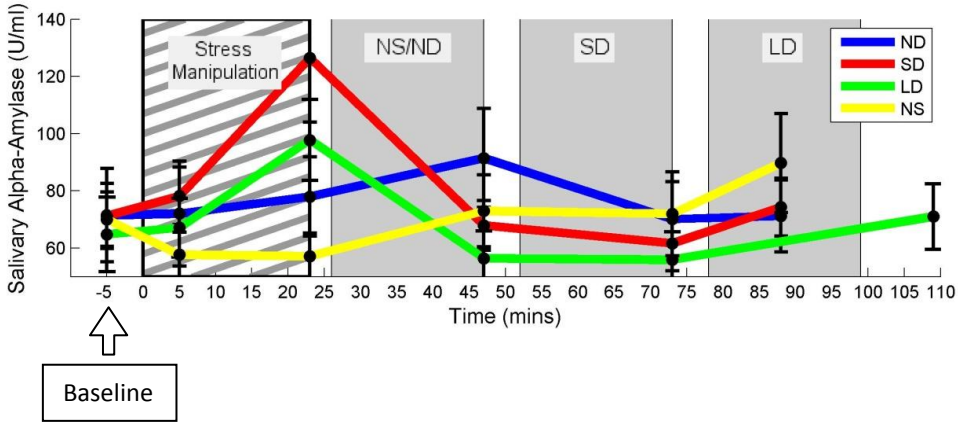

Figure 7: sAA responses. Each black point represents the beginning of collection of a saliva sample. The striped box represents the time during which the stress manipulation was being completed. Each of the gray boxes represent the time during which the RB task was performed for each condition.

Cognitive Task Performance

Rule-Based Task Accuracy. A four block (within) x four condition (between) mixed repeated measures ANOVA was conducted for RB task accuracy. A significant main effect of block ($F(2.05,151.72) = 37.76$, $p<.001$, $MSE = 62.48$, $\eta_p^2 = .34$) revealed an increase in accuracy across blocks (ex. block 4 accuracy was significantly higher than block 1 accuracy. $p < .05$) indicating participants were able to learn the task. Importantly, there was a significant main effect of timing condition ($F(3,74) = 3.86$, $p = .013$, $MSE = 233.77$, $\eta_p^2 = .14$) driven by higher accuracy in the LD condition relative to the NS and the SD (p's < .05; see Figure 8) conditions. Accuracy in the LD condition was also marginally higher than accuracy in the ND condition (p = .057).

Inconsistent with hypotheses, the ND condition was not impaired on the task compared to the NS condition ($p > .05$). None of the remaining pairwise comparisons were significant (p's > .05). The block x condition interaction was not significant ($F(6.15,151.72) = 1.72$, $p = .117$, $MSE = 62.48$, $\eta_p^2 = .07$). In sum, the accuracy analyses are consistent with the hypothesis of a delayed enhancement in RB task performance.

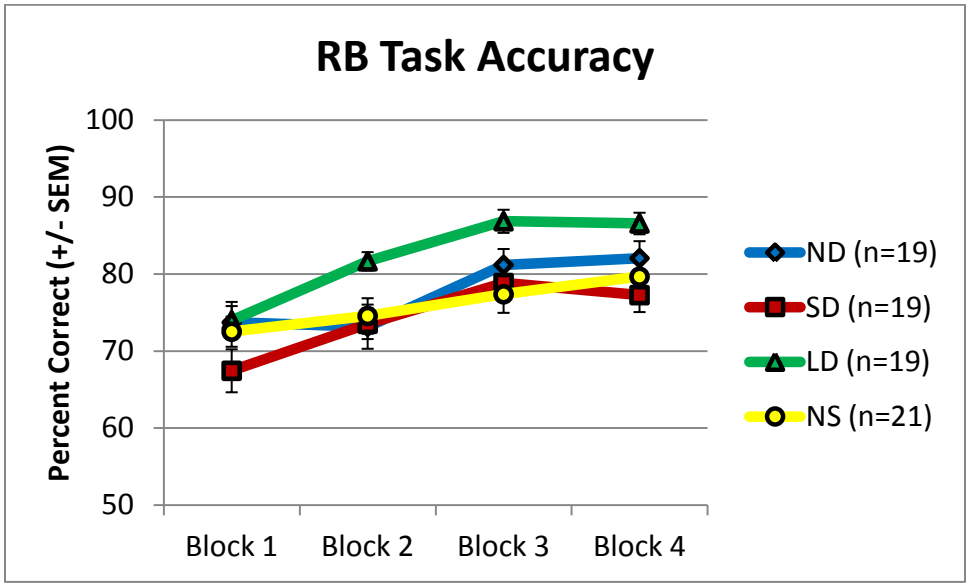

Figure 8: RB task accuracy for Experiment 1.

Rule-Based Task Strategy. In addition to accuracy, acute stress may have affected how participants performed the RB task. For example, acute social-evaluative stress may have biased participants toward a suboptimal, unidimensional decision strategy in which one of the stimulus dimensions (e.g. orientation) was ignored, and the decision was made purely from the other dimension (e.g. spatial frequency). To explore this issue, model-based analyses were conducted to evaluate variability in the decision strategies used during the RB task (Ashby, 1992). Four models were investigated. The unidimensional classifier assumes that participants use a decision criterion that focuses exclusively on spatial frequency or orientation. The conjunctive classifier

assumes that participants use decision criteria on both spatial frequency and orientation, representing optimal strategy use for this task. The general linear classifier assumes that the participant combines the stimulus information from both dimensions prior to making a categorization decision. Finally, random-responder (RR) models assume that the participant simply guesses during the task. Each of these models was fit separately to the data from every block for all participants using a standard maximum likelihood procedure for parameter estimation (Ashby, 1992; Wickens, 1982) and the Bayes information criterion for goodness-of-fit (Schwarz, 1978). See the Appendix for a description of the models and fitting procedures.

Strategy analyses focused on block 4 of the task as to provide ample time for strategy development. (see Table 2)[4]. Participants in the LD condition were significantly more likely to use a conjunctive strategy than a unidimensional strategy than participants in the ND ($\chi^2(1,38) = 4.39$, $p = .04$) and SD ($\chi^2(1,37) = 6.41$, $p = .01$) conditions, but there was no significant difference in strategy use between the LD and NS conditions ($\chi^2(1,40) = 2.49$, $p = .12$). No other differences were significant (all p's > .27).

Table 2: Participant Strategy Usage Counts

Strategy Use During Block 4				
	ND	*SD*	*LD*	*NS*
Unidimensional	9	10	3	8
Conjunctive	10	8	16	13

[4]Block 4 was chosen because this is the time when participants have best learned the task. The random responder model and the general linear classifier provided the best fit for 1 and 0 participants (respectively). Thus, these models were omitted from strategy analyses.

Relationship Between Stress and RB Task Performance

To fully understand the impact of stress on RB category learning it is important to determine the relationship between the stress response, both physiological and appraised, and RB task performance. In order to assess RB task accuracy at a time where performance had stabilized, all of the following regression analyses concentrate on block 4 task only[5]. The following hierarchical regression model (Step1 predictors: stress condition, stress response; Step2 predictors: stress response, stress condition x stress response interaction; Outcome variable: block 4 accuracy) were designed to determine the extent to which stress responses moderated the relationship between condition and RB task accuracy. Each model was dummy coded such that the ND, SD, and LD conditions can be compared to the NS condition and was evaluated separately for each stress response variable (i.e., Stress Appraisal, HR reactivity, MAP reactivity, CORT reactivity, sAA reactivity). Thus, the results of the step 1 analysis will assess the main effect of stress condition for the participants represented in each in each model. The results of the step 2 analysis will assess if the relationship between the stress response and RB task accuracy differs by condition for each stress response variable. Because the primary focus of this analysis is to understand the moderating effects of the stress response variables, I will focus the description of the results on the stress response x condition interaction at step 2. To further characterize each interaction, simple slopes were estimated for the NS, ND, SD, and LD conditions. The simple slope analysis allows for the investigation of the relationship between stress reactivity and accuracy within each condition. For simplicity, all statistics (for both steps)

[5] A RB task performance x timing condition ANOVA comparing block 4 RB task accuracy revealed a significant main effect of condition ($F(3,74) = 3.902$, $p = .012$, $MSE = 77.195$, $\eta_p^2 = .137$). Follow up post-hoc tests suggest the effect is driven by higher task accuracy in the LD condition relative to the NS and SD($p's < .05$) conditions. This finding is in line with a previously reported repeated measures ANOVA results revealing a main effect of condition across blocks.

Model summary of hierarchical regression analyses from Experiment 1

Variable	Stress Appraisal										HR Reactivity								
	b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2		b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2
Step 1				2.82	.03			.14						2.47	.053			.13	
ND	3.05	.87	.39								1.4	.42	.67						
SD	-1.76	-.08	.61								-2.79	-.88	.38						
LD	7.61	.35	.04								5.77	1.71	.09						
Stress response	-.277	-.05	.72								.089	.88	.38						
Step 2						1.1	.36	.18	.04							3.12	.032	.24	.11
ND	13.62	1.56	.12								-6.09	5.43	.27						
SD	9.32	.94	.35								-10.93	4.51	.02						
LD	3.01	.28	.78								-1.02	4.51	.82						
Stress response	.85	.7	.49								-1.06	.39	.009						
Stress response X ND	-2.61	-1.37	.18								1.23	.45	.008						
Stress response X SD	-2.73	-1.27	.21								1.29	.43	.004						
Stress response X LD	.27	.13	.9								1.19	.42	.006						

Table 3: Model summary of hierarchical regression analyses from Experiment 1. Degrees of freedom vary due to missing data associated with various equipment malfunction and unusable saliva samples. Regression statistics are reported as unstandardized ts and bs.

Model summary of hierarchical regression analyses from Experiment 1

Variable	MAP Reactivity										CORT Reactivity								
	b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2		b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2
Step 1				2.4	.06			.14						4.81	.002			.22	
ND	2.26	.73	.47								3.21	1.18	.24						
SD	-.9	-.28	.78								-3.33	-1.25	.21						
LD	7.27	2.2	.03								4.23	1.44	.15						
Stress response	.06	.63	.54								.5	1.99	.051						
Step 2						2.48	.07	.23	.1							.58	.63	.24	.02
ND	-4.7	-.86	.39								5.23	1.66	.1						
SD	-8.28	-1.88	.07								-3.71	-1.3	.2						
LD	7.64	1.68	.1								3.51	1.12	.27						
Stress response	-.3	-1.34	.19								.23	.13	.89						
Stress response X ND	.66	1.73	.09								-.45	-.25	.8						
Stress response X SD	.61	2.27	.03								.51	.28	.78						
Stress response X LD	.21	.77	.44								.41	.24	.81						

Table 4: Model summary of hierarchical regression analyses from Experiment 1. Degrees of freedom vary due to missing data associated with various equipment malfunction and unusable saliva samples. Regression statistics are reported as unstandardized ts and bs.

Model summary of hierarchical regression analyses from Experiment 1

Variable	sAA Reactivity								
	b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2
Step 1				3.02	.02			.16	
ND	3.79	1.3	.2						
SD	-1.7	-.54	.59						
LD	7.17	2.49	.02						
Stress response	-.01	-.47	.64						
Step 2						.3	.83	.17	.01
ND	3.47	1.05	.3						
SD	-.75	-.22	.83						
LD	6.15	1.87	.07						
Stress response	-.004	-.04	.97						
Stress response X ND	.006	.06	.95						
Stress response X SD	-.03	-.28	.78						
Stress response X LD	.02	.22	.82						

Table 5: Model summary of hierarchical regression analyses from Experiment 1. Degrees of freedom vary due to missing data associated with various equipment malfunction and unusable saliva samples. Regression statistics are reported as unstandardized ts and bs.

are reported in Tables 3-5 and simple slopes are reported in Figures 9-13. The x-axis for each figure is centered at the mean across all conditions.

Stress Appraisal. There was not a significant stress appraisal x condition interaction indicating the relationship between the stress appraisal did not differ by condition. The stress appraisal was also not found to be a significant predictor of RB task performance in the ND, SD, or LD conditions, suggesting there is no significant relationship between the stress appraisal and RB task performance for any of the stress conditions (Figure 9).

HR Reactivity. There was a significant HR reactivity x condition interaction indicating that the relationship between HR reactivity and RB task performance differed by condition. However, HR reactivity was not found to be a significant predictor of RB task performance in the ND, SD, or LD

conditions, suggesting there is no significant relationship between HR reactivity and RB task performance for any of the stress conditions (Figure 10).

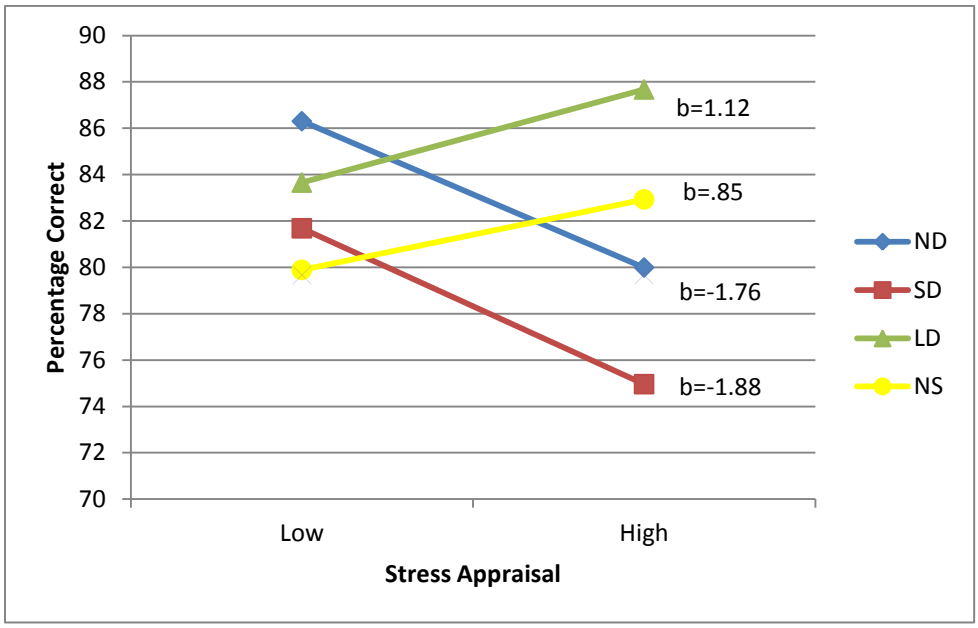

Figure 9: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean.

MAP Reactivity. There was a marginally significant MAP reactivity x condition interaction indicating that the relationship between MAP reactivity and RB task performance differed marginally by condition. MAP reactivity was found to be a significant predictor of RB task performance in the SD condition, but not the ND or LD conditions, suggesting there is a significant positive relationship between MAP reactivity and RB task performance in the SD condition, and no significant relationship between MAP reactivity and RB task performance in the ND or LD conditions (Figure 11).

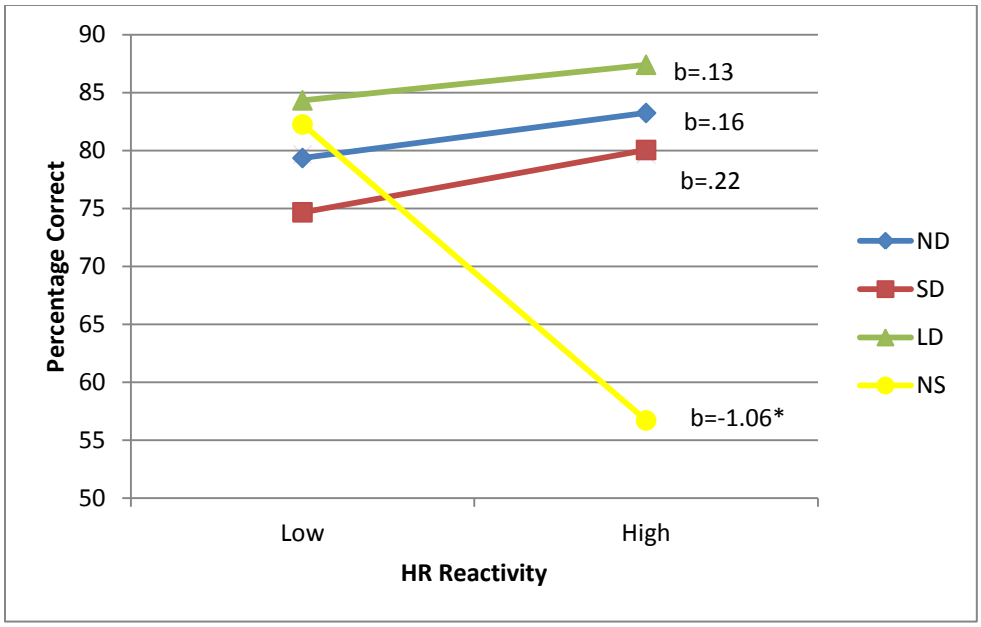

Figure 10: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean. * represents p < .05.

