ABSTRACT

ENNIS, GILDA E. Physiological Stress Responses Associated with Cognitive Challenge: Individual Differences and Relationship to Memory. (Under the direction of Shevaun D. Neupert, Ph.D.)

Previous research has suggested that middle-aged and older educated adults have increased cortisol reactivity to cognitive challenge compared to younger educated participants and less educated adults (Neupert, Miller, & Lachman, 2006). Additional research has indicated that cortisol reactivity to laboratory stressors (i.e. the Trier Social Stress Test) may be dependent upon the personality traits of extraversion, openness and neuroticism, and that such reactivity in some cases may be moderated by gender (Oswald et al., 2006). Analyzing cortisol elevations in response to a battery of cognitive tests is important, because cortisol increases may have an influence upon assessments of declarative and working memory at the end of the testing battery. Experimental studies (e.g. Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Lupien et al., 1997; Lupien, Gillin, & Hauger, 1999) have suggested that cortisol elevations - not related to the to-be-remembered material - may generally have impairing effects upon declarative and working memory, cognitive processes dependent upon the glucocorticoid receptor rich regions of the hippocampus and prefrontal cortex (Eichenbaum, 2001; Lupien, Maheu, Tu, Fiocco, & Schramek, 2007). The effects of cortisol upon memory, however, are complex and may be dependent upon whether elevations occur in the morning or afternoon. In regards to working memory, cortisol induced impairments may also be

dependent upon activation of the sympathetic nervous system (Elzinga & Roelofs, 2005; Roozendaal, McReynolds, & McGaugh, 2004).

Utilizing data from the Boston oversample of the second phase of the Midlife Development in the United States, a national survey of health and well-being funded by the National Institute on Aging, cortisol reactivity associated with an in-home cognitive challenge was examined to determine whether reactivity varied according to age and education, and the personality traits of extraversion, openness, and neuroticism. Whether the effects of personality were moderated by age, education or gender was also investigated. Analyses were also conducted to determine whether cortisol increases associated with completed cognitive tests hindered subsequent performance on declarative and working memory assessed at the end of the testing battery. Whether these effects were dependent upon age and time of day of testing were also explored. In the case of working memory, sympathetic arousal, as measured by increased heart rate and sweat production, was also considered as an additional moderator.

Findings suggested that highly educated older and middle-aged adults did not express an increased cortisol response over time. Of the personality variables tested, extraversion was associated with an increased cortisol response. This was further qualified by an Age X Extraversion interaction indicating that younger and middle-aged extraverted adults expressed the least change in cortisol throughout the cognitive testing period compared to older adults and middle-aged and younger adults scoring low in extraversion. Contrary to expectations, cortisol elevations alone were not significantly associated with performance on declarative or working memory testing. Working memory, however, was associated with an Age X Cortisol Change X Time of Day interaction. Results suggested that older adult cortisol responders

performed better on letter-number sequencing in the afternoon than 1) same-aged participants who did not experience a cortisol increase in the afternoon and 2) same aged-participants who did experience a cortisol increase in the morning. Older adults who experienced a cortisol response in the afternoon tended to be highly educated.

Further analysis also suggested that working memory performance depended upon an interaction between cortisol response and sympathetic arousal, as measured by skin conductance level and standard deviation. Those participants with increased cortisol and sympathetic arousal appeared to perform as well on letter-number sequencing as those without a cortisol or sympathetic response.

Physiological Stress Responses Associated with Cognitive Challenge: Individual Differences and Relationship to Memory

by
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BIOGRAPHY

Gilda Edwards Ennis received a B.S. in Medical Technology from the University of North Carolina at Chapel Hill. After working as a staff technologist and supervisor, Gilda taught for fourteen years within the department of Medical Laboratory Technology at Wake Technical Community College in Raleigh, North Carolina. Seeking to understand more about the social sciences, Gilda enrolled in the M.A. program in liberal studies at North Carolina State University and finished her degree in 2005. Following this, she enrolled in a combined M.S. and Ph.D. program in psychology at North Carolina State University and is currently studying within the lifespan developmental area under the supervision of Shevaun D. Neupert, Ph.D. Gilda's master's research focused upon physiological reactivity associated with a cognitive challenge. She explored individual differences in cortisol reactivity and whether physiological responses to cognitive testing were related to declarative and working memory performance. Gilda earned her Master's of Science in Psychology in May 2009.

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Introduction

For some individuals, the task of taking cognitive tests can cause a physiological stress response, where the hypothalamic-pituitary-adrenocortical (HPA) axis is activated, resulting in the increased release of cortisol from the adrenal cortex. This physiological reaction to cognitive testing seems more likely to occur in older adults than younger adults (Steptoe, Kunz-Ebrecht, Wright, & Feldman, 2005), and may be moderated by education, where older adults with advanced education have a more significantly elevated cortisol response during and after testing than older adults with less education (Neupert, Miller, & Lachman, 2006).

Activations of the HPA axis tend to occur when individuals interpret situations as threatening (McEwen, 2000). The process of completing cognitive tests may be threatening to older adults if they perceive that such testing may reveal declining intellectual capacity. This threat could be exaggerated in older adults with advanced education, since cognitive capabilities may be especially valued and loss of such abilities may be seen as a loss of social esteem and social status (Dickerson & Kemeny, 2004).

In addition to age and education, personality differences may also contribute to the intensity of a cortisol response to cognitive testing. Although researchers have not examined whether personality differences contribute to varying cortisol levels in response to completing a battery of cognitive tests, some have investigated whether exposure to a laboratory based psychosocial stressor (e.g. public speaking) would elicit varying cortisol responses depending upon personality trait. In one study, participants scoring high in

openness and extraversion demonstrated greater cortisol elevations than those ranking low in these two personality characteristics (Oswald, Mathena, & Wand, 2004). In another analysis, extraversion in men (but not women) was significantly related to cortisol increase following exposure to a psychosocial stressor (Oswald et al., 2006). In this same study, women scoring high in the trait of neuroticism experienced only a slight increase in cortisol to the laboratory based psychosocial stressor, while the relationship between cortisol response and neuroticism in men was not significant (Oswald et al., 2006). Thus, personality and gender may interact to influence the cortisol response to psychosocial stress.