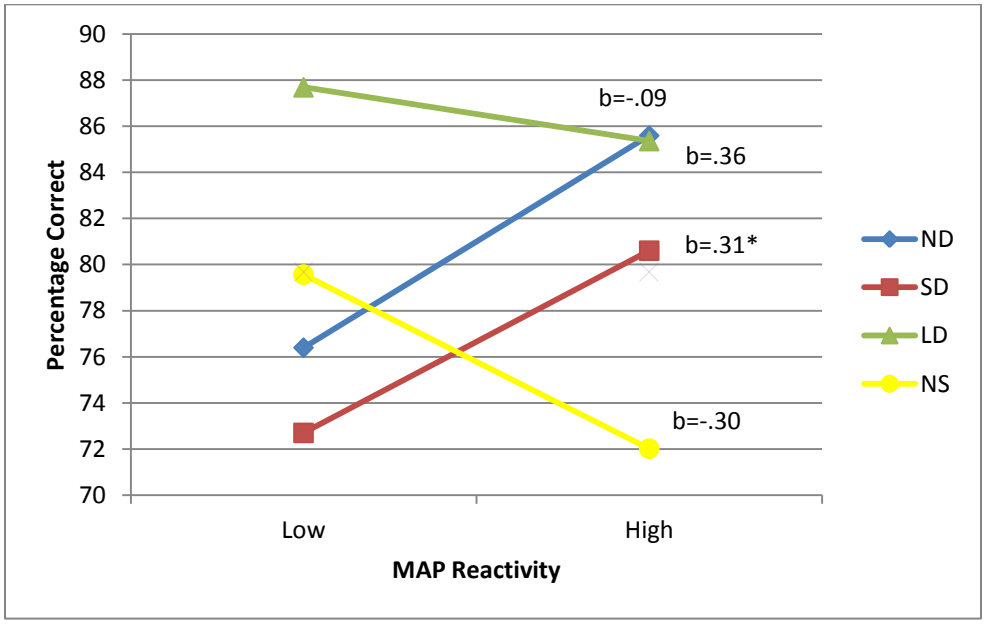

Figure 11: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean. * represents p < .05.

CORT Reactivity. There was a significant CORT reactivity x condition interaction indicating that the relationship between CORT reactivity and RB task performance differed by condition. However, CORT reactivity was found to be a significant predictor of RB task performance in the LD condition, but not the ND or SD conditions, suggesting there is a significant positive relationship between CORT reactivity and RB task performance in the LD condition, but no relationship between CORT reactivity and RB task performance in the ND and SD conditions. This result supports the prediction that HPA activation during stress is positively associated with delayed task performance, and is in line with the delayed RB task enhancement following stress (Figure 12).

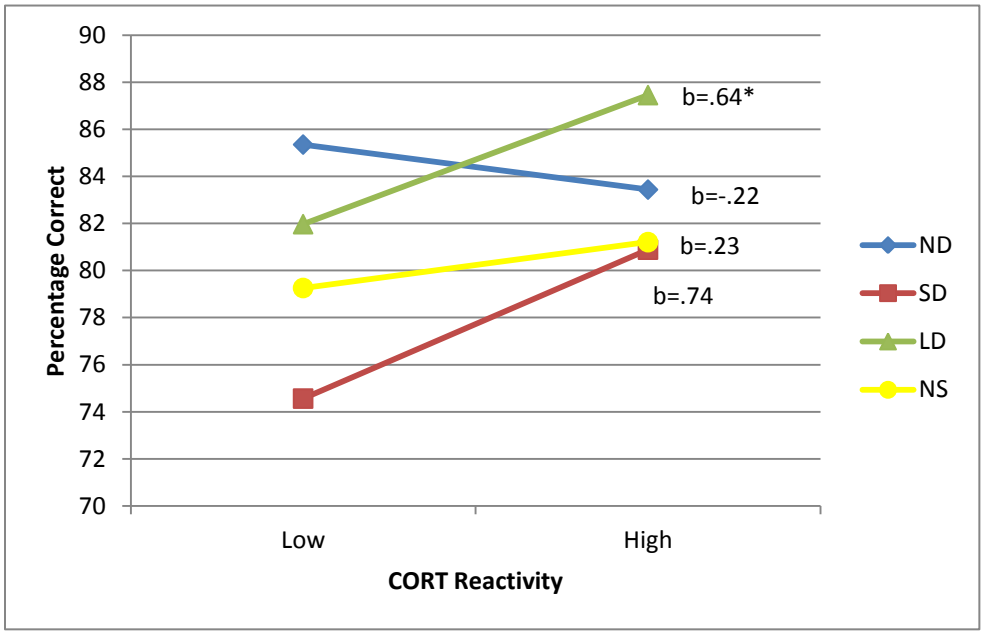

Figure 12: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean. * represents $p < .05$.

sAA Reactivity. There was not a significant sAA reactivity x condition interaction indicating that the relationship between sAA reactivity and RB task performance doesn't differ by condition. Also, sAA reactivity was not found to be a significant predictor of RB task performance in the

ND, SD, or LD conditions, suggesting there is no significant relationship between sAA reactivity and RB task performance for any of the stress conditions (Figure 13).

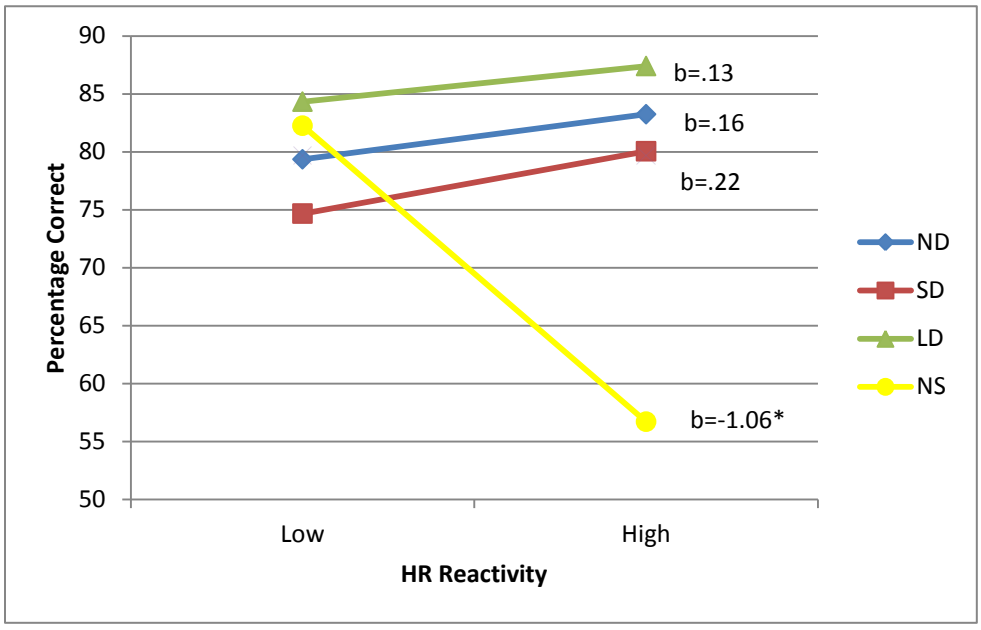

Figure 13: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean. * represents $p < .05$.

Supplemental Analyses

In order to ensure a consistently high level of data collection, Experiment 1 did not exclude OC users or women in any phase of their menstrual cycle. As suggested from Kudielka & colleagues (2009), both OC users and women in the follicular phase of their menstrual cycle should be excluded for the most consistent endocrine responses. Given this suggestion and the endocrine mediated hypotheses from Experiment 1, the following analyses will evaluate both the endocrine response and RB category learning for a subset of participants excluding participants who reported the use of OCs or being in the follicular phase of their menstrual cycle during testing (sample sizes: $ND_M = 7$, $ND_F = 3$, $SD_M = 5$, $SD_F = 4$, $LD_M = 10$, $LD_F = 3$, $NS_M = 10$, $NS_F = 2$).

Baseline Salivary Endocrine Response. As described previously baseline was chosen for each endocrine measure separately. Endocrine measure x timing condition ANOVAs conducted for baseline salivary measures suggest no main effect of timing condition for both CORT ($F(3,34) = 2.47$, $p = .08$, $MSE = 15$, $\eta_p^2 = .18$) and sAA ($F(3,34) = .691$, $p = .56$, $MSE = 2596.58$, $\eta_p^2 = .06$). The absence of a difference between conditions in baseline salivary measures allow for subsequent salivary analyses to be assessed in terms of reactivity, or a change from baseline.

Salivary Endocrine Reactivity. Reactivity scores were calculated by subtracting the salivary sample at baseline from the sample immediately following the stress manipulation (higher scores indicate higher reactivity).

A series of one sample t-tests indicated significant increase in CORT reactivity in the ND ($t(6) = 4.43$, $p = .004$), and LD ($t(10) = 3.1$, $p = .01$) but not the SD ($t(8) = 1.78$, $p = .11$) conditions. As expected no increase in CORT reactivity was found in the NS ($t(10) = -.48$, $p = .65$) condition. A CORT reactivity x timing condition ANOVA reveals a main effect of timing condition ($F(3,34) = 4.72$, $p = .007$, $MSE = 22.15$, $\eta_p^2 = .29$), and post-hoc pairwise comparisons indicate the main effect is driven by a significantly greater CORT reactivity in LD condition than the NS condition ($p < .05$) and a marginally greater CORT reactivity in the ND condition than the NS condition ($p = .059$). All other group comparisons were non-significant ($p > .05$).

A series of one sample t-tests indicated sAA reactivity was not significantly different from 0 in the ND ($t(6) = 1.92$, $p = .10$), SD ($t(8) = 1.98$, $p = .08$), LD ($t(10) = 1.64$, $p = .13$), or NS ($t(10) = -.87$, $p = .4$) conditions. A sAA reactivity x timing condition ANOVA reveals a non-significant main effect of timing condition ($F(1,34) = 1.74$, $p = .18$, $MSE = 2364.74$, $\eta_p^2 = .13$). Results indicate the stressor did not significantly induce SAM activation for the present subset of participants.

Rule-Based Task Accuracy. A four block (within) x four condition (between) mixed ANOVA was conducted for RB task accuracy. A significant main effect of block ($F(1.77, 63.86) = 26.71$, $p < .001$, MSE = 79, $\eta_p^2 = .43$) revealed an increase in accuracy across blocks (ex. block 4 accuracy was significantly higher than block 1 accuracy. $p < .05$) indicating participants were able to learn the task. Importantly, there was a significant main effect of timing condition ($F(3,74) = 3.86$, $p = .01$, MSE = 233.77, $\eta_p^2 = .14$) driven by higher accuracy in the LD condition relative to the NS and ND (p's < .05) conditions. Accuracy in the LD condition was also marginally higher than accuracy in the ND condition ($p = .057$). Inconsistent with hypotheses, the ND condition was not impaired on the task compared to the NS condition ($p > .05$). None of the remaining pairwise comparisons were significant (p's > .05). The block x condition interaction was not significant ($F(5.32, 63.86) = .73$, $p = .61$, MSE = 79, $\eta_p^2 = .06$). In sum, consistent with the findings analyzed for the entire sample, accuracy analyses suggest a delayed enhancement in RB task performance within this subset of participants.

Experiment 1 Discussion

Results from Experiment 1 provide evidence for a RB task enhancement following an extensive delay from the stressor as the LD condition performed significantly better than the NS condition. Furthermore, performance in the LD condition was associated with higher CORT reactivity during the stressor, providing support for an HPA mediated effect. These results are in line with the present hypotheses. On the contrary, findings from the ND and SD conditions were less clear. In contrast to my predictions, the ND condition was not impaired on RB task performance as compared to the NS condition, and no relationship was found between physiological responses and task performance. Similarly no differences were found in RB task performance between the SD and NS conditions and like the ND condition, no relationship was

found between task performance in the SD condition and endocrine response. This latter finding was in line with the present hypotheses.

Experiment 1 was designed to compare all stress conditions to a single comparison condition rather than using 3 comparison conditions to match the time-course of all three experimental conditions. The NS condition from Experiment 1 is best designed for comparison to the ND condition, given the comparable delay from the stressor. The NS condition may be considered an adequate comparison to the SD and LD conditions, however the difference of delay prior to testing introduces a confound to the design. It would be surprising, however it is possible that the nature of remaining in the lab may be playing a role in the enhancement in RB task performance for the LD condition compared to the NS condition. To verify the confound is not playing a role in this effect, Experiment 2 will attempt to replicate this delayed enhancement equating the delay from stress between the LD and NS conditions.

Another confound in the Experiment 1 design is created from the filler task. During the filler task participants are asked to use an online grocery shopping website to choose items that they feel like buying at the present time. All data from this task was collected for a separate project and was not designed to play any role in the present work. Participants in the LD condition completed the filler task prior to testing on the RB categorization task, whereas participants in the NS condition did not complete the filler task until after the RB category learning task creating a group difference. Given the SD condition also participated in the filler task prior the RB task, and performance in this group was not enhanced, it would be surprising if the filler task had an influence. Although unlikely to play a role, the filler

It is possible that the delayed enhanced findings from Experiment 1 are indeed related to the influence of stress on WM, however given number of sub-processes that underlay RB

category learning, alternative explanations could be made. To assess whether WM is also enhanced during the delayed time frame of Experiment 1, Experiment 2 will employ a more traditional WM task in addition to the RB category learning task.

CHAPTER 3

EXPERIMENT 2

The most interesting finding from Experiment 1 was the delayed enhancement of RB task performance. To my knowledge this was the first finding to show enhanced explicit learning following acute stress in humans. Experiment 2 will continue to examine this delayed effect of RB category learning, and will also employ a more traditional WM task (n-back). The inclusion of the n-back task will allow for the present findings to better contribute to the abundant stress-WM literature. Given the WM dependent nature of RB category learning it is possible the delayed enhancement in RB task performance is related to the relationship between stress and WM. If this is true, comparable performance differences between the RB and n-back tasks is possible. In order to build upon Experiment 1, both RB and n-back task performance in Experiment 2 will be assessed during the same lengthy delay. To address the confounded length of time between the NS and stress conditions from Experiment 1, the non-stressful comparison condition from Experiment 2 will include an equivalent delay before cognitive task assessment. A replication of the LD condition from Experiment 1 will be labeled as the "stress" condition and the non-stressful comparison condition will be labeled the "no-stress" condition for Experiment 2. Experiment 2 follows a 2 (Stress Condition) x 2 (Task) between subjects design.

I hypothesize performance in both the RB and n-back tasks will be significantly enhanced in the stress condition compared to the NS condition. This hypothesis is based upon the findings from Experiment 1 and the delayed enhancements in WM shown after hydrocortisone treatment in humans (Henckens et al., 2011) and post GR activation in rodents (Yuen et al., 2009).

Sample size goals for each group will be based upon a power analysis using the LD enhancement effect from Experiment 1. A power analysis was completed using the difference in mean accuracy between the No-Stress (M = 76.04, SD = 7.91) and the Long-Delay (M = 82.28, SD = 5.11) conditions with an effect size of (d = .937) and a one-tailed test (α = .05). According to the power analysis the correct number of participants necessary to reach a conservative power of .9 is 21 per condition. Data collection for Experiment 2 will attempt to reach this sample size for all conditions. Based upon suggestions from a review by Kudielka & colleagues (2009), Experiment 2 will use University prescreen data to best exclude participants who use OCs.

Method

Participants & Design

Participants (N = 78 undergraduate students from the University of Maine, 32 female; represents sample size after exclusions described below) arrived for a study on "Learning & Memory" and sensors to monitor cardiovascular and hemodynamic reactivity were applied (ECG, ICG, BP). Participants then relaxed for a 20 min baseline. Fourteen females reported using OCs and 26 were naturally cycling (11 luteal phase, 15 follicular phase). None of the participants in this study reported any of the following: depression, bi-polar disorder, heart-disease, obesity, panic disorder, schizophrenia, psychosis, hypertension, alcohol or drug problems, neurological problems, anxiety, irregular menstrual cycle, heart problems, or pregnancy. All participants reported normal or corrected-to-normal vision. Participants received course credit for their participation. BP response could not be collected from 27 participants due to equipment malfunction. Three participants were excluded from task analyses due to technical problems, and 2 participants who completed the categorization task were excluded as outliers performing worse than 3 SD from the mean for average accuracy across blocks. All participants were

randomly assigned to complete either a social stressor (n = 42) or a no-stress comparison condition (NS; n = 36). Participants in both the stress and no-stress conditions were randomly assigned to complete either the categorization task or n-back task following a long delay interval relative to stressor offset (NS, $n_{categorization}$ = 17, $n_{n\text{-}back}$ = 19; stress, $n_{categorization}$ = 21, $n_{n\text{-}back}$ = 21).

Social Stress Manipulation

Trier Social Stress Test. All aspects of the stress condition are the same as Experiment 1.

No-Stress TSST. The no-stress condition will now be completing the task following the same delay from the stress manipulation as the stress condition. All other aspects of the no-stress condition are the same as Experiment 1.

Stress Markers

All aspects of stress measurement were the same as Experiment 1, including stress appraisal, cardiovascular, and endocrine collection. Salivary samples were not processed due to budgetary constraints.

Rule-Based Categorization Task

All aspects of the RB task are the same as Experiment 1.

N-back Task

During every trial the participant saw a number displayed on the screen and was prompted to decide whether or not the number they just saw was the same as the number "n" trials prior (see figure 14; Chatham et al., 2011). The n alternated between 2 and 3 each block of the task, following the pattern 2-3-2-3-2-3 for every participant. For each trial, participants responded by pushing the keyboard characters 'n' and 'j' labeled "Yes" and "No", representing

either that the current number is the same as the number from trial n-back, or the current number is not the same as the number from trial n-back, respectively (see Figure 14). For example, if the current block is a 2-back block, the participant must respond whether or not the number from the current trial is the same as the number from 2 trials prior. Participants completed 6 blocks of 63 trials. The first 3 trials of 3-back blocks do not have a stimulus of n prior to refer to and thus will be excluded from all analyses. To be consistent, the first 3 trials will also be excluded from 2-back blocks, leaving 60 trials per block for analysis.

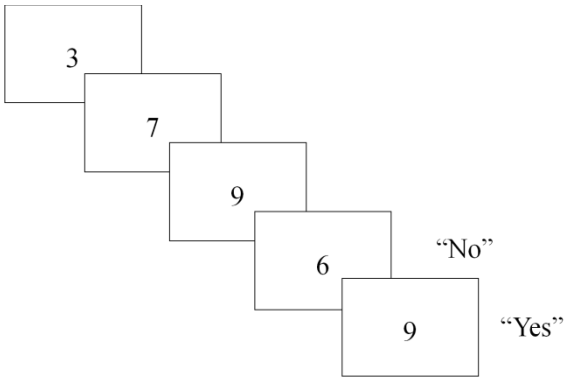

Figure 14: Representation of the hypothetical first 5 trials of a 2-back block of the n-back task. The participant sees a single stimulus on each trial, and in this case is correctly responding during trials 4 and 5.