Analyzing cortisol elevations in response to a battery of cognitive tests is important, because cortisol increases may have an influence upon assessments of declarative and working memory at the end of the testing battery. Experimental studies (e.g. Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Lupien et al., 1997; Lupien, Gillin, & Hauger, 1999) have suggested that cortisol elevations - not related to the to-be-remembered material - may generally have impairing effects upon declarative and working memory, cognitive processes dependent upon the glucocorticoid receptor rich regions of the hippocampus and prefrontal cortex (Eichenbaum, 2001; Lupien, Maheu, Tu, Fiocco, & Schramek, 2007). The effects of cortisol upon memory, however, are complex and may be dependent upon whether elevations occur in the morning or afternoon.

Cortisol levels follow a diurnal pattern. For individuals who awake in the morning and return to sleep during the night, cortisol levels peak in the morning and slowly decline in the afternoon until reaching a trough in the evening and during sleep (Hennig, Kieferdorf, Moritz, Huwe, & Netter, 1998; Lupien et al. 2007). Due to the greater elevation of cortisol in the morning compared to the afternoon and evening, at least for those who follow regular waking and sleeping patterns and have a normal diurnal cortisol rhythm, stress-induced elevations in cortisol may be more likely to impair memory function during the AM period (Het, Ramlow, & Wolf, 2005; Lupien, et al., 2002). Studies investigating time of day of effects (e.g. Het et al., 2005, Lupien et al., 2002) have primarily focused upon declarative memory. Whether these same effects influence working memory performance deserves further investigation (Het et al., 2005).

In regards to working memory, cortisol induced impairments may also be dependent upon activation of the sympathetic nervous system (Elzinga & Roelofs, 2005; Roozendaal, McReynolds, & McGaugh, 2004), which can also occur in response to threatening situations. Sympathetic arousal results in the classic fight or flight response, due to the release of noradrenaline and adrenaline, resulting in elevated heart rate, increased sweat production, and higher blood pressure (Gunnar & Quevedo, 2007).

Many studies examining the effects of acute cortisol elevations upon declarative and working memory performance (e.g. Elzinga & Roelofs, 2005; Kirschbaum et al. 1996) and the combined effects of cortisol and sympathetic arousal upon working memory (Elzinga & Roelofs, 2005) have focused on young adults. Few studies have

addressed whether the acute physiological response to stress has similar effects upon cognitive performance in middle-aged and older adults.

This study examined whether an increased cortisol response to a battery of cognitive tests differed according to age, education, and the personality traits of openness, extraversion, and neuroticism. Whether elevated cortisol was associated with interactions of age and education, personality and gender, personality and age, and personality and education was also explored. Since experimental studies (e.g. Kirschbaum et al. 1996; Lupien et al. 1997; Lupien et al., 1999) have indicated that cortisol elevations not related to the to-be-remembered material may generally have impairing effects upon declarative and working memory, analyses were conducted to determine whether cortisol increases associated with completed cognitive tests hindered subsequent performance on declarative and working memory assessed at the end of a testing battery. Whether these effects were dependent upon age and time of day of testing were also explored. In the case of working memory, sympathetic arousal, as measured by increased heart rate and sweat production, was also considered as an additional moderator.

Review of Literature

Definition of stress

McEwen (2000) defined stress as "an event or events that are interpreted as threatening to an individual and which elicit physiological and behavioral responses" (p. 173). Through a process of cognitive appraisal, individuals may first determine the degree of risk or potential threat that a situation presents (Lazarus & Folkman, 1984). This initial interpretation of an event, referred to as "primary appraisal" by Lazarus and

Folkman (1984), may be followed by a "secondary appraisal" that determines whether resources, skills, or abilities are adequate to cope with the potentially threatening situation. When resources for coping are seen as sufficient to offset the pressing demands of a particular event or situation, then that event or situation may be appraised as challenging; however, when resources are perceived as inadequate, the event or situation tends to be seen as a threat.

Completing a battery of cognitive tests may be perceived as threatening to some older adults, if they believe that they do not have adequate skills or abilities to perform well on such tests. This perception of threat may be accentuated if the participants believe that poor performance will be judged negatively by others, resulting in a loss of social esteem or status. According to Dickerson and Kemeny (2004), individuals possess a social self-preservation system that is sensitive to conditions of social evaluative threat or occasions where "an important aspect of the self-identity is or could be negatively judged by others" (p. 358). Actual or anticipated negative social evaluation was suggested by Dickerson and Kemeny as capable of activating a physiological response causing the release of the stress hormone cortisol.

This study defined stress as an event or events that provoke a physiological response through activation of the HPA axis, causing the adrenocortical release of cortisol (McEwen, 2000). The cognitive testing situation was interpreted as a stressful event when the cortisol concentration became 2.5 nmol/L higher than the level prior to the commencement of the tests (Kirschbaum et al. 1996). Sympathetic arousal, which can

also be a physiological response to perceived threats or stressors (Gunnar & Quevedo, 2007), was not considered as reflecting a psychological stress response, but did receive consideration as a potential moderator of cortisol's effects upon working memory.

The physiological stress response

Physiological responses to stress are partly mediated by the HPA axis, a system that reacts more slowly to a perceived threat or challenge than the instantaneous reactivity of the sympathetic nervous system. Stimulation of the HPA axis begins with the hypothalamus, which releases corticotropin-releasing hormone (CRH), causing pituitary gland secretion of adrenocorticotropin (ACTH). This latter hormone travels through the blood stream to trigger the adrenal cortex to release glucocorticoids, which include cortisol as their major constituent (Lupien et al. 2007). Following the perception of a stressor, cortisol levels may peak in about 15 to 30 minutes and then decline slowly to pre-stressor levels 60 to 90 minutes later due to cortisol's negative feedback on the HPA axis (de Kloet, Joels, & Holsboer, 2005).