Procedure

After providing informed consent, participants were randomly assigned to complete the RB or n-back tasks in the TSST/no-stress conditions. Following cardiovascular equipment preparation, participants were asked to relax for a 20 minute baseline period as described in Experiment 1. Following relaxation, participants randomly assigned to the stress condition took part in the modified TSST, and participants randomly assigned to the no-stress condition took part in the non stressful comparison version of the modified TSST (see Figure 15). Participants

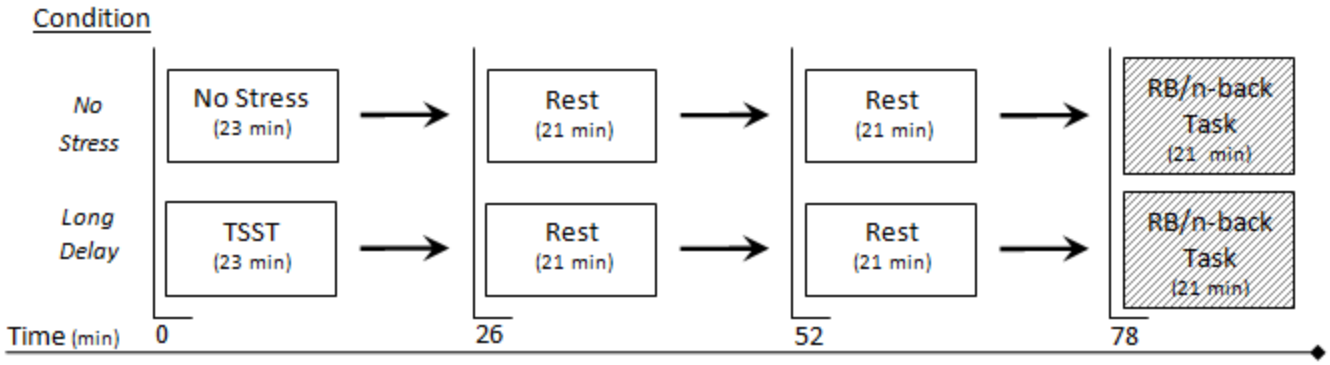

Figure 15: Procedure and timeline of Experiment 2. The rest periods are displayed separately in order to facilitate comparison with Figure 2.

then rested for 55 min, followed by participation in either the RB or n-back task. After completion of the cognitive task, all cardiovascular equipment was removed and participants were debriefed and thanked for their participation.

Results

Analyses of appraisal, cardiovascular, and salivary endocrine data as well as RB task performance are provided below. Appraisal data from self-report is designed to assess the self-perception of stress, and the cardiovascular response is meant to measure physiological arousal. Group comparisons for the RB and n-back task will provide cognitive assessment during different time frames following a delay from the stressor, and follow up regression analyses will assess the relationship between the stress measures and task performance.

Stress Markers

Stress Appraisal. After completion of the TSST, all participants were asked to rate how stressful their experience was on a likert scale from 1-7 (Figure 16). A stress appraisal x stress condition ANOVA reveals a main effect of stress condition ($F(1,73) = 59.78$, $p < .001$, $MSE = 2.37$, $\eta_p^2 = .45$) displaying that participants completing the TSST found their experience more stressful than participants completing the non-stressful comparison manipulation. As expected there was no main effect of task ($F(1,73) = 1.61$, $p = .21$, $MSE = 2.37$, $\eta_p^2 = .02$) and no stress condition x task interaction ($F(1,73) = .003$, $p = .96$, $MSE = 2.37$, $\eta_p^2 < .001$).

Baseline Cardiovascular Response. A stress condition x task ANOVA conducted on baseline HR response reveals no main effect of condition ($F(1,71) = 1.32$, $p = .26$, $MSE = 109.84$, $\eta_p^2 = .02$) or task ($F(1,71) = .76$, $p = .39$, $MSE = 109.84$, $\eta_p^2 = .01$) and no condition x task interaction ($F(1,71) =$

3.07, p = .08, MSE = 109.84, η_p^2 = .04). These results are consistent with the expected null differences of HR responses during baseline.

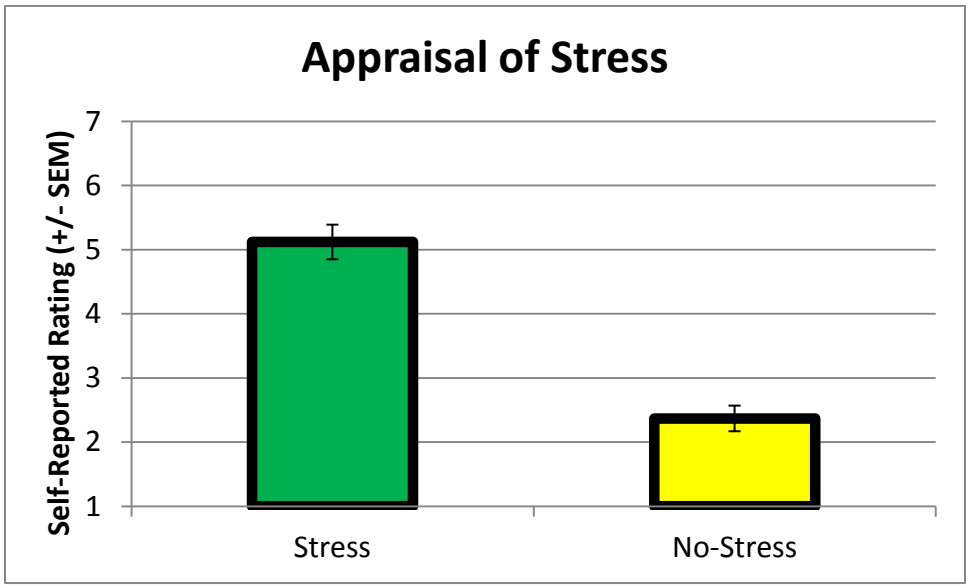

Figure 16: Stress appraisal data from Experiment 2.

A stress condition x task ANOVA conducted on baseline MAP response reveals no main effect of condition (F(1,44) = 2, p = .17, MSE = 174.25, η_p^2 = .04) or task (F(1,44) = .27, p = .61, MSE = 174.25, η_p^2 = .006) and no stress condition x task interaction (F(1,44) = .54, p = .47, MSE = 174.25, η_p^2 = .01). These results confirm the expected null differences of MAP responses during baseline.

Cardiovascular Reactivity. As in Experiment 1, all reactivity scores were calculated by subtracting the average cardiovascular response during the last 2 min of baseline from the last 2 min of the stress manipulation (higher scores indicate higher reactivity). A set of one-sample t-tests reveal both the stress (t(40) = 7.97, p < .001) and no-stress (t(33) = 5.64, p < .001) conditions had significant HR reactivity relative to 0. A stress condition x task ANOVA reveals a

main effect of stress condition ($F(1,71) = 14.09$, $p < .001$, $MSE = 127.14$, $\eta_p^2 = .17$) in that participants in the stress condition had a higher HR reactivity compared to the no-stress condition (Figure 17). No main effect of task ($F(1,71) = 3.05$, $p = .09$, $MSE = 127.14$, $\eta_p^2 = .04$) or condition x task interaction were found ($F(1,71) = .18$, $p = .67$, $MSE = 127.14$, $\eta_p^2 = .003$). Taken together these results indicate the TSST was more stressful as measured by HR than the no-stress TSST.

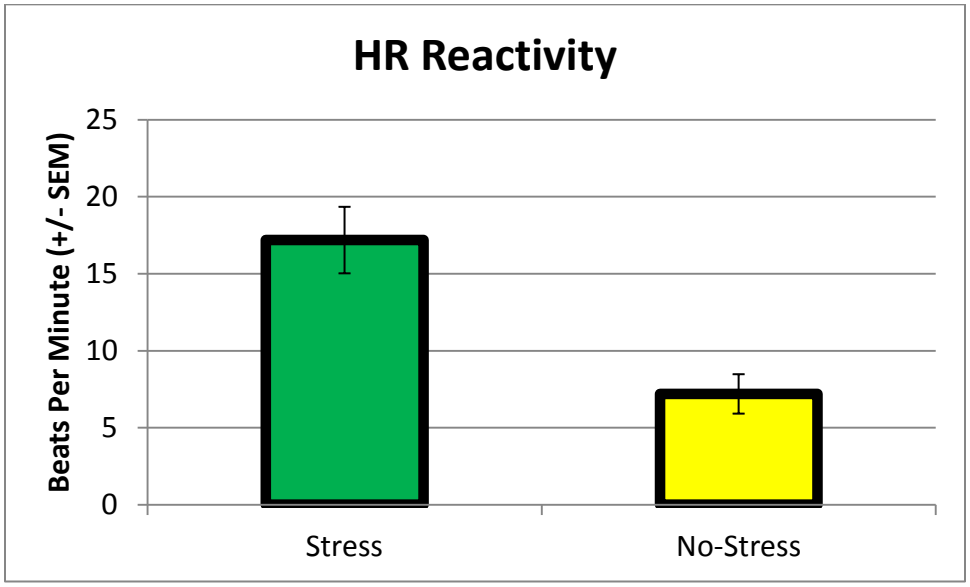

Figure 17: HR reactivity data from Experiment 2.

A set of one sample t-tests revealed significant MAP reactivity in the stress condition ($t(19) = 7.11$, $p < .001$) and marginally significant MAP reactivity in the no-stress condition ($t(22) = 2.06$, $p = .052$). A stress condition x task ANOVA analyzing MAP reactivity between groups revealed a main effect of stress condition ($F(1,39) = 19.94$, $p < .001$, $MSE = 113.28$, $\eta_p^2 = .34$) but no main effect of task ($F(1,39) = .7$, $p = .41$, $MSE = 113.28$, $\eta_p^2 = .02$) or stress condition x task interaction ($F(1,39) = .11$, $p = .74$, $MSE = 113.28$, $\eta_p^2 = .003$; Figure 18). Taken together these results indicate the TSST was more stressful as measured by MAP than the no-stress TSST.

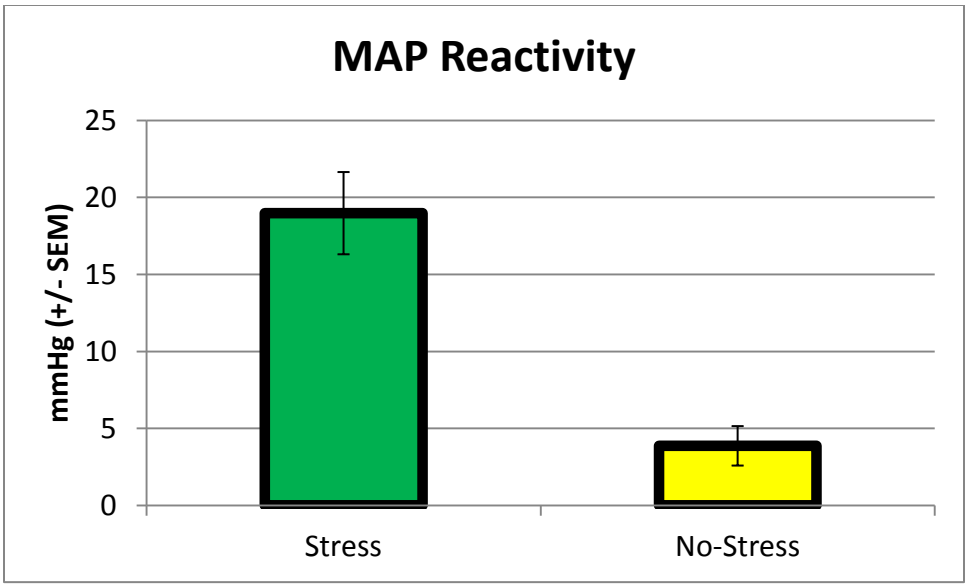

Figure 18: MAP reactivity data from Experiment 2.

Cognitive Task Performance

Rule-Based Task Accuracy. A four block (within) x two stress condition (between) mixed ANOVA was conducted on RB task accuracy to compare performance between the stress and no-stress conditions (Figure 19). A significant main effect of block ($F(2.47, 88.8) = 23.13$, $p < .001$, $MSE = 38.83$, $\eta_p^2 = .39$) revealed participants were successful in learning the task. Importantly there was a main effect of stress condition ($F(1,36) = 4.59$, $p = .04$, $MSE = 102.5$, $\eta_p^2 = .11$) driven by higher task accuracy in the stress condition than the NS condition. The block x stress condition interaction was not significant ($F(2.467, 88.8) = .36$, $p = .74$, $MSE = 38.83$, $\eta_p^2 = .01$). Taken together these results are in line with my hypothesis and replicate Experiment 1 suggesting participants had enhanced RB task performance following a delay from stress as compared to a non-stress comparison group.

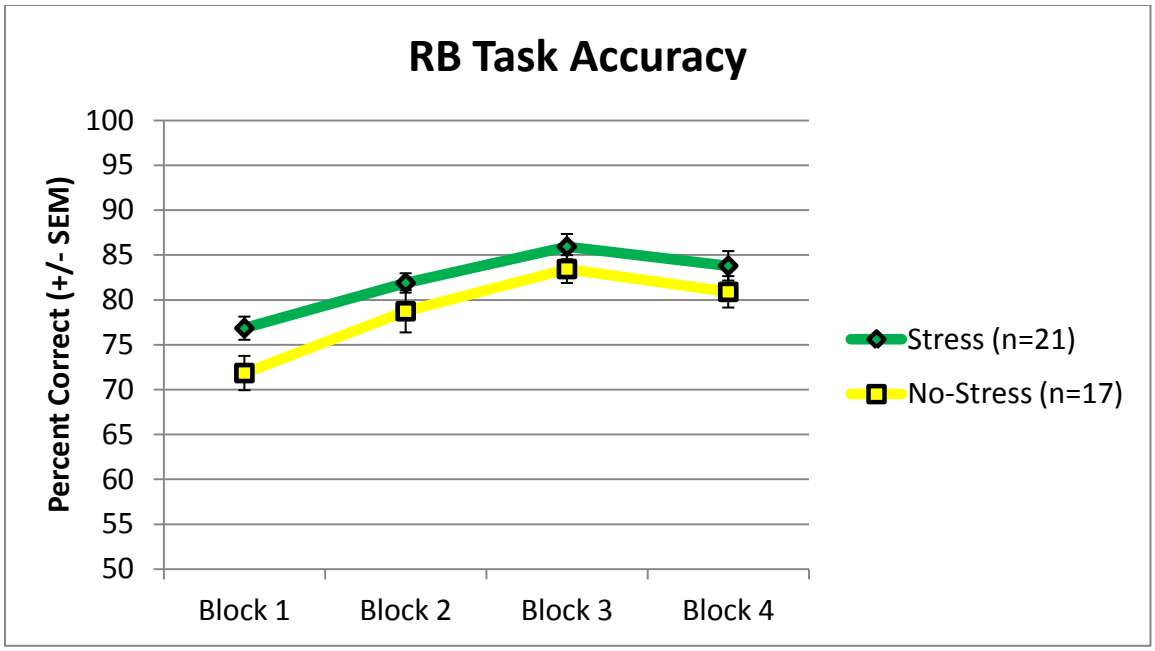

Figure 19: RB task accuracy from Experiment 2.

N-back Task Accuracy. To analyze performance on the n-back task each of the 2-back and each of the 3-back blocks were combined. A repeated measures ANOVA comparing task accuracy for the stress condition and the NS condition using block type (2-back vs. 3-back) as the repeated measures factor was analyzed (Figure 20). A main effect of block type ($F(1,38) = 53.51$, $p < .001$, $MSE = 25.81$, $\eta_p^2 = .59$) was found, revealing higher performance during the 2-back blocks ($M = 84.29$, $SD = 11.04$) than 3-back blocks ($M = 75.85$, $SD = 12.1$). No main effect of stress condition ($F(1,38) = .99$, $p = .33$, $MSE = 241.94$, $\eta_p^2 = .025$), or block type x stress condition interaction ($F(1,38) = 1.7$, $p = .2$, $MSE = 25.81$, $\eta_p^2 = .04$) were found indicating n-back task performance was not different between the stress and NS conditions. This finding suggests stress did not have an impact on n-back task performance.

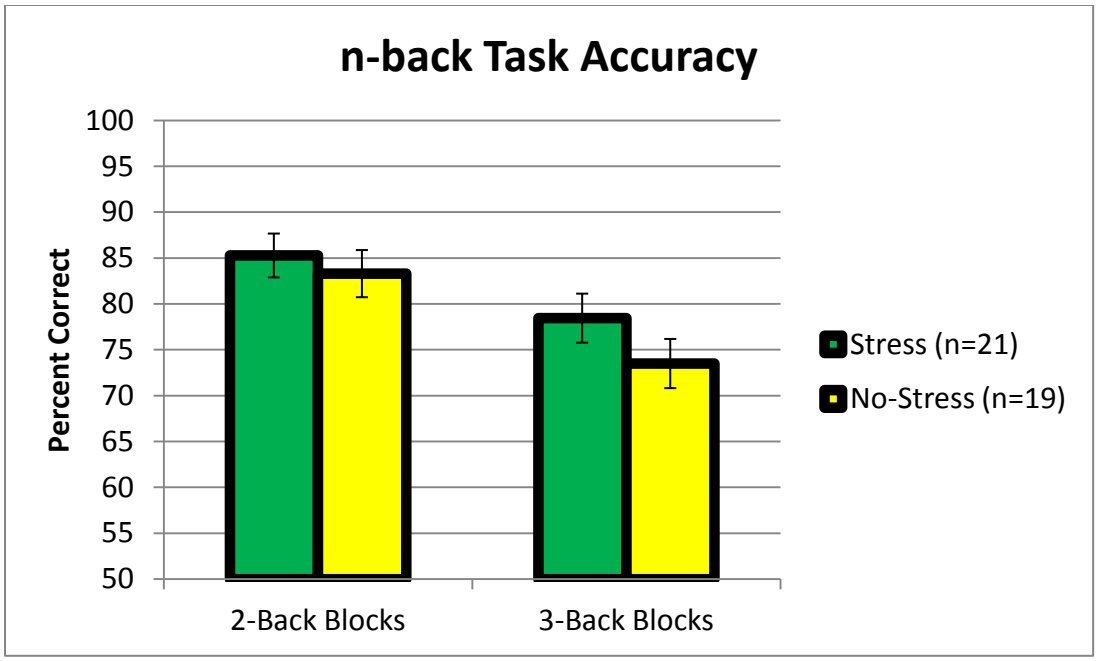

Figure 20: n-back task accuracy from Experiment 2.

As an alternative measure of performance, early signal detection theory literature (Green & Swets, (1966); Nevin (1969)) proposes the assessment of d' to estimate the ease of detecting a target stimulus. The measure of d' includes both false alarm trials, that is responding 'yes' when the correct response was 'no, and hits, or responding 'yes' when the correct response was 'yes'. For the n-back task d' was calculated by subtracting the z transformed rate of false alarms from the z transformed rate of hits. An independent samples t-test revealed participants in the stress condition were not different from the NS condition at detecting correct stimuli ($t(38) = 1.31$, $p = .2$).

Rule-Based Task Strategy. RB task strategy was analyzed using the same procedure as in Experiment 1. See the Appendix for a description of the models and fitting procedures.

To be consistent with Experiment 1, strategy analyses will focus on block 4 of the task as to provide ample time for strategy development [6]. Participants in both the stress and NS conditions were more likely to use conjunctive strategies than unidimensional strategies (see Table 6), and a chi-squared analysis revealed no difference between the conditions in participants' reliance of one strategy over another ($\chi^2(1,38) = .003$, $p = .96$).

Table 6: Participant Strategy Usage Counts

Strategy Use During Block 4		
	Stress	*No-Stress*
Unidimensional	6	5
Conjunctive	15	12

Relationship Between Stress and RB Task Performance

To fully understand the impact of stress on RB category learning it is important to determine the relationship between the stress response, both physiological and appraised, and RB task performance. In order to assess RB task accuracy at a time where performance had stabilized, all of the following regression analyses concentrate on block 4 task only[7]. The following hierarchical regression model (Step1 predictors: stress condition, stress response; Step2 predictors: stress response, stress condition x stress response interaction; Outcome variable: block 4 accuracy) were designed to determine the extent to which stress responses

[6] Block 4 was chosen because this is the time when participants have best learned the task. The random responder model and the general linear classifier provided the best fit for 0 participants each. Thus, these models were omitted from analyses.

[7] A RB task performance x stress condition ANOVA comparing block 4 RB task accuracy revealed no effect of condition ($F(1,36) = 1.45$, $p = .237$, $MSE = 54.53$, $\eta_p^2 = .039$).

Model summary of hierarchical regression analyses from Experiment 2.

Variable	Stress Appraisal - RB Task										HR Reactivity - RB Task								
	b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2		b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2
Step 1				.46	.64			.03						2.02	.15			.11	
Stress condition	2.4	.72	.48								1.11	.41	.68						
Stress response	-.01	-.02	.99								.17	1.6	.12						
Step 2						3.4	.07	.12	.09							.53	.47	.12	.01
Stress condition	-8.67	-.59	.21								3.25	.81	.42						
Stress response	-2.17	-.6	.14								.31	1.41	.17						
Stress response X stress condition	3.17	1.28	.07								-.19	-.73	.47						

Table 7: Model summary of hierarchical regression analyses from Experiment 2. Degrees of freedom vary due to missing data associated with various equipment malfunction. Regression statistics are reported as unstandardized ts and bs.

Model summary of hierarchical regression analyses from Experiment 2.

Variable	MAP Reactivity - RB Task										Stress Appraisal - N-back								
	b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2		b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2
Step 1				1.45	.26			.13						5.25	.01			.22	
Stress condition	1.76	.49	.63								-12.14	-2.86	.007						
Stress response	.16	1.07	.3								3.16	3.05	.004						
Step 2						.71	.41	.16	.03							1.28	.27	.25	.03
Stress condition	4.26	.91	.38								-19.91	-2.47	.019						
Stress response	.29	1.34	.2								.98	.45	.66						
Stress response X stress condition	-.25	-.84	.41								2.81	1.13	.27						

Table 8: Model summary of hierarchical regression analyses from Experiment 2. Degrees of freedom vary due to missing data associated with various equipment malfunction. Regression statistics are reported as unstandardized ts and bs.

Model summary of hierarchical regression analyses from Experiment 2.

Variable	HR Reactivity - N-back										MAP Reactivity - N-back								
	b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2		b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2
Step 1				.85	.44			.05						.94	.41			.1	
Stress condition	-5.02	-1.29	.21								-5.09	-.95	.36						
Stress response	.06	.34	.74								.28	1.36	.19						
Step 2						4.28	.046	.15	.11							.0	.97	.1	.0
Stress condition	1.47	.3	.77								-4.92	-.69	.5						
Stress response	.87	2.05	.048								.29	.78	.45						
Stress response X stress condition	-.95	-2.07	.046								-.02	-.04	.97						

Table 9: Model summary of hierarchical regression analyses from Experiment 2. Degrees of freedom vary due to missing data associated with various equipment malfunction. Regression statistics are reported as unstandardized ts and bs.

moderated the relationship between condition and RB task accuracy. Each model was dummy coded such that the stress condition can be compared to the no-stress condition and was evaluated separately for each stress response variable (i.e., Stress Appraisal, HR reactivity, MAP reactivity). Thus, the results of the step 1 analysis will assess the main effect of stress condition for the participants represented in each in each model. The results of the step 2 analysis will assess if the relationship between the stress response and RB task accuracy differs by condition for each stress response variable. Because the primary focus of this analysis is to understand the moderating effects of the stress response variables, I will focus the description of the results on the stress response x condition interaction at step 2. To further characterize each interaction, simple slopes were estimated for both the stress and no-stress conditions. The simple slope analysis allows for the investigation of the relationship between stress reactivity and accuracy separately within each condition. For simplicity, all statistics (for both steps) are reported in Tables 7-8 and simple slopes are reported in Figures 21-23. The x-axis for each figure is centered at the mean across both conditions.

Stress Appraisal. There was a marginally significant stress appraisal x condition interaction indicating that the relationship between the stress appraisal and RB task performance marginally differs by condition. Also, the stress appraisal was not found to be a significant predictor of RB task performance in the stress condition, suggesting there is no significant relationship between stress appraisal and RB task performance for the stress condition (Figure 21).

HR Reactivity. There was no significant HR reactivity x condition interaction indicating that the relationship between HR reactivity and RB task performance doesn't differ by condition. Also, HR reactivity was not found to be a significant predictor of RB task performance in the stress

condition, suggesting there is no significant relationship between HR reactivity and RB task performance for the stress condition (Figure 22).

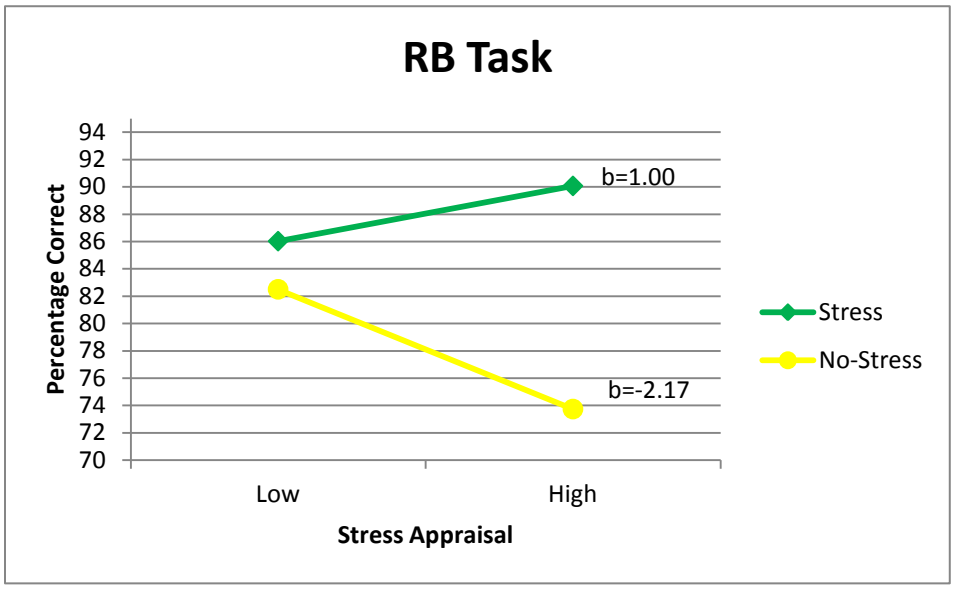

Figure 21: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean.

MAP Reactivity. There was no significant MAP reactivity x condition interaction indicating that the relationship between MAP reactivity and RB task performance doesn't differ by condition. Also, MAP reactivity was not found to be a significant predictor of RB task performance in the stress condition, suggesting there is no significant relationship between MAP reactivity and RB task performance for the stress condition (Figure 23).

Relationship Between Stress and N-back Task Performance

Like the RB task, I will also consider the relationship between stress, both physiological and appraised, and n-back task performance. Given that the n-back task does not assess learning performance as in the RB task, but instead WM performance, the following analyses

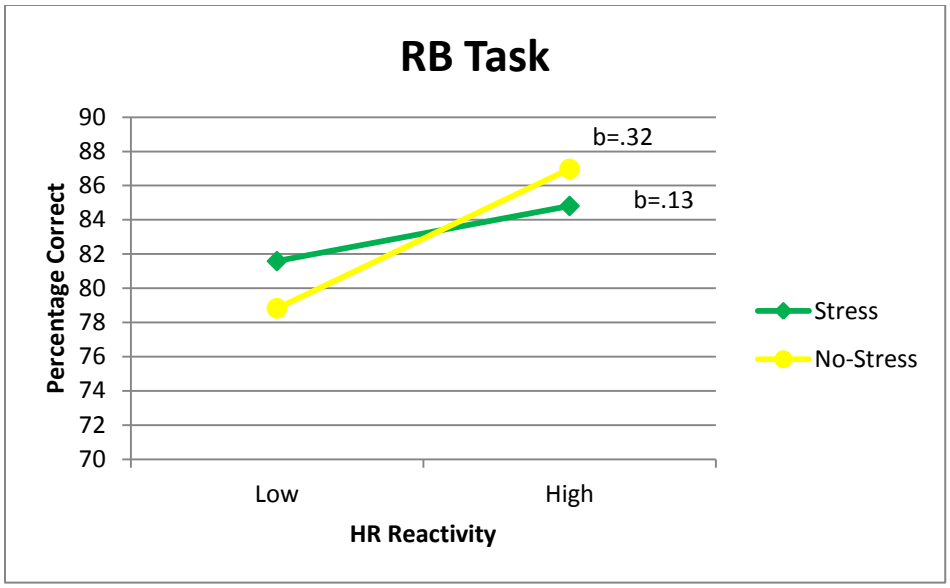

Figure 22: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean.

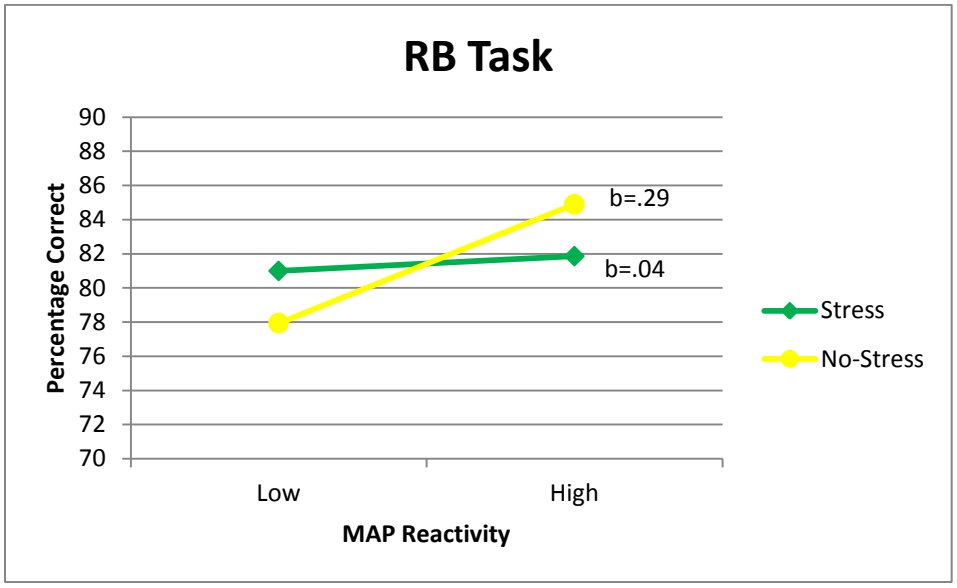

Figure 23: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean.

will address performance as a mean of accuracy for all blocks of the task[8]. The following hierarchical regression model (Step1 predictors: stress condition, stress response; Step2 predictors: stress response, stress condition x stress response interaction; Outcome variable: n-back task accuracy) were designed to determine the extent to which stress responses moderated the relationship between condition and n-back task accuracy. Each model was dummy coded such that the stress condition can be compared to the no-stress condition and was evaluated separately for each stress response variable (i.e., Stress Appraisal, HR reactivity, MAP reactivity). Thus, the results of the step 1 analysis will assess the main effect of stress condition for the participants represented in each in each model. The results of the step 2 analysis will assess if the relationship between the stress response and n-back task accuracy differs by condition for each stress response variable. Because the primary focus of this analysis is to understand the moderating effects of the stress response variables, I will focus the description of the results on the stress response x condition interaction at step 2. To further characterize each interaction, simple slopes were estimated for both the stress and no-stress conditions. The simple slope analysis allows for the investigation of the relationship between stress reactivity and accuracy separately within each condition. For simplicity, all statistics (for both steps) are reported in Tables 8-9 and simple slopes are reported in Figures 24-26. The x-axis for each figure is centered at the mean across both conditions.

Stress Appraisal. There was no significant stress appraisal x condition interaction indicating that the relationship between the stress appraisal and n-back task performance doesn't differ by condition. However, the stress appraisal was found to be a significant predictor of n-back task

[8] A stress condition x n-back task performance (collapses across all blocks) ANOVA revealed no effect of condition ($F(1,38) = .99$, $p = .326$, $MSE = 120.97$, $\eta_p^2 = .03$). In sum, and different than the aforementioned repeated measures ANOVA results, findings did not reveal a significant difference between conditions, indicating performance was not different following stress or a non-stressful comparison.

performance in the stress condition, suggesting there is a significant positive relationship between the stress appraisal and n-back performance for the stress condition (Figure 24).

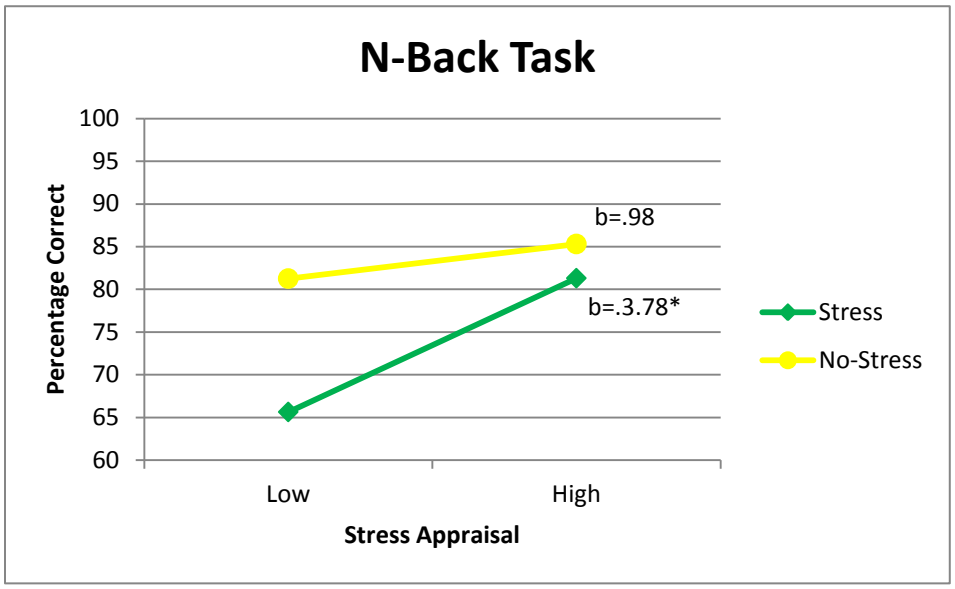

Figure 24: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean. * represents $p < .05$.

HR Reactivity. There was a significant HR reactivity x condition interaction indicating that the relationship between HR reactivity and n-back performance differs by condition. However, HR reactivity was not found to be a significant predictor of n-back task performance in the stress condition, suggesting there is no significant relationship between HR reactivity and n-back task performance for the stress condition (Figure 25).