Cortisol increases the availability of glucose for energy production, while also reducing the inflammatory effects of adrenaline and noradrenaline released from sympathetic activation (de Kloet et al., 2005). Being liposoluble, cortisol easily crosses the blood brain-barrier where it subsequently binds to glucocorticoid receptors that are located predominantly in the hippocampus and prefrontal cortex, brain regions involved in learning and memory (Lupien, Buss, Schramek, Maheu & Pruessner, 2005; Lupien et al., 2007).

Perceptions of threatening situations can also result in the activation of the sympathetic nervous system, causing the synaptic release of noradrenaline and the indirect release of both noradrenaline and adrenaline from the adrenal medulla into the blood stream. These catecholamine neurotransmitters prepare the body for the metabolic demands of the classic fight or flight response by mobilizing immune and inflammatory responses, increasing sweat production, elevating heart and respiratory rates, and increasing the flow of blood to the brain and muscles (Gunnar & Quevedo, 2007). Because of the effects of catecholamines upon the heart and sweat glands, sympathetic arousal was operationalized as increased level and variability in heart rate and sweat production, with the latter being determined through skin conductance measures.

Although neither type of catecholamine can cross the blood brain barrier, peripheral adrenaline stimulates β-adrenoreceptors in sensory vagal afferents, which terminate in a structure within the medulla known as the nucleus of the solitary tract (NTS) (van Stegeren, Wolf, Everaerd, Scheltens, Barkhof, & Rombouts, 2007). Neural projections from the NTS continue into the amygdala and then into other forebrain regions, leading to the activation of the noradrenergic system through postsynaptic binding of noradrenaline to adrenoreceptors in these areas (LaBar & Cabeza, 2006; McGaugh, 2000; Roozendaal, Okuda, De Quervain, & McGaugh, 2006; Roozendaal, Okuda, Van der Zee, & McGaugh, 2006; van Stegeren et al., 2007). According to animal studies, noradrenergic activation of the amygdala appears to regulate glucocorticoids' inhibitory effects upon working memory (Roozendaal, McReynolds et al., 2004), as well

as glucocorticoids' effects upon declarative long-term memory consolidation and retrieval. Glucocorticoids appear to interact with noradrenergic activation to enhance declarative long-term memory consolidation and inhibit declarative long-term memory retrieval (Roozendaal, Hahn, Nathan, de Quervain, & McGaugh, 2004; Roozendaal, Okuda, Van der Zee et al., 2006).

Such interactive effects, however, do not appear to play a role in declarative memory testing where recall is assessed immediately or within 30 minutes of material presentation. In this case, impairments seem to be associated primarily with glucocorticoid elevations, particularly in the morning when naturally-occurring levels are high (Wolf, 2008). The relationship of stress-induced cortisol elevations upon working memory and declarative memory tested via immediate and briefly delayed recall was the focus of the present study. The potential interactive effects of cortisol and sympathetic arousal upon working memory were also explored.

Age and education differences in the acute cortisol stress response to a cognitive test battery

As previously discussed, a situation may achieve salience as a stressor when it is perceived as threatening. According to the social self-preservation system hypothesis (Dikerson & Kemeny, 2004), physiological stress responses can be provoked when individuals believe that actual or anticipated social evaluation may reveal the absence of a personally and socially desirable trait or ability. This type of assessment is defined as

social evaluative threat and encompasses a situation where “an important aspect of the self-identity is or could be negatively judged by others” (p. 358).

In light of the social self-preservation system, cognitive testing may be more threatening to individuals who value having above average cognitive abilities and believe that such testing could demonstrate some loss of this cherished capacity. Older adults who possess a bachelor’s or more advanced degree may be particularly susceptible to the social evaluative threat that cognitive testing may present, especially if they are concerned that such testing might reveal a decline in the valued ability of intellectual functioning.

Neupert et al. (2006) investigated whether a series of cognitive tests evaluating the domains of vocabulary, short-term working memory, speed, and reasoning would elicit a physiological stress response in 74 adults, ages 25 to 74, from the Boston oversample of the first phase of the Midlife in the United States (MIDUS) Survey. Findings revealed that middle-aged and older adults having higher levels of education (i.e. a four year college degree or more) produced a significantly more positive cortisol slope throughout the testing occasions than the less educated participants and younger educated adults.

A somewhat similar investigation also examined whether the intensity of the stress response to cognitive challenge would vary depending upon education in an older adult sample, aged 65 to 80 years, selected from the London area (Steptoe et al. 2005). The higher educated group possessed educational qualifications extending from high school certificates to university degrees. The less educated group had completed only elementary school and had no additional education. The cortisol response to cognitive testing within these two older adult groups was

also compared to a younger adult group, aged 27 to 42 years, who held at a minimum a high school certificate.

Unlike the findings from Neupert et al. (2006), there were no differences in cortisol reactivity to cognitive challenge between the older adult group with higher education and the group with lower education. It should be noted, however, that the educational qualifications within the higher educated groups from both studies may not be comparable. The Steptoe et al. (2005) study's older higher educated group included participants who had only completed high school with participants who had university degrees; the Neupert et al. (2006) higher educated older adult sample did not include those with a high school or two-year post-secondary degree. Rather, it was composed of those older adults who had a bachelor's degree or more.

Differences in sample composition related to the differing aims of both research teams. Neupert et al. (2006) hypothesized that the cortisol response to cognitive testing would be higher in the educated older adult sample because of social evaluative threat; thus a greater focus upon those with a bachelor's and more advanced degree was necessitated. Steptoe et al. (2005), however, hypothesized that cortisol increase to cognitive testing would be greater in the lower educated sample, since individuals with lower socioeconomic status (SES) were thought to have greater activation of physiological stress processes to perceived threats (e.g. McEwen & Seeman, 1999; Steptoe & Marmot, 2002). Thus, Steptoe et al. sought to separate participants from the lowest SES (i.e. those who had no education beyond elementary school) from the rest of the group, leaving the higher educated group comprised of participants who had completed high school at a minimum.