MAP Reactivity. There was no significant MAP reactivity x condition interaction indicating that the relationship between MAP reactivity and n-back task performance doesn't differ by condition. Also, MAP reactivity was not found to be a significant predictor of n-back task

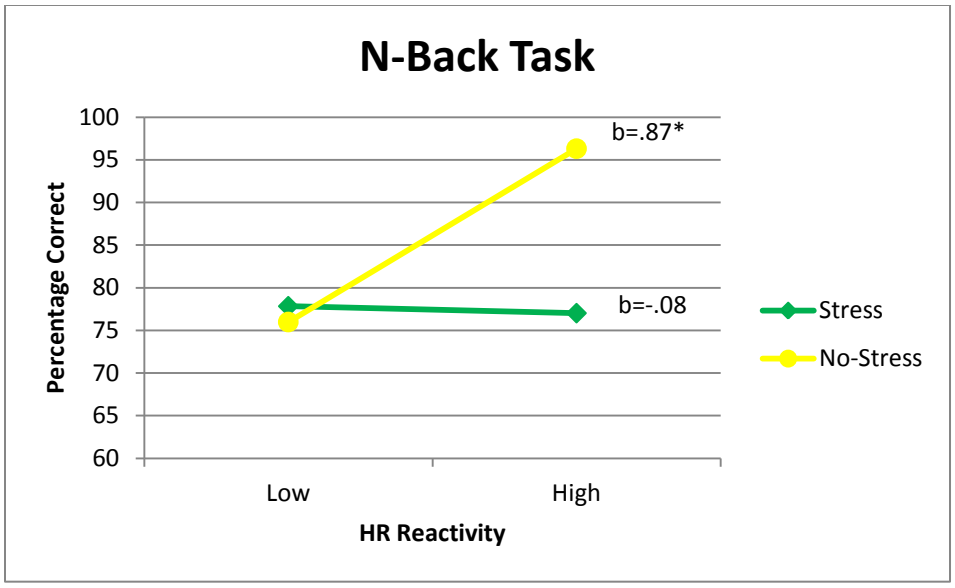

Figure 25: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean. * represents $p < .05$.

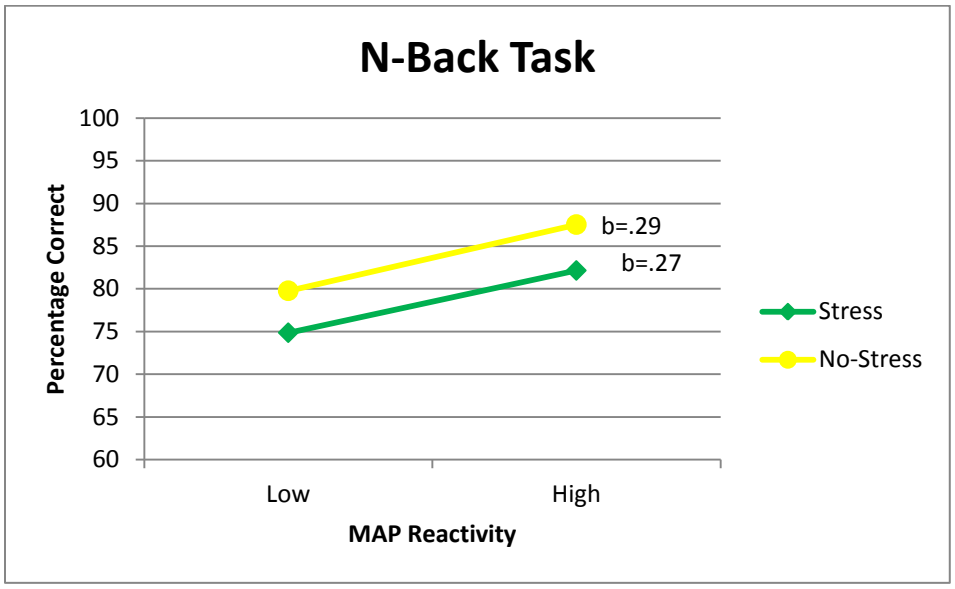

Figure 26: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean.

performance in the stress condition, suggesting there is no significant relationship between MAP reactivity and n-back task performance for the stress condition (Figure 26).

Supplemental Analyses

In an attempt to collect the best data as possible Experiment 2 only recruited participants who had not reported the use of OCs for an early semester subject pool prescreen. As suggested from Kudielka & colleagues (2009), both OC users and women in the follicular phase of their menstrual cycle should be excluded for the most consistent endocrine responses. Given this suggestion and the endocrine mediated hypotheses from Experiment 2, the following analyses will evaluate both the RB and n-back tasks for a subset of participants excluding participants who reported the use of OCs or being in the follicular phase of their menstrual cycle during testing(sample sizes: RB task: $Stress_M$ = 8, $Stress_F$ = 2, No-$Stress_M$ = 12, No-$Stress_F$ = 1; N-back: $Stress_M$ = 16, $Stress_F$ = 2, No-$Stress_M$ = 11, No-$Stress_F$ = 3).

Rule-Based Task Accuracy. A four block (within) x two stress condition (between) mixed ANOVA was conducted on RB task accuracy to compare performance between the stress and no-stress conditions. A significant main effect of block ($F(1.87, 35.6) = 14.92$, $p < .001$, $MSE = 53.29$, $\eta_p^2 = .44$) revealed participants were successful in learning the task. No main effect of stress condition was found ($F(1,19) = .47$, $p = .5$, $MSE = 84.28$, $\eta_p^2 = .02$) and the block x stress condition interaction was not significant ($F(1.87, 35.6) = .2$, $p = .81$, $MSE = 53.29$, $\eta_p^2 = .01$). Taken together these results suggest although a main effect of stress condition was found with the entire sample, it was not present in this subset of participants.

N-back Task Accuracy. To analyze performance on the n-back task each of the 2-back and each of the 3-back blocks were combined. A repeated measures ANOVA comparing task accuracy for the stress condition and the NS condition using block type (2-back vs. 3-back) as the repeated

measures factor was analyzed. A main effect of block type ($F(1,29) = 39.84$, $p < .001$, $MSE = 29.46$, $\eta_p^2 = .58$) was found, revealing higher performance during the 2-back blocks ($M = 85.39$, $SD = 11.57$) than 3-back blocks ($M = 76.39$, $SD = 13.33$). A marginal effect of stress condition ($F(1,29) = 3.52$, $p = .07$, $MSE = 260.36$, $\eta_p^2 = .11$) suggests participants in the stress condition performed marginally worse on the n-back task than the no-stress condition. No block type x stress condition interaction ($F(1,29) = .66$, $p = .42$, $MSE = 29.46$, $\eta_p^2 = .02$) was found.

Experiment 2 Discussion

Experiment 2 was designed in attempt to replicate a delayed enhancement in RB category learning following the stressor found in Experiment 1, and assess whether these enhancements will extend to a traditional WM task. Findings from Experiment 2 replicate the delayed RB category learning enhancement from Experiment 1. This successful replication is important and provides stronger evidence for the legitimacy of this finding. Contrary to the influence of stress on RB category learning, results did not reveal a significant enhancement in WM task performance following the stressor. This finding is unexpected given the present hypothesis, and previous literature suggesting WM is enhanced following a delay from stress (Henckens et al., 2011, Oei et al., 2009; Yuen et al., 2009) . Potential explanations for these results will be discussed in the general discussion.

The RB task enhancement replication provides support for the notion that the influence of stress was truly enhancing and not an artifact of type 1 error. Furthermore this finding gives support that the potential confounds from Experiment 1, including differing post stressor delays, and potential filler task influence, did not significantly affect the delayed RB task enhancement. Due to funding obstacles, saliva samples have yet to be analyzed for Experiment 2, and thus it is unclear if the relationship between CORT and RB task performance remains.

Overall the findings from Experiment 2 provide evidence of a delayed RB task enhancement 78 min following stressor onset. This finding bolsters the original delayed enhancement found in Experiment 1, however these enhancements were not extended to the n-back task. Given these differences it seems as if the relationship between stress and both RB category learning and WM are not identical. Taking a different approach, Experiment 3 will attempt to explore if this RB category learning enhancement extends to a time point previously shown to enhance WM task performance (Henckens et al., 2011; Yuen et al., 2009).

CHAPTER 4

EXPERIMENT 3

The results from Experiment 2 may suggest the effects stress on WM was not the same as the effects on WM performance. Experiment 2 looked at this by assessing WM at the time frame previously shown to impair RB category learning, however another approach is to assess RB task performance during the time frame previously shown to enhance WM task performance following stress. Taking this approach, Experiment 3 will assess if RB category learning enhancements remain for a prolonged length of time comparable to previously found genomic WM enhancements(Henckens et al., 2011; Yuen et al., 2009). Thus Experiment 3 will evaluate task performance during a similar delay from the LD condition in Experiment 1 and stress condition in Experiment 2 (phase 1) as well as a time point ~4 hrs post-stress in an attempt to capture the genomic influence of stress. Instead of using the TSST, Experiment 3 will incorporate a socially evaluated CPT manipulation to investigate whether or not the delayed enhancement of RB category learning is stressor specific. Performance following the CPT will be compared to a warm water comparison condition (WPT).

I hypothesize that RB task performance will be enhanced at both phases following the CPT condition as compared to the WPT condition. This hypothesis is based upon the findings of a delayed RB task enhancement from Experiments 1 and 2 and delayed enhancements in WM post hydrocortisone treatment in found in humans (Henckens et al., 2011) and post GR activation in rodents (Yuen et al., 2009).

Just as in Experiment 2 sample size goals for each group will be based upon a power analysis using the LD enhancement effect from Experiment 1. According to the power analysis the correct number of participants necessary to reach a conservative power of .9 is 21 per

condition. Also, Experiment 2 will use University prescreen data to best exclude participants who use OCs.

Method

Participants & Design

Participants (N = 56 undergraduate students from the University of Maine, 24 female; Age, M = 19.46, SD = 2.15; represents sample size after exclusions described below) arrived for a study on "Circles & Boxes". Nine females reported using OCs and 19 reported as naturally cycling (14 luteal phase, 5 follicular phase). None of the participants in this study reported any of the following: depression, bi-polar disorder, heart-disease, obesity, panic disorder, schizophrenia, psychosis, hypertension, alcohol or drug problems, neurological problems, anxiety, irregular menstrual cycle, heart problems, or pregnancy. All participants reported normal or corrected-to-normal vision. Participants received course credit for their participation. All participants were randomly assigned to complete either a cold-pressor task (CPT; n = 26) or a WPT (n = 30). The experiment was broken up into 2 phases, separated by ~3 hrs within a single day. One participant was excluded as an outlier due to performing worse than 3 SD from the mean on the cognitive task during phase 1, and will be removed from all analyses. During phase 2, one participant was recorded as an outlier due to performing worse than 3 SD from the mean on the cognitive task, one participant had technical issues during the completion of the task, and 4 participants did not return to the lab. These participants will all be included for data collected during phase 1 of the experiment only, leaving 50 participants for phase 2 analyses ($n_{WPT} = 26$, $n_{CPT} = 24$).

Stress Manipulation

Cold Pressor Task. Participants randomly assigned to complete the socially evaluated cold pressor task (CPT) were required to submerge their non-dominant hand and wrist in cold water (water temperature (Fahrenheit): M = 34°, SD = 2.25, range = 30° - 38°) for up to 3 min while being recorded on video and in the presence of a female experimenter (Schwabe et al., 2008b). A small plastic container was used to hold the water, and was refrigerated until the desired temperature. Any ice present was removed before the participant immersed their hand. The participant was asked to sit in a comfortable chair and instructed to face and look at a laptop camera. The participant was told they would be recorded on video so their facial expressions could be analyzed at a later time. The experimenter also remained in the room facing the participant as to further supply the feeling of evaluation during the manipulation. Participants were free to remove their hand from the cold water if they are uncomfortable at any time without consequence.

Warm Pressor Task. Participants assigned to complete the WPT had exactly the same experience as the CPT except the water was warm (water temperature (Fahrenheit): M = 97.41°, SD = 2.70, range = 94° - 101°), they were not recorded on video, and the experimenter was not present. This manipulation is meant as a comparison condition excluding the stressful aspects of the task (see Schwabe et al., 2008b). Again participants were told they are free to remove their hand at any time without consequence.

Stress Markers

Stress Appraisal. To assess the efficacy of the stress manipulation, participants were asked to rate (immediately after the CPT or WPT followed by a 3 min saliva sample) the extent to which they found the experience to be stressful, unpleasant, painful, challenging, and threatening (on

a 1 “strongly disagree” to 7 “strongly agree” scale). The latter two ratings were included for comparison to an ongoing project and, therefore, only the stressfulness, painfulness, and unpleasantness ratings will be analyzed.

Salivary Endocrine Measures. All aspects of endocrine measurement will be the same as Experiment 1. Salivary samples were not processed due to budgetary constraints.

Rule-Based Categorization Task

Participants completed the RB categorization task twice throughout the experiment . As to not become overly familiar with the task, a new set of stimuli were included (see Table 10 for the category parameters and Figure 27 for new stimuli examples). In addition to the sine wave gratings from Experiments 1 and 2, participants also categorized unframed rectangles containing green lit pixels ("density boxes"; stimuli adopted from Smith et al., 2013). Eighty-four stimuli were used with 42 assigned to each of the two response categories. To create these category structures, a variation of the randomization technique (Ashby & Gott, 1988) was used. Each cluster of stimuli was defined as a bivariate uniform distribution with a minimum and maximum on each dimension and was assigned to either category A or category B

Each stimulus (labeled: density boxes) was generated (offline) by drawing a random sample (*x, y*). Stimulus rectangles varied in size and pixel density. Both dimensions had 101 levels (Levels 0-100). Rectangle width (W) and height (H; in screen pixels) were calculated as 2 * *level* + 100 and level + 50, respectively. Thus, rectangle size ranged from W-100 x H-50 (Level 0) to W-300 x H-150 (Level 100). Pixel density, that is the proportion of pixel position that were illuminated, was calculated as $0.05 \times 1.018^{level}$. Thus, density varied from .0500 (Level 0) to .2977 (Level 100). Stimuli showing the range of the dimensions can be seen in figure 27. Each stimulus

Table 10: Parameters Used to Generate the Figure 2b Categories

Category Cluster	**Size**		**Density**		**Number Per Cluster**
	Min	*Max*	*Min*	*Max*	
Category A	0	50	50	100	14
Category A	50	100	50	100	14
Category A	0	50	0	50	14
Category B	50	100	0	50	42

Note. Size and Density values are in arbitrary units. The number of stimuli per cluster was chosen in order to ensure equal category base rates.

was presented on a black background and subtended a visual angle of 4.35° at a viewing distance of approximately 51 cm. The stimuli were generated and presented on a 20-in. LCD with 800 x 600 resolution using the Psychophysics Toolbox extensions (Brainard, 1997; Pelli, 1997) for MATLAB.

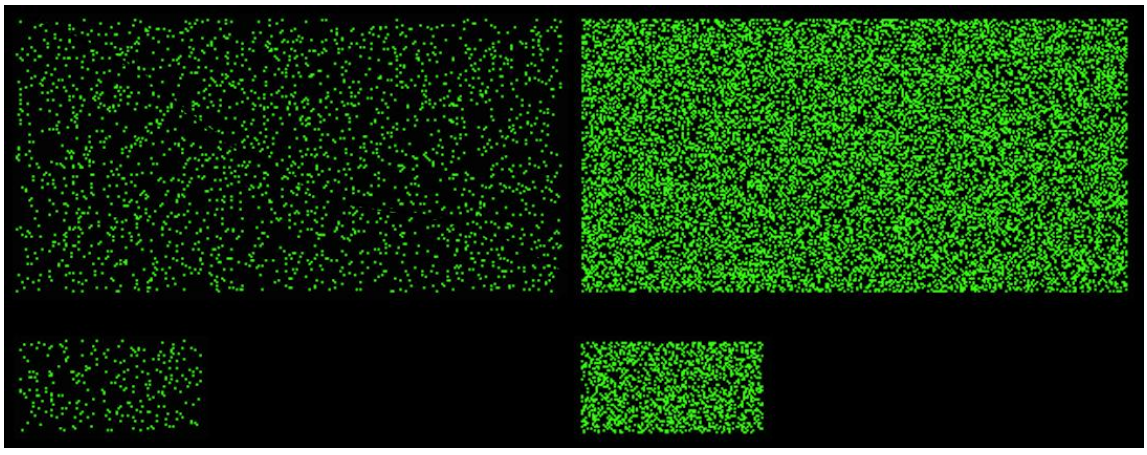

Figure 27: Density box stimuli examples from Experiment 3. Size ranges from level 0 (bottom) to level 100 (top). Density ranges from level 0 (left) to level 100 (right).

Procedure

Participants arrived at the lab between 12:00 and 1:00 pm. After completing an informed consent, all participants partook in a 20 minute relaxation period as described in Experiment 1 (see Figure 28). Following baseline, participants were randomly assigned to complete either the CPT or WPT. Following the manipulation, and 60 minutes of rest, all participants proceeded to complete the RB category learning task for the first time and were randomly assigned to either categorizing sine wave gratings or density boxes (counterbalanced). Following completion of phase 1, participants left the lab and were asked to return 3 hrs later. Upon returning (M = 183 min later), participants began phase 2 of the experiment; during this time they again completed the RB task (M = 279 min post CPT onset), using the remaining stimulus set (e.g., density boxes if grating were used during phase 1). After completion of the RB task and post-task questionnaires the participants were be debriefed and thanked for their participation.

Results

Stress Markers

Stress Appraisal. Participants were asked a number of questions to assess their perception of the experience during the stress manipulation. Separate stress appraisal x stress condition ANOVAs reveal the CPT to be reported as significantly more stressful ($F(1,54) = 70.7$, $p < .001$, $MSE = 1.37$, $\eta_p^2 = .57$), unpleasant ($F(1,54) = 220.21$, $p < .001$, $MSE = 1.15$, $\eta_p^2 = .80$) and painful ($F(1,54) = 140.26$, $p < .001$, $MSE = 1.09$, $\eta_p^2 = .72$) than experiencing the WPT (Figure 29). These results confirm the strength of the CPT as a stress manipulation.

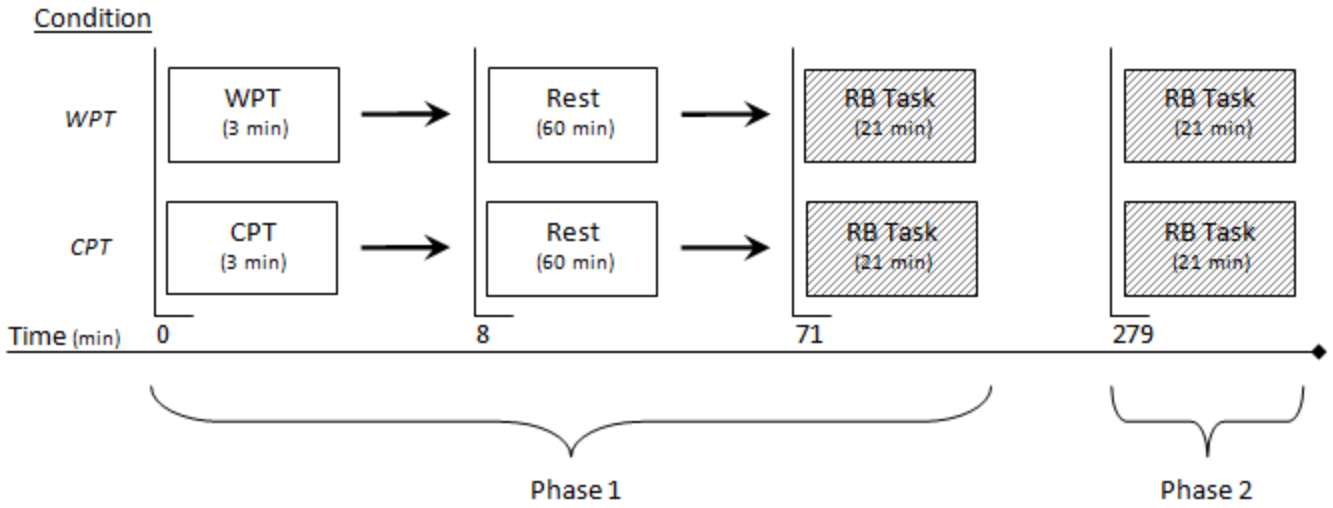

Figure 28: Procedure and timeline of Experiment 3.

Cognitive Task Performance

Rule-Based Task Accuracy.

A four block (within) x two condition (between) repeated measures ANOVA was conducted to compare RB task accuracy between the CPT and WPT conditions for each of 2 time points[9]. The ANOVA for phase 1 revealed a main effect of block ($F(2.48, 134.11) = 53.40$, $p < .001$, $MSE = 41.23$, $\eta_p^2 = .50$) with no block x stress condition ($F(2.48, 134.11) = .71$, $p = .52$, $MSE = 41.23$, $\eta_p^2 = .01$) interaction (Figure 30). These results suggest all participants were able to learn the RB task regardless of stress condition. Between subjects analyses reveal a marginal main effect of stress condition ($F(1,54) = 3.49$, $p = .07$, $MSE = 197.48$, $\eta_p^2 = .06$). The marginal

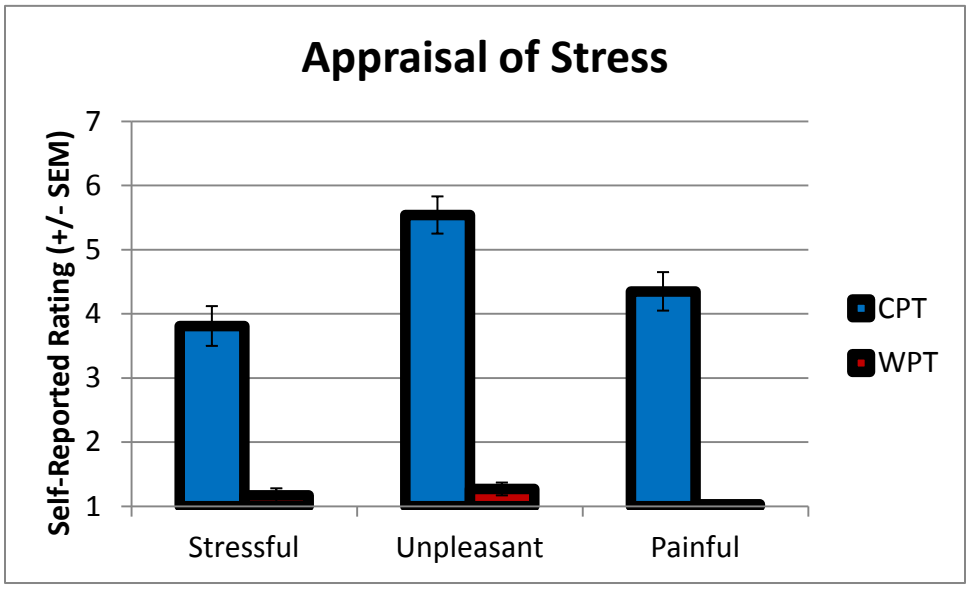

Figure 29: Stress appraisal data for Experiment 3.

[9] A two stimulus (within) x two condition (between) repeated measures ANOVA conducted to compare RB task accuracy collapsing across phase, reveals a significant effect of stimulus type ($F(1,48) = 5.42$, $p = .02$, $\eta_p^2 = .1$), however because a four block (within) x two condition (between) repeated measures ANOVA compared separately for each phase revealed no main effect of either gratings (phase1: $F(1,25) = 3.2$, $p = .09$, $\eta_p^2 = .11$; phase 2: $F(1,22) = .1$, $p = .75$, $\eta_p^2 = .01$) or boxes (phase1: $F(1,27) = .84$, $p = .37$, $\eta_p^2 = .03$; phase 2: $F(1,24) = .96$, $p = .34$, $\eta_p^2 = .04$), all of the Experiment 3 analyses will collapse across stimulus type.

main effect of stress condition is particularly interesting and in line with hypotheses revealing an increase in performance following the CPT compared to performance following the WPT.

The ANOVA for task accuracy during phase 2 revealed a main effect of block ($F(2.47, 118.45) = 62.6$, $p < .001$, $MSE = 29.97$, $\eta_p^2 = .57$) with no block x stress condition ($F(2.47, 118.45) = .41$, $p = .71$, $MSE = 29.97$, $\eta_p^2 = .008$ (Figure 31). Just as for phase 1, results suggest all participants were able to learn the RB task regardless of stress condition during phase 2. Analysis of between-subjects comparison reveals no main effect of stress condition ($F(1,48) = .18$, $p = .68$, $MSE = 110.11$, $\eta_p^2 = .004$). These results indicate the stress manipulation did not impact task performance during phase 2.

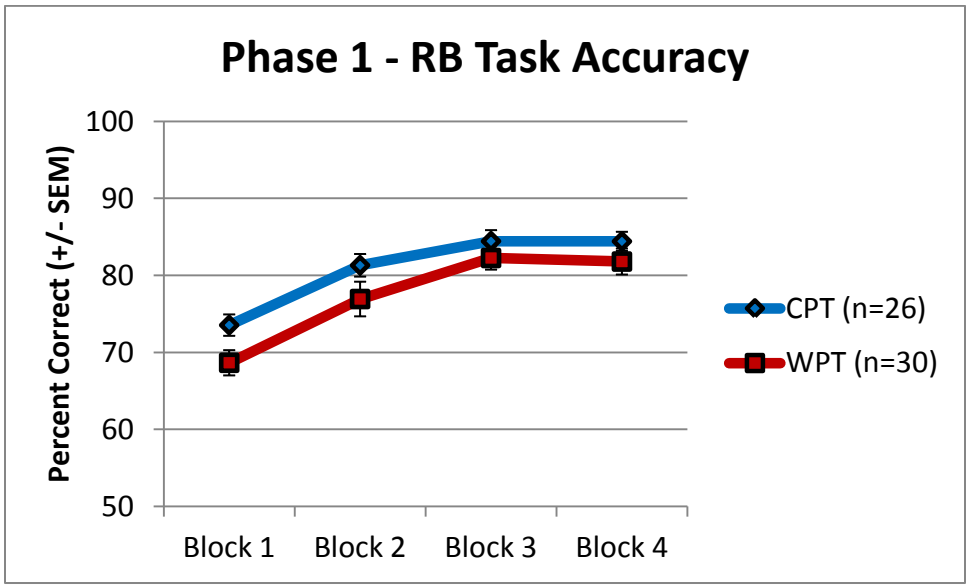

Figure 30: RB task accuracy for phase 1 of Experiment 3.

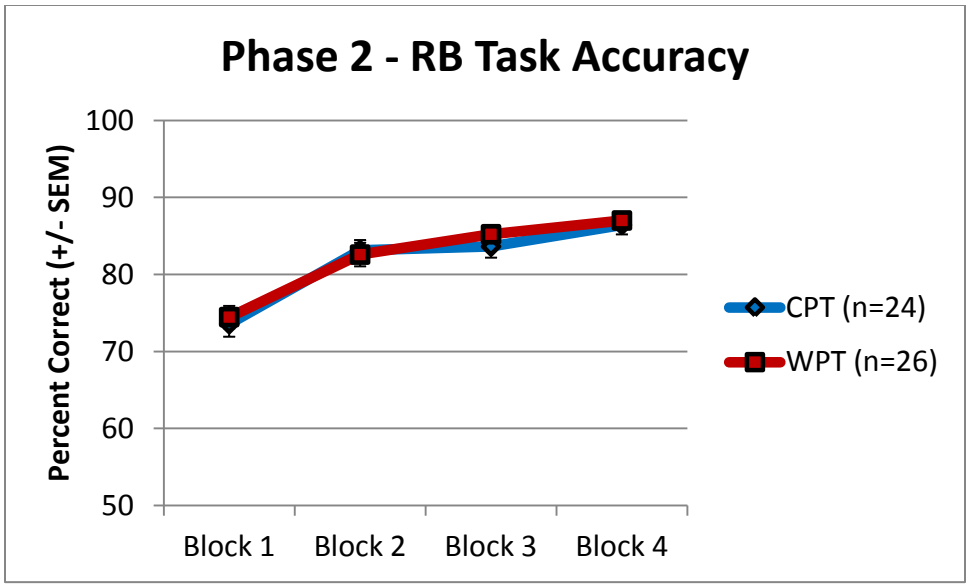

Figure 31: RB task accuracy for phase 2 of Experiment 3.

Rule-Based Task Strategy. To be consistent with Experiments 1 and 2, strategy analyses will focus on block 4 of the task as to provide ample time for strategy development [10]. Participants in the in both the CPT and WPT conditions were more likely to use conjunctive strategies than unidimensional strategies at both phases of the experiment (see table 11). Chi-squared analyses revealed no difference between the conditions in their reliance on one strategy over another at both phase 1 ($\chi^2(1,55) = .01$, $p = .93$) and phase 2 ($\chi^2(1,50) = .25$, $p = .62$).

Relationship Between Stress and RB Task Performance

To fully understand the impact of stress on RB category learning it is important to determine the relationship between the appraisal of stress and RB task performance. In order to assess RB task accuracy at a time where performance had stabilized, all of the following

[10]Block 4 was chosen because this is the time when participants have best learned the task. The random responder model and the general linear classifier provided the best fit for 1 and 0 participants respectively during time 1 and 0 participants during time 2 . Thus, these models were omitted from analyses.

regression analyses concentrate on block 4 task only[11]. The following hierarchical regression model (Step1 predictors: stress condition, stress response; Step2 predictors: stress response,

Table 11: Participant Strategy Usage Counts

Strategy Use Counts				
	Phase 1		*Phase 2*	
	CPT	*WPT*	*CPT*	*WPT*
Unidimensional	6	7	5	4
Conjunctive	20	22	19	22

stress condition x stress response interaction; Outcome variable: block 4 accuracy) were designed to determine the extent to which stress responses moderated the relationship between condition and RB task accuracy. Each model was dummy coded such that the CPT condition can be compared to the WPT condition and was evaluated separately for each stress response variable (i.e., Stress Appraisal, Unpleasantness Appraisal, Painfulness Appraisal). Thus, the results of the step 1 analysis will assess the main effect of stress condition for the participants represented in each in each model. The results of the step 2 analysis will assess if the relationship between the stress response and RB task accuracy differs by condition for each stress response variable. Because the primary focus of this analysis is to understand the moderating effects of the stress response variables, I will focus the description of the results on the stress response x condition interaction at step 2. To further characterize each interaction, simple slopes were estimated for both the CPT and WPT conditions. The simple slope analysis allows for the investigation of the relationship between appraisals and accuracy separately

[11] An ANOVA comparing block 4 RB task accuracy revealed no main effect during phase 1 ($F(1,54) = 1.49$, $p = .23$, $MSE = 64.32$, $\eta_p^2 = .027$) or phase 2 ($F(1,48) = .14$, $p = .71$, $MSE = 29.39$, $\eta_p^2 = .003$).

within each condition. For simplicity, all statistics (for both steps) are reported in Tables 12-14 and simple slopes are reported in Figures 32-37. The x-axis for each figure is centered at the mean across both conditions.

Phase 1. There was no significant interaction between the stress appraisal, or unpleasantness appraisal and RB task performance during phase 1, indicating that the relationship between both the appraisal of stress and unpleasantness with RB task performance did not differ by condition. Also, the stress appraisal and unpleasantness appraisal were not found to be significant predictors of RB task performance in the CPT condition, suggesting there is no significant relationship between the appraisal of stress (Figure 32) or unpleasantness (Figure 33) and RB task accuracy in the CPT condition during phase 1. There was a marginally significant painfulness appraisal x condition interaction indicating that the relationship between the appraisal of the task as painful and RB task performance marginally differed by condition during phase 1. However, the appraisal of painfulness was not found to be a significant predictor of RB task performance in the CPT condition, suggesting there is a no significant relationship between the appraisal of the task as painful and RB task accuracy in the CPT condition (Figure 34).

Phase 2. There was no significant stress, unpleasantness, or painfulness appraisal x condition interaction during phase indicating the relationships between the appraisal of stress, unpleasantness, or painfulness and RB task performance don't differ by condition. Also, all three appraisals were not found to be significant predictors of RB task performance in the CPT condition, suggesting there is no significant relationship between stress (Figure 35), unpleasantness (Figure 36), or painfulness (Figure 37) appraisals and RB task performance for the CPT condition during phase 2.

Model summary of hierarchical regression analyses from Experiment 3.

Variable	Stress Appraisal - Phase 1										Unpleasantness Appraisal - Phase 1								
	b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2		b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2
Step 1				.73	.49			.03						1.07	.35			.04	
CPT condition	2.69	.82	.42								6.18	1.27	.21						
Stress response	-.03	-.03	.98								-.83	-.82	.42						
Step 2						.28	.6	.03	.01							.04	.85	.04	.0
CPT condition	4.9	.92	.36								5.12	.69	.5						
Stress response	1.23	.48	.63								-1.35	-.47	.64						
Stress response X CPT condition	-1.45	-.53	.6								.59	.19	.85						

Table 12: Model summary of hierarchical regression analyses from Experiment 3. Regression statistics are reported as unstandardized ts and bs.

Model summary of hierarchical regression analyses from Experiment 3.

Variable	Painfulness Appraisal - Phase 1									Stress Appraisal - Phase 2								
	b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2	b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2
Step 1				1.37	.26			.05					1.04	.36			.04	
CPT condition	6.47	1.59	.12							-2.95	-1.29	.2						
Stress response	-1.16	-1.11	.27							.9	1.39	.17						
Step 2						3.36	.07	.11	.06						.69	.41	.06	.01
CPT condition	-9.55	-1	.32							-5.36	-1.45	.15						
Stress response	-15.65	-1.97	.055							-.41	-.24	.81						
Stress response X CPT condition	14.73	1.893	.07							1.53	.83	.41						

Table 13: Model summary of hierarchical regression analyses from Experiment 3. Regression statistics are reported as unstandardized ts and bs.

Model summary of hierarchical regression analyses from Experiment 3.

Variable	Unpleasantness Appraisal - Phase 2										Painfulness Appraisal - Phase 2								
	b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2		b	t	p	F	Sig. F	ΔF	Sig. ΔF	R^2	ΔR^2
Step 1				.16	.85			.01						.25	.78			.01	
CPT condition	-1.9	-.55	.58								-2.05	-.71	.48						
Stress response	.31	.43	.67								.45	.60	.55						
Step 2						.85	.36	.03	.02							.1	.75	.01	.0
CPT condition	-5.5	-1.06	.3								-4.06	-.59	.56						
Stress response	-1.47	-.71	.48								-1.35	-.24	.81						
Stress response X CPT condition	2.02	.92	.36								1.83	.32	.75						

Table 14: Model summary of hierarchical regression analyses from Experiment 3. Regression statistics are reported as unstandardized ts and bs.

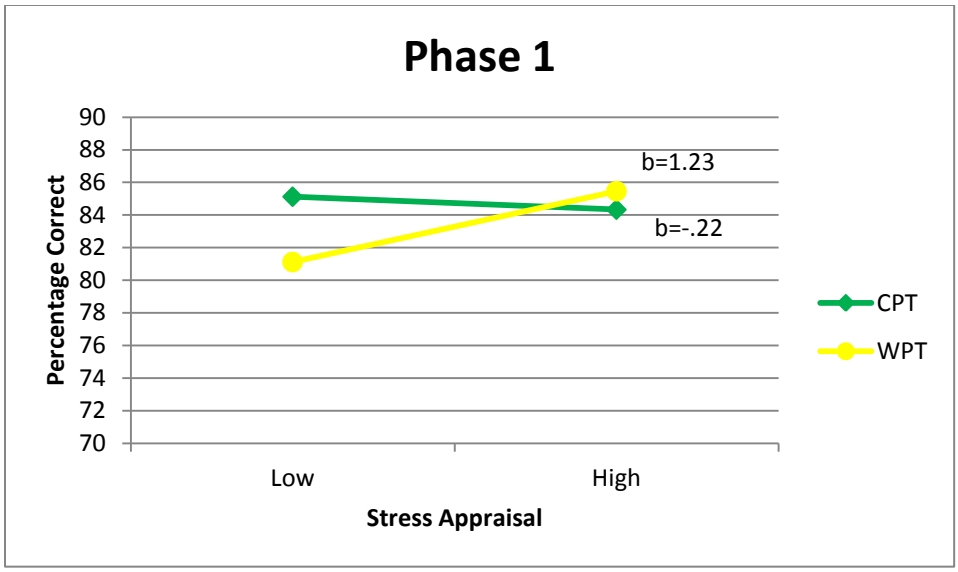

Figure 32: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean.