In the Steptoe et al. (2005) study, both older adult groups, regardless of educational attainment, demonstrated greater cortisol increases compared to the younger educated sample. Thus, it is possible that the cognitive testing seemed more threatening to the older adult group, since it evoked a greater cortisol response.

These findings suggest that older adults may experience greater cortisol elevations to cognitive testing than younger adults. According to Neupert et al. (2006), education may moderate this relationship for middle-aged and older adults, whereby increased cortisol depends upon middle-aged and older adults having at least a bachelor's degree. This study examined whether middle-aged and older adults experienced a greater cortisol response to cognitive testing than younger adults, and whether education moderated this relationship.

Additional studies (Oswald et al., 2004, 2006) discussed in the next section have indicated that personality may also play a role in the acute cortisol response to a psychosocial stressor. The stressor utilized in these studies, however, was not a cognitive challenge as in the Neupert et al. (2006) or Steptoe et al. (2005) research. A laboratory-based stressor known as the Trier Social Stress Test (TSST; involves the public performance of five minutes of free speech and five minutes of mental arithmetic, see Kirschbaum, Pirke, & Hellhammer, 1993) was employed. Application of the TSST procedure provoked different cortisol responses depending upon personality trait (Oswald et al., 2004, 2006). There is limited research investigating the relationship between personality and potential cortisol responses to cognitive challenge, thus the present study explored whether personality was related to a cortisol

response to cognitive testing and whether this relationship was dependent upon gender, age or education.

Personality differences in the acute cortisol stress response

Oswald et al. (2006) found that in a sample of adults (aged 18-30 years; males: M = 21.7, SD = 2.8; females: M = 21.4, SD = 2.8), openness was positively associated with an increased cortisol response to the TSST, with those scoring high on this personality dimension having higher cortisol responses than those scoring low. This positive association was primarily explained by high scores on the openness subscales of actions and ideas.

Previous research has also shown a positive relationship between openness and an increased cortisol response to the TSST (Oswald et al., 2004). Although the potential mechanisms explaining this relationship are not clear, examining the characteristics of those scoring high in openness may provide some potential clues. These individuals tend to "have a rich and complex emotional life," are imaginative, and enjoy novelty (Costa & McCrae, 1992, p.6). Perhaps high emotional engagement, an active imagination, and novelty seeking predispose those having high openness to increased cortisol responses when exposed to psychosocial stressors. Although further research is needed to understand what mediates the relationship between high openness and an increased cortisol response to psychosocial stressors, this was not the focus of the present study.

Neither agreeableness nor conscientiousness was significantly associated with cortisol responses to the stressor in the Oswald et al. (2006) study. However, significant interactions between gender and either extraversion or neuroticism were associated with cortisol responses

to the TSST. These interactions were identified when the group's cortisol-time curve was deconstructed into the component parts of baseline cortisol, peak cortisol, fold stimulation (peak/baseline), and delta (peak minus baseline).

Fold stimulation and delta were found to correlate with extraversion and neuroticism in a gender-dependent manner. Specifically, a significant positive relationship was reported between fold stimulation and extraversion in men ($p = .04$), while a trend towards a negative relationship between fold stimulation and neuroticism was found in women ($p = .06$). Correlation of fold stimulation with the subscales for extraversion (i.e. warmth, gregariousness, assertiveness, activity, excitement, and positive emotion) explained that the positive association between fold stimulation and extraversion in men was accounted for primarily by warmth, activity, and positive emotions. Thus, those men scoring highest on the warmth, activity, and positive emotions subscales had the greatest increase between baseline and peak cortisol levels following the psychosocial stressor.

It is not clear why men scoring high in extraversion had a greater cortisol response to the stressor than those scoring low in this personality dimension. Previous research by Oswald et al. (2004) reported that high extraversion and high scores on the extraversion subscales of gregariousness, activity, and excitement-seeking were positively correlated with increased cortisol fold stimulation in the study group as a whole. Individuals who are defined as extraverted tend to be sociable and enjoy activity and excitement (Costa & McCrae, 1992), and these traits may predispose them to a more labile HPA response than those who are low in extraversion (Oswald et al., 2004). Although not the focus of this study, further research is

needed to explore why extraversion may be related to increased cortisol responses to psychosocial stressors.

Extraverted females did not experience an increased cortisol response to the stressor in the Oswald et al. (2006) research, while no gender differences were found in the earlier Oswald et al. (2004) study. The demographic characteristics in the Oswald et al. (2004) research (age: $M = 21.6$; $SD = 3.6$) were similar to that in the Oswald et al. (2006) study and the assessment of personality was the same (i.e. the Revised NEO Personality Inventory; see Costa and McCrae, 1992). Sample sizes, however, were smaller in the Oswald et al. (2004) study (males: $N = 9$; females: $N = 5$) compared to the Oswald et al. (2006) analysis (males: $N = 43$; females: $N = 25$). Thus, the detection of moderation effects may not have been possible in Oswald et al. (2004) due to the smaller sample size.

Gender differences in the Oswald et al. (2006) research may have been due to testing women who were in the follicular phase of their menstrual cycle. Women in the follicular phase of their menstrual cycle tend to produce smaller cortisol responses to a laboratory based psychosocial stressor than men (Kajantie & Philips, 2006). Exposure to a stressor during the luteal phase of the menstrual cycle results in cortisol responses similar to those found in men (Kajantie & Philips, 2006); thus, it is possible that if testing had been carried out while women were in the luteal phase of their menstrual cycle, there may have been no differences in the cortisol response of extraverted men and women.

As mentioned previously, a trend towards a negative relationship between fold stimulation and neuroticism was found in women and not men ($p = .06$). Whether this gender

difference would have remained if women were tested in the luteal phase of their menstrual cycle is also not known. This study investigated whether to control for women who were in the follicular phase of their menstrual cycle in order to equate as far as possible the potential stress-induced cortisol responses of men and women.

Correlation of fold stimulation with the neuroticism subscales (i.e. anxiety, anger, depression, self-consciousness, impulsiveness, and vulnerability) indicated that the initial association between fold stimulation and neuroticism in women was accounted for by a significant and negative relationship between cortisol fold stimulation and depression and self-consciousness. No significant associations were found between cortisol and the other neuroticism subscales. Thus, those women scoring highest on the depression and self-consciousness subscales had the smallest increase between their baseline and peak cortisol levels following the psychosocial stressor. It is possible that women scoring high in neuroticism had high baseline cortisol levels that did not increase much following the psychosocial stressor. Initial analyses revealed that participants "with higher pre-stress cortisol also had higher peak levels during the TSST than subjects with lower pre-stress cortisol" (Oswald et al., 2006, p. 1586). Additionally, "baseline cortisol levels were negatively associated with fold stimulation; subjects who had higher pre-stress cortisol levels had less relative change in levels than subjects whose initial levels were lower" (p. 1586). Data supporting that pre and post stressor levels of cortisol remained high in women scoring high in neuroticism, however, were not reported.

Since neuroticism is characterized by increased perceptions of stress and increased negative affect to stressors (Mroczek & Almeida, 2004), it may seem paradoxical that the female participants scoring high in trait neuroticism within Oswald et al.'s (2006) study experienced only a slight increase in cortisol following stressor exposure. Increased emotional reactivity to minor daily problems has also been found in male neurotics (Suls, Green, & Hillis, 1998), and yet there was no significant association between high neuroticism in males and cortisol response following the psychosocial stressor in either of the Oswald et al. (2004, 2006) studies. Further, there was no significant association between neuroticism and cortisol response to the stressor in either men or women in the Oswald et al. (2004) study.

The absence of an increased HPA response to the psychosocial stressor within neurotic participants could have been due to pre-existing HPA axis dysregulation. Adults scoring high in neuroticism have been found to have significantly higher cortisol levels 30 to 60 minutes after awakening than those scoring low in this personality dimension. This difference was not dependent upon gender or age, when testing young to middle aged adults only (range = 21-57 years) (Portella, Harmer, Flint, Cowen, & Goodwin, 2005). Following 60 minutes after awakening, however, there were no significant differences in cortisol levels between the low and high neuroticism groups.

Dysregulation of the HPA axis has been found in adult men who test high for trait neuroticism and lack a history of psychiatric illness (Zobel et al., 2004). After supplying a small dose of a synthetic glucocorticoid (i.e. dexamethasone) to adults with an average age of 35.9 years (*SD* = 13.6, range = 22.3-49.5), male participants scoring high in

neuroticism produced a cortisol response, indicating that the HPA axis was unable to respond to the glucocorticoid by dampening cortisol release as is typically expected in adults with normal HPA function. This same effect was not found in women scoring high in trait neuroticism, however, and whether dysregulation was associated with a blunted cortisol response to a psychosocial stressor was not explored.

The current study sought to examine whether increases in cortisol over time, while completing a battery of cognitive tests, differed according to the personality traits of openness, extraversion, and neuroticism. To gain an understanding of changes in within-person trajectories of cortisol over time, multilevel modeling was utilized instead of the calculation of fold stimulation and delta, which cannot provide intraindividual fluctuation/change information. Multilevel modeling was employed to determine whether intraindividual changes in cortisol trajectories were dependent upon the person-level factor of personality. Whether within-person increases in cortisol over time were also dependent upon between-person differences in gender, age or education was also investigated. Analyzing the degree of cortisol increase to cognitive testing is important, because acute cortisol elevations to completed tests may have a negative influence upon subsequent tests examining declarative and working memory (e.g. Kirschbaum et al. 1996; Lupien et al. 1997; Lupien et al., 1999; Tops, van der Pompe, Baas, Mulder, Boer, Meijman, & Korf, 2003).

Effects of acute cortisol elevations and sympathetic arousal upon declarative memory

Acute increases in cortisol have not been associated with impairments in non-declarative or implicit memory; whereas acute elevations have been associated with

various effects upon declarative or explicit memory, (Kirschbaum et al., 1996; Lupien et al. 1997; Lupien et al., 2007; Wolf, 2008; LaBar & Cabeza, 2006), defined here as the conscious recollection of previously learned facts or observed events (Eichenbaum, 2001). Elevated cortisol levels have differing effects depending upon the phase of memory tested (i.e. encoding/consolidation or retrieval) and the length of time between presented material and recall (i.e. immediate or a brief delay of 10 to 30 minutes vs. a long-term delay of hours to days). Sympathetic arousal appears to play a role when testing long-term consolidation or long-term retrieval. (Abercrombie, Speck, & Monticelli, 2006; Cahill, Gorski, & Le, 2003; Roozendaal, Hahn, Nathan, de Quervain, & McGaugh, 2004; Roozendaal, Okuda, Van der Zee et al., 2006; Wolf, 2008). However, the effects of sympathetic activation are less clear when declarative memory is tested through immediate or briefly delayed (10-30 minutes) recall, when phase of memory effects cannot be easily determined (Wolf, 2008).

Cortisol elevations appear to interact with sympathetic activation to enhance declarative long-term memory consolidation (Abercrombie et al., 2006; Cahill, Gorski, & Le, 2003; Roozendaal Okuda, Van der Zee et al. 2006) and impair declarative long-term memory retrieval (Roozendaal, Hahn et al., 2004; Wolf, 2008). These effects seem to be strongest when testing memory for emotionally arousing information (Wolf, 2008). When analyzing the effects of cortisol increase on the immediate or slightly delayed recollection of material, recall of neutral items appears to be more impaired than the recollection of emotionally arousing ones (Tops, et al. 2003; Wolf, 2008).

This study examined whether cortisol elevations, associated with the completion of previous cognitive tests, hindered immediate and briefly delayed recall tested towards the end of a cognitive testing battery. Since consolidation and retrieval processes could not be analyzed specifically, sympathetic arousal was not investigated as a potential moderator of cortisol-associated declarative memory impairment. Rather, potential moderators included time of day of testing and age, and these will be addressed in the next sections.