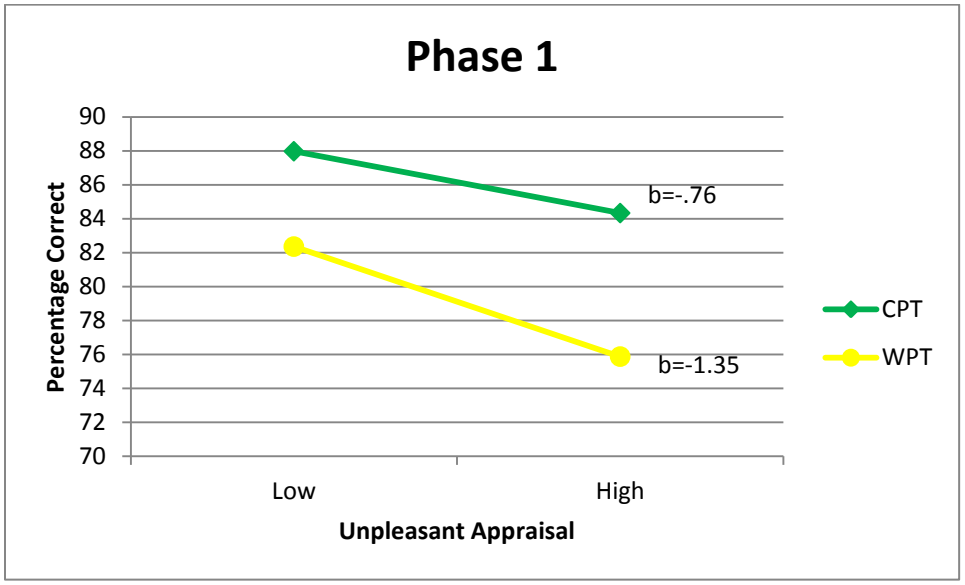

Figure 33: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean.

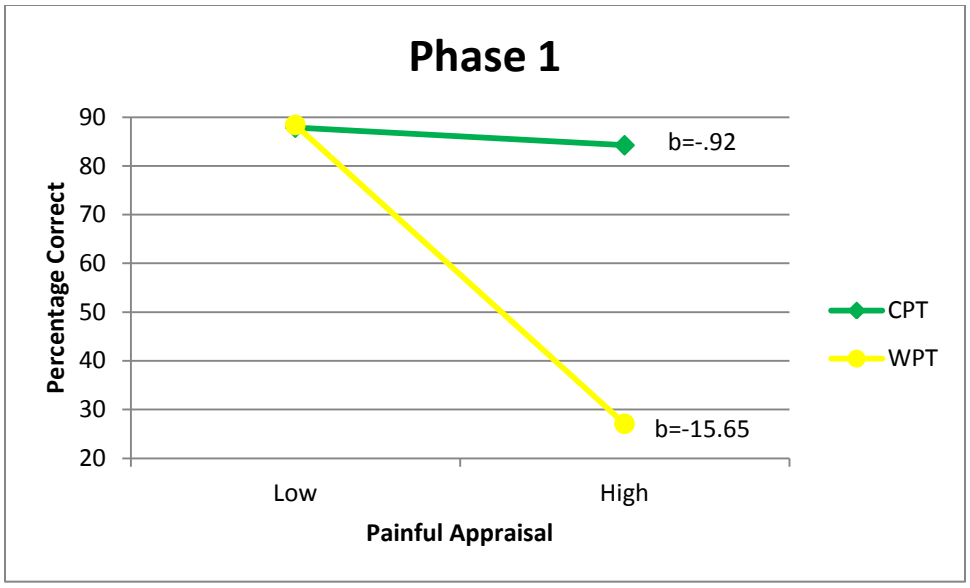

Figure 34: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean.

Supplemental Analyses

In attempts to collect the best data as possible Experiment 3 only recruited participants who had not reported the use of OCs for an early semester subject pool prescreen. As suggested from Kudielka & colleagues (2009), both OC users and women in the follicular phase of their menstrual cycle should be excluded for the most consistent endocrine responses. Given this suggestion and the endocrine mediated hypotheses from Experiment 3, the following analyses will evaluate RB task performance during both phases of the experiment for a subset of participants excluding participants who reported the use of OCs or being in the follicular phase of their menstrual cycle during testing (sample sizes: $CPT_M = 12$, $CPT_F = 6$, $WPT_M = 20$, $WPT_F = 7$)

A four block (within) x two condition (between) repeated measures ANOVA was conducted to compare RB task accuracy between the CPT and WPT conditions for each of 2 time points. The ANOVA for phase 1 revealed a main effect of block ($F(2.28, 98.11) = 41.25$, $p < .001$,

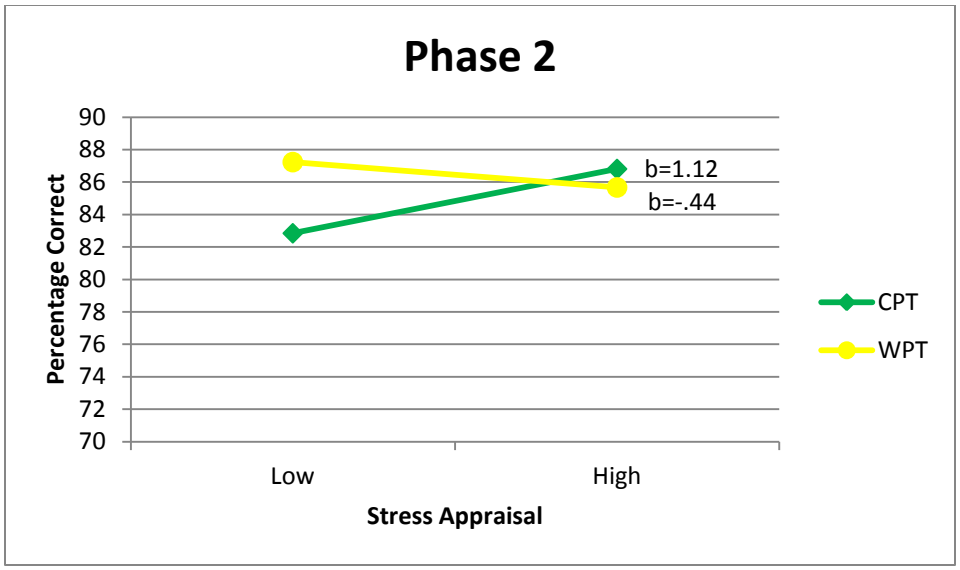

Figure 35: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean.

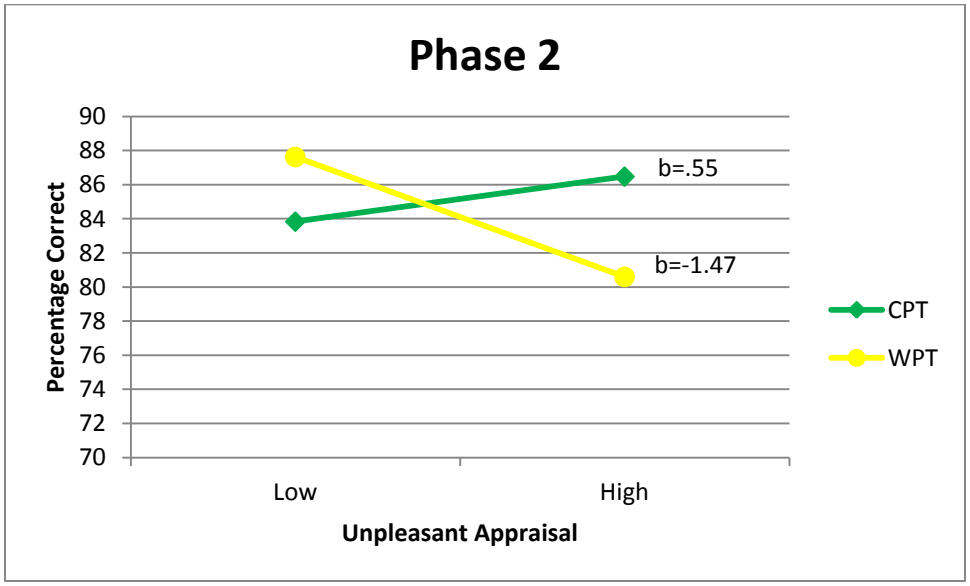

Figure 36: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean.

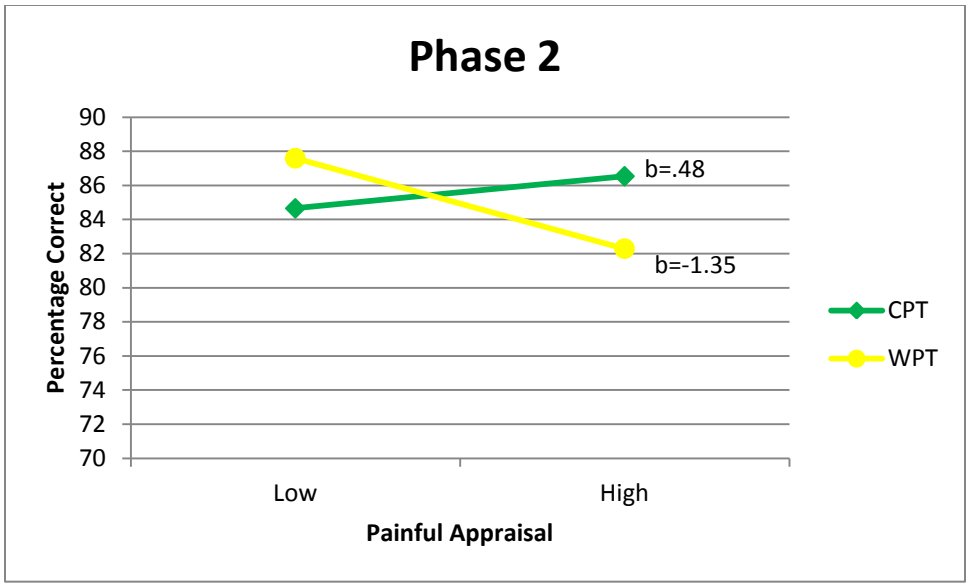

Figure 37: Hierarchical regression model plot. X-axis represents +/- 1 SD from the mean.

MSE = 46.39, η_p^2 = .49) with no block x stress condition (F(2.28, 98.11) = .53, p = .62, MSE = 46.39, η_p^2 = .01) interaction. These results suggest all participants were able to learn the RB task regardless of stress condition. Between subjects analyses reveal the main effect of stress condition (F(1,43) = 2.7, p = .11, MSE = 186.74, η_p^2 = .06) was non-significant.

The ANOVA for task accuracy during phase 2 revealed a main effect of block (F(2.43, 89.88) = 63.39, p < .001, MSE = 25.99, η_p^2 = .63) with no block x stress condition (F(2.43, 89.88) = .27, p = .8, MSE = 25.99, η_p^2 = .007 (Figure 20). Just as for phase 1, results suggest all participants were able to learn the RB task regardless of stress condition during phase 2. Analysis of between-subjects comparison reveals no main effect of stress condition (F(1,37) = 1.62, p = .21, MSE = 108.66, η_p^2 = .04).

Experiment 3 Discussion

Experiment 3 was designed to assess RB category learning during two phases following a stressor; Phase 1 was chosen to compare the delayed enhancements found in Experiments 1 and 2. The Phase 2 was chosen to identify if the delayed RB enhancement would persist for hrs following a stressor. The findings from Experiment 3 reveal a marginal enhancement of RB task performance following the stressor during phase 1, and are in line with hypotheses. Contrary to hypotheses, the RB task enhancement did not extend to a time frame greater than 4 hrs following the stressor, and in fact, no differences were found between conditions during this phase.

The marginal enhancement of RB category learning during phase 1 of Experiment 3 is in line with the delayed effects found in Experiments 1 and 2, and suggests the influence of stress on RB category learning may extend beyond influence from the TSST. Given that these conditions were marginally rather than significantly different from one another, it may be best to remain cautious to interpret findings as a replication. The manipulation itself was different between Experiment 3 and Experiments 1 & 2, potentially attenuating the overall effect. Given the data driven a priori hypotheses for phase 1 of Experiment 3, I feel comfortable speculating the group difference from phase 1 is not incidental.

CHAPTER 5

GENERAL DISCUSSION

The present dissertation work was designed to further explore a relatively neglected sector in the stress and cognition literature, that is the nature of the post-stress time-course. Data from the present experiments proposes a point in time ~71-78 min post-stress onset in which RB category learning is enhanced. The delayed enhancement was present in Experiment 1, and additionally was associated with CORT reactivity following stress. Using a more direct methodology, results from Experiment 2 again revealed delayed enhancement in RB task performance, however this effect did not extend to a more traditional WM task. The differing results between the two tasks questions the cognitive nature of the stress induced enhancement. Results from Experiment 3 reveal a marginally delayed enhancement during a similar window of time following a new stressor, suggesting enhanced RB task performance may not be specific to the TSST. However, importantly, the marginally enhanced RB task performance did not remain when tested ~4 hrs post stress onset. Taken together results from the three studies support a delayed enhancement of RB category learning given a lengthy enough delay from stress, that remains for a limited duration.

Summary

Experiment 1

RB category learning relies on explicit processing (Ashby et al., 1998) and WM (Waldron & Ashby, 2001; Zeithamova & Maddox, 2006), thus a prediction of a task impairment at this time was based on previous research suggesting stress impairs explicit learning tasks (Plessow et al., 2012a; Plessow et al., 2012b; Shields et al., 2015), and WM tasks (Oei et al., 2006; Schoofs et

al., 2006). Contrary to hypotheses, performance in the ND condition tested 3 min after cessation of the TSST was not impaired as compared to the NS condition.

As predicted RB task performance differences were not found between the SD and NS conditions. This finding is in line with a priori hypotheses, and although an expected result, it is unclear how meaningful this is. The SD condition was chosen 29 min after cessation of the stressor in order to best capture a timeframe of elevated HPA activation after a recovery of SAM. The hypothesized null effect was based upon research suggesting a combination of HPA and SAM is required for stress induced WM impairments (Barsegyan et al., 2010). Thus a recovery of SAM was predicted to resolve the negative influence of stress on RB category learning (hypothesized for the ND condition) during the SD time frame. To be clear, if the hypothesized impairment in RB task performance had been present in the ND condition, then no difference in performance between the SD and NS condition would have provided support that SAM is required for an impairment.

As predicted, performance was enhanced in the LD condition compared to the NS condition. The results suggest stress enhances RB category learning 78 min after the onset of the stressor. This finding is in line with a priori hypotheses, and was predicted from previous delayed WM enhancements attributed to genomic mechanisms following stress (Henckens et al., 2011; Yuen et al., 2009). To my knowledge RB category learning has never been assessed during a delay following stress before, and therefore it isn't clear if the effect is related to previous findings from the stress-WM literature.

Experiment 2

Experiment 2 was designed to further assess two questions. First, is the delayed RB enhancement from Experiment 1 a true effect? Given that RB category learning has never been

studied during this window post-stress, a replication of an enhancement would substantiate findings from Experiment 1. Second, do the delayed enhancements extend to a more traditional WM task? RB category learning has been shown to rely upon WM resources (Waldron & Ashby, 2001; Zeithamova & Maddox, 2006) and furthermore hypotheses were largely based upon research suggesting a delayed enhancement in WM task performance (Henckens et al., 2011; Oei et al., 2009; Yuen et al., 2009). Taken together it seems plausible that WM would be similarly enhanced during this delayed window. To study WM, the n-back task was used. Results from Experiment 2 again revealed a delayed RB task performance enhancement. Additionally, the no-stress condition was adjusted so the post-manipulation delay matched between the stress and no-stress conditions. The replication with this adjustment ensures the Experiment 1 findings were not driven by a confound in the post-manipulation delay. In contrast to predictions, n-back task performance was not enhanced in the LD condition as compared to the NS condition, suggesting stress doesn't have the same impact on WM and RB category learning during this window of time following stress.

Experiment 3

Experiment 3 was designed to assess RB category learning during the same delayed time-frame tested in Experiments 1 and 2 (phase 1) as well as a new ~4 hour delayed time-frame (phase 2), following the CPT. A 4 hour delay was chosen from previous literature assessing WM during this delay (Henckens et al., 2011; Yuen et al., 2009). Given the duration of the CPT is far shorter than the TSST, the phase 1 delay was chosen to best compare the nature of the time-course relative to the onset of stress (~71 min). The phase 2 delay was chosen to examine if the influence of stress remains for a further extended length of time. Experiment 3 used a within-subjects design assessing task performance for the same participants at both phases. First, a

marginal enhancement in RB task performance during phase 1 suggests RB task performance may be influenced similarly by both the TSST and CPT. Although not statistically significant, phase 1 findings provide support for a delayed enhancement following a stressor. Phase 2 revealed no differences in task performance between the CPT and WPT conditions, suggesting the delayed enhancement of RB category learning does not remain for an extended length of time.