Effects of acute cortisol elevations upon immediate or briefly delayed recall. To examine the effects of acute endogenous, or naturally-occurring, cortisol elevations upon memory in humans, cortisol can be manipulated by exposing participants to a laboratory stressor (e.g. the TSST). After exposure to the stressor, some participants (i.e. the 'responders') produce a significant increase in cortisol compared to baseline levels, while others (i.e. the 'non-responders') do not.

When testing for the immediate and briefly delayed recall of generic or neutral word lists, cortisol responders remembered fewer words than cortisol non-responders (Kirschbaum et al., 1996; Lupien et al., 1997). This impairing effect was reported in young adult cortisol responders (undergraduate students), who were exposed to the laboratory stressor immediately prior to learning (Kirschbaum et al., 1996), and elderly adult responders (62-83 years of age), who were exposed to the stressor after learning and prior to a briefly delayed recall (Lupien et al., 1997).

The impairing effects of cortisol upon immediate recall have been demonstrated in a pharmacological study involving male undergraduate students (18-27 years of age). In this study, cortisol was orally administered two hours prior to the immediate free recollection of neutral, pleasant, and unpleasant words (Tops et al., 2003). The average cortisol level in the treatment group at the time of cognitive testing was approximately 22.0 nmol/L, a value only slightly higher than the mean value observed in the cortisol responders in the Kirschbaum et al. (1996) and Lupien et al. (1997) studies (17.7 nmol/L and 19.5 nmol/L, respectively). Participants in the treatment group recalled fewer pleasant and neutral words than those in the placebo group. There were no significant differences between groups in the recollection of unpleasant words.

Additional evidence suggests that the effects of cortisol upon delayed recall may also be dependent upon time of day of testing (Het et al., 2005, Lupien et al., 2002). Cortisol levels peak in the morning and slowly decline in the afternoon until reaching a trough in the evening and during sleep, at least for healthy individuals who awake in the morning and return to sleep during the night (this pattern may reverse in some who stay awake through the night and sleep during the day; see Hennig, et al. 1998). Due to the greater elevation of cortisol in the AM phase than in the PM, stress-induced cortisol elevations may be more likely to impair memory function in the morning than in the afternoon (Het et al., 2005, Lupien et al., 2002).

According to a meta-analysis examining the cognitive effects of the pharmacological administration of cortisol prior to the learning of words, pictures, faces

or objects, morning administration of cortisol significantly impaired delayed recall (d = -.40), while afternoon dosing produced a small, but significant memory enhancement (d = .22) (Het et al., 2005). The cognitive effect of cortisol dosing in each of the studies was compared to a placebo group, and each study reviewed was conducted either in the morning or afternoon. Effect sizes represented averages, and there was substantial variability of effects for those studies conducted in the morning ($-.77 \leq d \leq -.03$, $N = 4$) and in the afternoon ($.02 \leq d \leq .41$, $N = 8$). This may have been due to the use of different memory tasks, varying time delays in the testing of memory lasting from 3 minutes to 1 week, and different pharmacological doses of cortisol. Time of day effects were not analyzed for studies where cortisol was given prior to the retrieval of already learned material, since impairing effects were found for all studies ($N = 4$) examined. The average effect size for these latter studies was moderate (d = -.49) and significantly different from zero.

The present study investigated whether endogenous cortisol elevations to previously completed cognitive tests had an impairing effect upon declarative memory tested via immediate and delayed (approximately 30 minutes) recall of subsequently learned material. Whether effects were dependent upon age and time of day of testing was also analyzed. Although it was recognized that acute cortisol increase appears to hinder memory for neutral items rather than emotionally arousing ones (Tops et al., 2003), the proposed study was only able to assess general recall and was not able to

differentiate whether recall of neutral items was more impaired than the recollection of emotionally arousing ones.

Effects of acute cortisol elevations and sympathetic arousal upon working memory

Working memory is a form of memory where information is "actively maintained and/or manipulated in conscious awareness over a short period of time" (LaBar & Cabeza, 2006, p. 56). It is a memory function that is dependent upon the prefrontal cortex (Owen, Downes, Sahakian, Polkey, & Robbins, 1990), a cortical region rich in glucocorticoid receptors (Lupien et al., 2007). Studies examining cortisol's effects upon working memory have produced mixed findings. In selected research, where participants were undergraduate students randomly assigned to a stress or control condition, the working memory of cortisol responders in the stress group was either: 1) not affected (Kuhlmann, Piel, & Wolf, 2005), 2) impaired at high working memory loads, but not at low loads (Oei, Everaerd, Elzinga, Van Well, & Bermond, 2006), or 3) impaired only during a period of acute sympathetic activation during exposure to the laboratory stressor (Elzinga & Roelofs, 2005). Factors contributing to differences in these findings might include variations in stress-induced peak cortisol concentrations, type of working memory test employed, and whether cortisol elevations occurred concurrently with sympathetic arousal.

Prior to working memory assessment, peak cortisol levels in the stress group reached an average of 34.4 nmol/L in the Oei et al. (2006) study. In contrast, peak concentrations (i.e. 16.0 -17.0 nmol/L) in the Kuhlmann et al. (2005) research, where no

significant effects were found, were lower. Oei et al. utilized a test of working memory (i.e. the Sternberg item recognition task; Sternberg, 1966) that assessed low and high working memory processing load, and found that working memory was significantly impaired in the stress group at high working memory loads, but not at low loads, compared to the control group. Kuhlmann et al. used a working memory task (i.e. the forward and backward digit span test; Weschler, 1987) that may not have produced high working memory loads for the participants. This combined with the lower cortisol levels in their research might explain why a significant association between stress-induced cortisol increase and working memory performance was not found.

Elzinga and Roelofs (2005), unlike the two previously discussed studies (Kuhlmann et al. 2005; Oei et al. 2006), examined whether the influence of cortisol upon working memory was dependent upon sympathetic arousal, as measured by increased heart rate and blood pressure. To assess this interaction, participants assigned to the stress condition were tested with three parallel versions of the forward and backward digit-span test during three separate conditions: one at baseline prior to the TSST, a second following the TSST and in front of the TSST audience, and a third after the stress task when no audience was present.

Although cortisol responders in the stress condition demonstrated peak cortisol levels (i.e. 16 – 18 nmol/L) comparable to those in research where no significant effects between cortisol increase and working memory were reported (Kuhlmann et al., 2005), the working memory capacity of the cortisol responders in the Elzinga and Roelofs

(2005) study declined significantly compared to baseline measures. This dysfunction in working memory, however, happened only during sympathetic activation (as measured by increased heart rate and blood pressure) occurring during working memory testing in front of the TSST audience. When measures of sympathetic arousal returned to baseline during testing without the audience being present, there were no significant differences between the cortisol responder and non-responder groups in working memory performance, even though cortisol levels remained high in the cortisol responder group (see Figure 1).

Sympathetic arousal leads to noradrenergic activation through the postsynaptic binding of noradrenaline to adrenoreceptors in the amygdala and other forebrain regions (McGaugh, 2000; van Stegeren et al., 2007). Elzinga and Roelofs' (2005) findings that sympathetic arousal appears to interact with cortisol elevations to impair working memory seem congruent with previous animal research, suggesting that glucocorticoids' impairing effects upon working memory is dependent upon noradrenergic activation, and perhaps noradrenergic activation within the amygdala specifically (Roozendaal, McReynolds et al. 2004).

When corticosterone (i.e. the major glucocorticoid in rats) was infused into rats with drug-induced lesions of the amygdala (specifically the basolateral amygdala) and into rats with functional amygdala, working memory performance was impaired in the rats with intact amygdala, but performance was not impaired in rats with amygdala lesions. This indicated that lesions of the amygdala blocked the working memory

impairment associated with corticosterone and that an intact basolateral amygdala was needed for this impairment (Roozendaal, McReynolds et al., 2004). Although not tested specifically, corticosterone infusions in the rats with functional amygdala may have resulted in noradrenergic activation within the amygdala, which helped to modulate the corticosterone memory impairment. Glucocorticoid infusions can increase levels of noradrenaline in a rat's brain and facilitate noradrenergic activation (Roozendaal, Okuda, De Quervain et al., 2006). The importance of noradrenergic activation in working memory impairment was addressed in the Roozendaal, McReynolds et al. (2004) research by infusing a drug that dampens sympathetic arousal by inhibiting activation of adrenoreceptors (i.e. the β- adrenoreceptor antagonist propranolol). When this drug and corticosterone were injected into rats prior to testing, the impairing effects of corticosterone upon working memory were prevented, thus implying that corticosterone induced impairment of working memory also involves noradrenergic activation, and perhaps noradrenergic activation in a functional basolateral amygdala (Roozendaal, McReynolds et al., 2004.) Thus, the Roozendaal, McReynolds et al. (2004) research provides a possible explanation for why the Elzinga and Roelofs' (2005) undergraduate participants, who had stress-induced elevations in cortisol and increased sympathetic arousal, may have experienced working memory impairment. Elevated cortisol and noradrenergic activation in the amygdala, due to increased arousal, may have worked together to facilitate this transient deficit.

Whether the effects of cortisol elevations upon working memory are also dependent upon the time of day of cortisol increase has not been sufficiently investigated (Het et al., 2005). Lupien, Buss et al. (2005) have suggested that high pharmacological doses of glucocorticoids given in the AM phase may have more impairing effects upon attentional/working memory abilities than similar doses administered during the PM. This suggestion, however, was based upon two studies, where attention (and not working memory specifically) was tested in the PM in one study (Hsu, Garside, Massey, & McAllister-Williams, 2003) and working memory was analyzed in the AM in another (Lupien, Gillin, & Hauger, 1999). Although attentional processes appear to play a role in working memory function (Cowan, 2000), it is uncertain from these studies whether working memory is differentially affected by cortisol increases in the morning and afternoon, since only one study tested working memory specifically.

In the research reviewed here, working memory testing occurred around 10 AM in two studies (Elzinga & Roelofs, 2005; Oei et al., 2006) where impairing effects were found, and at around 12 PM in another (Kuhlmann et al., 2005) where no significant effects were reported. As discussed previously, differences in these findings may have been due to variations in peak cortisol levels, type of working memory test employed, and sympathetic arousal levels. Further studies are needed to determine whether differences in working memory performance are also dependent upon time of day of cortisol increase.

The current study investigated whether endogenous cortisol elevations associated with previously completed cognitive tests had an impairing effect upon working memory. Whether effects were dependent upon age, time of day of testing and sympathetic arousal occurring during testing was also analyzed. Because β-adrenoreceptor antagonists may potentially influence working memory performance, and since this type of medication is commonly used to treat hypertension (Fisher & Williams, 2005), hypertensive status was used as a proxy for participants taking β-adrenergic receptor antagonists and was controlled in analyses where sympathetic arousal was examined as a moderator of working memory performance.

Effects of naturalistic stressors upon working memory

All of the studies reviewed in this proposal regarding cortisol's effects upon working and declarative memory have come from the experimental literature, where participants were exposed to a laboratory-based psychosocial stressor. Few studies have examined memory effects due to cortisol elevations in response to naturalistic stressors. One exception comes from Lewis, Nikolova, Chang, and Weekes (2008), who examined the effects of high examination stress upon working memory in undergraduate students. A within-subject comparison of working memory performance during a period of high examination stress (i.e. 3 or more tests or assignments due that week) and low examination stress (i.e. when no classes were in session) was conducted. Examination stress was measured by cortisol and a perceived stress scale.

Participants experienced no significant change in forward digit span measures between the low and high stress times; however, they did experience an improvement in the backward digit span during high examination stress. The undergraduate participants perceived significantly more stress in the high stress period and also demonstrated a significant increase in cortisol (M = 0.11 ug/dL or 3.0 nmol/L) compared to levels during the low stress period (M = .09 ug/L or 2.5 nmol/L). Thus, the undergraduates during the high examination stress period, when cortisol levels were elevated, exhibited enhanced working memory capacity, at least for backward digit span. This stands in contrast to some of the previously discussed experimental studies (e.g. Elzinga & Roelofs, 2005; Oei et al., 2006), where stress was associated with working memory impairment.