Stress Influence on WM

Previous studies have revealed RB category learning is dependent upon WM processing (Waldron & Ashby, 2001; Zeithamova & Maddox, 2006), and given this, it is reasonable to suspect the influence of stress on RB category learning may be impacted by the underlying nature of stress and WM. In particular, enhanced WM has been found following a delay in both animals (Yuen et al., 2009) and humans (Henckens et al., 2010; Oei et al., 2009) and has been associated with the HPA response (Shields et al., 2015). The delayed enhancement in RB category learning from the present experiments along with the relationship found between performance and CORT reactivity are in line with much of the stress-WM research, suggesting these constructs may be similarly influenced by stress. Furthermore results from Experiment 1 reveal a higher ratio of conjunctive to unidimensional strategy users in the LD condition. This finding suggests users were likely attending to both dimensions while categorizing and may indicate a stronger reliance on WM.

The RB category learning findings may be in line with the WM literature, however n-back performance was not enhanced following a 78 minute delay from stressor onset. It should further be noted that not only were participants not enhanced, but when OC users and women in the follicular phase of their menstrual cycle were excluded, a marginal impairment in n-back

task performance was present during this delayed time point. Given that the sample sizes are low (N_{stress} = 18, $N_{no\text{-}stress}$ = 13), this marginal effect should be taken with skepticism, however it provides pattern conflicting with predictions. The present 71-78 min time frame has been suggested as a point following stress in which genomic effects are beginning to take place (Joels et al., 2007; Shields et al., 2015), however this particular time frame itself has never been tested. If this is the case it is plausible that genomic cognitive effects had begun to take place.

Previously, WM enhancements have been found using hydrocortisone administration (Henckens et al., 2011; Oei et al., 2009; Shields et al., 2015) or GC receptor stimulation (Yuen et al., 2009). To my knowledge the present data is the first example of a study assessing WM performance following a delay from a stressor itself, and thus it is possible the delayed effects of stress and the artificially induced stress responses are not unitary. This may explain the null n-back findings during this time-frame following the TSST.

It also remains possible the n-back task isn't a perfect indicator of WM performance following stress. It is worth noting that n-back task performance has been shown to be enhanced following a delay from stress, however this finding was only present when analyzing a combination of task accuracy and response time as a behavioral measure (Henckens et al., 2011). Given the present Experiment 2 findings were in the expected direction of better task accuracy following stress compared to no-stress, it is possible n-back task is simply not sensitive enough to reveal a statistical accuracy effect. The use of a more cognitively demanding WM task would be necessary in order to assess if this is the case.

Stress Influence on RB Category Learning Compared with WM

Previous literature suggests a high pressure experience is sufficient to impair RB category learning (Beilock & Decaro, 2007; Markman et al., 2006). Additionally, a study from our

lab support replicates this finding, and additionally suggests this pressure manipulation is cardiovascularly arousing (McCoy et al., 2013). Given the previously found relationship between pressure and RB category learning, the null finding from the ND condition may suggest there is something psychologically different between the influence of pressure and stress. To my knowledge there have not been any other studies assessing RB task performance at the group level following a stress response and thus the RB category learning literature is too small to base predictions on alone. RB category learning is highly dependent upon WM processing (Waldron & Ashby, 2001; Zeithamova & Maddox, 2006), and given this reliance, the present predictions and background discussion have explored the effects of stress on RB category learning and WM. Previously WM has shown to be impaired initially following stress (Elzinga & Roelofs, 2005; Oei et al, 2006; Schoofs et al., 2006), and given these previously found impairments it was predicted that RB task performance would be impaired initially following the TSST, however this was not the case. No differences were found between RB task performance in the ND and NS conditions in Experiment 1. This finding suggests the comparison between the influence of stress on RB category learning and WM under stress may not be so straight forward.

It is unclear exactly why the predicted impairment of RB category learning following stress was not present. It is possible the influence of stress on the underlying processes of RB category learning is different that the influence on WM. It should be clarified that because RB category learning requires WM resources does not mean the two constructs are synonymous. RB category learning in the present design has been used as a probe for an explicit learning system made up of numerous sub-processes. In addition to WM, explicit learning is made up of multiple executive functions, which have shown to be affected very differently by stress (Shields et al., 2015). For example, stress has been shown to negatively influenced WM (Schoofs et al., 2006; Elzinga & Roelofs, 2005), and cognitive control (Plessow et al., 2012a; Plessow et al.,

2012b) immediately following stress, yet enhancing effects on inhibition have been found following a stress response (Shields et al., 2015). The influence of stress on inhibition in particular may suggest RB tasks requiring a greater selective attention would counteract the negative effects of stress. Given explicit learning and hence RB category learning is dependent upon all three of these processes, it is possible that the influence of stress on RB category learning isn't different than WM, but instead more complex. In other words, it is possible the influence of stress on each sub-process of RB category learning is impacted differently, and the complexity of the entire stress-RB category learning relationship complicates the story.

The delayed enhancement in RB category learning was of much interest, and again this finding was hypothesized to be driven by the impact of stress on WM. Given the potential differences in the cognitive processes underlying the RB task and the n-back task, perhaps it is not surprising that results differ between the two constructs at this time frame as well. Again if this is the case, it isn't clear exactly what underlying process is actually being affected by stress and further cognitive research may be necessary to uncover this difference. It was hypothesized that the greater number of conjunctive strategy users than unidimensional strategy users in the LD condition from Experiment 1 as compared to the NS condition suggested an influence of WM. Again this is possible, however it is also possible that the influence of stress upon separate underlying explicit mechanisms influenced this strategy difference as well.

The delayed enhancement of RB category learning at the group level explains a lot, and the additional relationship found between CORT reactivity suggests this delayed enhancement was indeed impacted by the early stress response. This finding suggests that CORT may have a similar genomic influence on RB category learning during the present ~78 minute time frame as it does for WM at a ~4 hour time frame (Yuen et al., 2009). The present results are not sufficient

to make a statement about the mechanism involved, however the relationship between CORT reactivity and RB task performance provides a basis for further research to explore this mechanism.

Nature of Timing

A major theme of the present thesis is the temporal nature of the stress response, and the large role that it plays in the stress-cognition relationship. Taking all 3 experiments into account, the present dissertation targeted 4 time points following stress (3 min, 29 min, 55 min [~71-78 min from onset], and ~4 hrs from onset). The first two time points were chosen to capture the rapid effects of stress, assessing the lasting influence of the endocrine response, whereas the further delayed time points were designed to capture a genomic timeframe following stress. The genomic time frame is best considered in terms of the onset of stress, rather than the cessation of the stressor because these time points are meant to explore the relationship with the effects of the initial stress response, and not the extent to which it changes over time.

It has been suggested that WM is impaired immediately following stress, and the rapid impairments are associated with a combination of HPA and SAM activation (Barsegyan et al., 2010). It has also been shown that a pressure induced stress response impaired RB category learning (McCoy et al., 2013). Taking both of these papers into account, a prediction was made that 3 min following stress RB category learning performance would be impaired, and because of the short duration of SAM, an impairment would not be present 29 min after the stressor. Results revealed no differences in RB performance at either time point following stress. Importantly the results are not completely conflicting with the literature however. The previously described impairment in RB category learning was driven by an online pressure

induction, and the present RB assessment came after a stressor. It is possible the methodological difference, although seemingly small, led to the behavioral discrepancy. One possible mechanism for this difference is influence of SAM activity. SAM was not measured in McCoy et al (2013), however a blunted sAA response was found in the ND condition of Experiment 1. It is unclear what drove this weak response however given that previous research has argued for SAM as a requirement for a cognitive impact of stress (Basegyan et al., 2010; Roozendaal & McGaugh, 2011), it is possible it played a role in these null findings. In particular the animal literature has revealed a combination of CORT and NE are necessary for stress induced impairments of WM (Barsegyan et al., 2010), and without a sufficient SAM response, a release of the necessary NE may also not have been present.

Experiments 2 and 3 were designed to assess whether or not the delayed enhancement in RB category learning was comparable to previously found delayed WM enhancements. This was done using 2 approaches. First, n-back performance was assessed 78 min after the onset of the stressor as a way to compare WM task performance to the presently found RB task enhancements. Second, Experiment 3 was designed to understand if a RB task enhancement was present during the same ~4 hour delay as found in the stress-WM literature (Henckens et al., 2010; Yuen et al., 2009). The approaches taken in Experiments 2 and 3 were designed together to assess whether or not the enhanced RB category learning from Experiment 1 was related to WM. When taking the results of all 3 experiments together, the null findings from the ND condition in Experiment 1 did not compare to the previous WM literature, the stress condition for the n-back task in Experiment 2 did not compare to delayed RB findings from Experiments 1 and 2, and CPT condition during phase 2 of Experiment 3 did not compared to research suggesting WM enhancements ~4 hours post-stress. In whole these results provide a

strong indication that the relationship between stress and WM and the relationship between stress and RB category learning do not follow the same time-course.

Nature of the Stressor

The present dissertation is meant to explore the influence of acute stressors, and not a particular stressor on explicit learning. It was predicted that the relationship between stress and explicit learning wouldn't differ across stressor given a sufficient stress response. The predictions were based upon the endocrine response to stress, and although saliva samples have yet to be analyzed for Experiment 3, the CPT was chosen because of prior research suggesting the strength of an endocrine response (Schwabe et al., 2008). The endocrine response to the CPT has been show to be comparable to the endocrine response to the TSST (Dickerson & Kemeny, 2004; Kirschbaum & Hellhammer, 1994). Results from phase 1 of Experiment 3 reveal a marginal enhancement, and hence a marginal replication from Experiments 1 and 2. Given this effect was not significant, a statement cannot be made of a direct comparison between the influence of the TSST and CPT on RB task performance during a window of ~71-78 min following stress, however I argue given a priori hypotheses and a trend of a RB task enhancement at this time point, that the question of whether or not the effect is stressor specific remains open.

When comparing results across stressors there are a few things to consider. First, many of the predictions for the present experiments were built from research on pressure manipulations (Markman & Maddox, 2006; McCoy et al., 2013), and artificial stress inductions (Barsegyan, et al., 2010; Henckens et al., 2011; Shields et al., 2015) as well as a variety of stressors and it is possible results would differ as a function of the type of stress induction. Second, even though the stressors were chosen to provide the greatest likelihood of a sufficient

HPA response, it cannot be guaranteed that all participants will respond the same. It has been argued that the stressor itself is not as important as the subjective appraisal of stress (Lazarus & Folkman, 1984). Furthermore responses to stress may differ greatly in between individuals (Blascovich, 2008; Seery et al., 2011). It is possible that the range of individual differences from one stressor are quite different than another. Even when provided a consistent stress response at the group level, laboratory stressors often cannot assume the behavioral results are identical across individuals. For example a study by Elzinga & Roelofs (2005) only found behavioral effects when splitting participants in terms of a CORT response following the TSST. Given the present research questions were designed to assess the relationship between a sufficient endocrine response and task performance, individual differences in the type of responses were not analyzed.

Limitations

As stated throughout the delayed RB category learning enhancements found ~71-78 min post stressor-onset are of great interest and contribution to the literature, however the difference in effect size must be noted. Results from Experiment 1 revealed a substantial effect size ($\eta_p^2 = .34$), suggesting the delayed RB task enhancement. Experiment 2 was sufficient to statistically replicate the finding however the effect was much lower ($\eta_p^2 = .11$). Additionally a marginally significant effect in Experiment 3 was even lower ($\eta_p^2 = .06$). These results reveal the consistency of the delayed RB enhancement during this delayed window of time, however there may be factors influencing these effect size differences.

Overall results in the present study tell a story about the relationship between stress and cognitive performance, however the inconsistency in endocrine response must be addressed. Participants in the ND and SD conditions from Experiment 1 did not have

consistently elevated levels of both CORT and sAA following the TSST. It is possible null cognitive results are related to the absence of a full stress response in these groups. Endocrine responses have yet to be analyzed for Experiments 2 and 3, therefore it remains possible a sufficient endocrine response was not present in all conditions. Analysis of saliva for these studies would provide more information and potentially reveal new information helping to uncover the story of the present findings.

It is possible the present findings were influenced by hormonal variables not controlled for in the study. As suggested by Kudielka & colleagues (2009), supplementary analyses were performed excluding any participants reporting the use of OCs, and women reporting to be in the follicular phase of their menstrual cycle during testing for each experiment. In order to ensure consistent data collection, some of these participants were included in the study, therefore sample sizes for the subsets including these exclusions were quite small. Improvements were made for recruitment in Experiments 2 and 3 compared to Experiment 1. Particularly, the recruitment of only participants not reporting the use of OCs during a pre-semester prescreen helped improve the consistency of the sample, however it wasn't possible to remove OC users and women in their follicular phase altogether. It is possible with a full exclusion of these participants, results would have been more reliable.

In Experiment 3 specifically, due to the University recruitment policy and the within-subjects design, participants were free to leave the lab for ~3 hrs in between phases of the study. Once participants completed phase 1 they were able to carry on with their day until returning for phase 2. The participants were not given any instructions other than when to return to the lab, so it is possible that external factors that were out of our hands influenced the study and thus affected RB task performance during phase 2.

General Experimental Conclusions

Taken together the findings in the present thesis contribute to a relatively broad field of stress & cognition. The most compelling and consistent finding was a RB task performance enhancement present during a period of time 71-78 min after the onset of stress. This effect was originally present in Experiment 1, replicated in Experiment 2, and marginally replicated in Experiment 3 using a new stressor. Taken together the results provide support for a genuine enhanced RB effect during a delayed time frame following stress. Furthermore, a relationship was found between the CORT response and RB task performance in Experiment 1 justifying the argument that the delayed enhancement is related to HPA activation. The results from this thesis also suggest a disconnect between the influence of stress on RB category learning and WM. This disconnect was blatantly present in Experiment 2, revealing no influence of stress on WM task performance, and less direct in Experiments 1 and 3, revealing null effects during time points previously shown to impact WM following stress (Elzinga & Roelofs, 2005; Henckens et al., 2011; Schoofs et al., 2008; Yuen et al., 2009). The results from Experiments 2 and 3 are surprising given the reliance of WM in RB category learning (Waldron & Ashby, 2001; Zeithamova & Maddox, 2006), and given the congruous relationship between PFC functioning and both WM (Baddeley 2012; Baier et al., 2010) and RB learning (Ashby et al., 1998; Ashby & Ell, 2001). I've discussed possible factors influencing this disconnect that may be present in the three experiments, however when taken as a whole the present results suggest the relationship between stress and RB category learning are not the same as the relationship between stress and WM. It is possible the genomic timeline between the two constructs are not in line.

Given the continued replication of a post-stress RB enhancement, a question arises as to the importance of the particular ~71-78 min delay? This time frame has been suggested as a

possible starting point for genomic cognitive effects (Hermans et al., 2013; Joels et al., 2011; Joiner et al., 2010), however to my knowledge explicit learning has not been assessed during this window of time following stress in either humans or animals. The present findings therefore are the first to introduce data supporting that stress impacts explicit learning, and more specifically RB category learning, during this time frame. Additionally, a relationship between RB task performance and CORT reactivity suggests the nature of this effect is related to the stressor itself. The present dissertation research bolsters the literature of stress with both explicit learning and RB category learning, and suggests a time frame to be considered for future research in the field of stress and cognition.

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APPENDIX

This appendix briefly describes the decision bound models. For more details, see Ashby (1992a) or Maddox and Ashby (1993). Four independent classifications of decision bound models are fit based upon current theories of how category learning strategies develop (see, e.g., Ashby et al., 1998). These models have received considerable empirical support (see Ashby & Maddox, 2005, and Maddox & Ashby, 2004, for reviews).

The General Linear Classifier (GLC)

This model assumes that the decision bound between each pair of categories is linear and requires the integration of perceived spatial frequency and orientation for sine wave grating stimuli, and perceived size and pixel density for density box stimuli . The GLC has three parameters: (slope and intercept of the linear bound and variance of internal (perceptual and criterial) noise (σ2)).

The Unidimensional Model

This model assumes that the participant sets a criterion on a single perceptual dimension and then makes an explicit decision about the level of the stimulus on that dimension (Ashby & Gott, 1988; Shaw, 1982). Two versions of the one-dimensional model were fit to these data: One assumed that participants attended selectively to spatial frequency for sine wave gratings, or size for density boxes, and the other assumed that participants attended selectively to orientation for sine wave gratings, or pixel density for density boxes. The one-dimensional models have two free parameters: a decision criterion on the relevant perceptual dimension and the variance of internal noise (σ^2).

The General Conjunctive Classifier (GCC)

Another type of explicit strategy available to participants is a conjunction strategy. As is the case with perceptual-integration strategies, conjunction strategies also require the

integration of spatial frequency and orientation information for sine wave gratings, and the integration of size and pixel density for density boxes. For example, a participant might set a criterion along the spatial frequency dimension to determine if the stimulus is high or low in spatial frequency and set a separate criterion on orientation, to determine if the angle is shallow or steep. The results of these independent decision processes might then be combined to make a response—for example, "Respond A if the stimulus is low and shallow." Although conjunction strategies require integration, they differ from GLC strategies in that the integration is post decisional. In other words, decisions are made about the stimulus value on each dimension, and the output of these decisions is explicitly integrated to generate a category response. Indeed, recent evidence supports this distinction between conjunction and GLC strategies (Maddox, Bohil, & Ing, 2004).

Conjunction models have three parameters (a criterion on each dimension, and σ^2). Based on inspection of the data from the individual participants, one version of the GCC model was fit to these data. The model assumed that individuals assigned a stimulus to Category B if it was high in spatial frequency and low in orientation (i.e., the bars are thin and shallow) for sine wave gratings, or large in size and low in pixel density for density boxes; otherwise the stimulus was assigned to Category A. The GCC has three free parameters: a decision criterion on each of the relevant perceptual dimensions and the variance of internal noise (σ^2).

One specific response fit, the optimal GCC model, assumes that participants were more likely using optimal conjunctive strategies than any suboptimal decision criteria. In other words, participants more likely figured out the optimal rule than relied on any type of suboptimal decision strategy. Rather than allowing the decision bounds to vary, this model sets them on each stimulus dimension to create the best possible decision criteria (see Figure 1). The optimal version of GCC has only one free parameter (σ^2)

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