The varying findings may be explained by considering the differences in the concentration of cortisol during stress. The mean cortisol level in the Lewis et al. (2008) study was far lower than peak levels measured in the Oei et al. (2006) or Elzinga and Roelofs (2005) study (i.e. 34.4 nmol/L and 16 – 18 nmol/L, respectively). Although speculative, time of day effects may also have played a role. Lewis et al. participants were not tested in the morning as in the Oei et al. and Elzinga and Roelofs research. Sessions began at around 3:30 PM or 5:30 PM and lasted for approximately two hours. Slight increases in cortisol during the afternoon and evening when circulating cortisol levels are lower than those in the morning may have contributed to working memory enhancement (Lupien, Buss, et al., 2005). Comparing effects of time of day of testing by

assessing participants in the morning and the afternoon would have been an important contribution to the Lewis et al. research.

There continues to be a need to investigate the effects of cortisol upon memory in a more naturalistic setting by taking into consideration the time of day of testing, and sympathetic arousal in the case of working memory, which was also not addressed in the Lewis et al. (2008) study. Although the present study did not examine naturalistic stressors per se, it did explore whether participants who completed a battery of cognitive tests within a naturalistic setting (i.e. their home) experienced a stress response, and whether working and declarative memory performance was dependent upon this response, as well as time of day of testing and age.

Effects of stress upon memory in aging adults

Much of the research regarding the effects of acute increases in endogenous cortisol upon memory has utilized participants who are young adults. Few studies have examined effects upon middle-aged and older individuals. The Lupien et al. (1997) research discussed in the section on declarative memory was one exception, and findings appeared to indicate that the impairing effects of endogenous cortisol upon recall found in young adults (Kirschbaum et al., 1996) can also be replicated in older adults. Although not exploring the effects of cortisol and sympathetic arousal specifically, other research has shown that naturally-occurring daily stress has similar effects upon working memory performance within young (*M age* = 20.21, *SD* = 1.09, range = 18-24) and older (*M age* = 80.23, *SD* = 6.30, range = 66-95) adults (Sliwinski, Smyth, Hofer, & Stawski, 2006).

Results indicated that the within-person reaction times of young and older adults were slower on a 2-back working memory task on days when at least one stressor was reported, compared to days when no stressors were reported. Cortisol and sympathetic arousal were not measured in this research, so it is not known whether potential stress-induced elevations of cortisol might have interacted with sympathetic arousal to influence cognitive performance deficits in the young and older adult participants.

Since aging in general may play a role in the performance of declarative and working memory tasks, it would be important to determine whether the effects of acute increases in cortisol upon declarative memory are dependent upon age, and whether the effects of the interaction of cortisol and sympathetic arousal upon working memory are also dependent upon age. Testing of age differences in working memory capacity has revealed that older adults (age, $M > 60$ years) perform more poorly than younger adults (age, $M < 30$ years) (Bopp & Verhaeghen, 2005). Further, cross-sectional studies have reported a linear decline in performance beginning in the decade of the 20s and 30s for a variety of working memory tasks (Borella, Caretti, & De Beni, 2008) and for the Weschler (1997) letter-number sequencing task (Myerson, Emery, White & Hale, 2003), which is a working memory test that was employed in the present analysis. Age differences have also been reported for declarative memory performance; however, declining function was not manifested in a longitudinal study until after 60 years of age (Rönnlund, Nyberg, Nilsson, & Bäckman, 2005). Similar results were found in a cross-sectional analysis when educational attainment was controlled (Rönnlund et al., 2005).

This study analyzed young, middle-aged, and older adults to determine whether the effects of acute increases in cortisol upon working and declarative memory differed by age and time of day of testing. Whether differences in working memory performance were dependent upon sympathetic arousal was also addressed.

Research Questions and Hypotheses

Unless hypotheses specifically suggested investigating the time of day of testing as an independent variable, all analyses controlled for time of day of testing to take into consideration the diurnal variation of cortisol.

1) Is cortisol reactivity (defined as a change in cortisol slope over time) that is associated with completing a cognitive testing battery dependent upon an interaction between age and education?

    a. Hypothesis: Middle-aged and older adults with higher education (i.e. bachelor's degree or more) will experience a greater cortisol response, as demonstrated by a more positive cortisol slope, to the cognitive testing battery than younger adults and less educated middle-aged and older adult participants (Neupert et al., 2006).

2) Is cortisol reactivity that is associated with completing a cognitive testing battery dependent upon the personality traits of extraversion, openness, or neuroticism?

    a. Hypothesis: Participants scoring high in the traits of extraversion and openness will experience a greater cortisol response, as demonstrated by a more

positive cortisol slope, than participants scoring low in these personality dimensions (Oswald et al., 2004, 2006).

b. Because of mixed results in the literature regarding the cortisol responses of neurotics to psychosocial stressors (Oswald et al., 2004, 2006), this study explored whether the cortisol response of those scoring high in neuroticism was different than those scoring low in neuroticism. No predictions were made concerning whether those scoring high in neuroticism would have a blunted cortisol response or an increased cortisol response compared to those scoring low in this personality dimension.

3) Is cortisol reactivity that is associated with completing a cognitive testing battery dependent upon an interaction between personality trait (i.e. extraversion, openness, or neuroticism) and gender (Oswald et al., 2006)?

4) Is cortisol reactivity that is associated with completing a cognitive testing battery dependent upon an interaction between: a) age and the personality traits of extraversion, openness, or neuroticism, or b) education and the personality traits of extraversion, openness, or neuroticism (Neupert et al., 2006; Oswald et al., 2006)?

5) Are working and declarative memory performance decrements dependent upon cortisol elevations, age, and time of day of testing?

a. Hypothesis: When controlling for the effects of education, participants exhibiting the highest cortisol elevations in response to tests completed prior to

the working and declarative memory tasks will perform less well on the working and declarative memory tests than those showing little or no cortisol response to the previous testing (e.g. Kirschbaum et al. 1996; Lupien et al. 1997; Lupien et al., 1999; Tops et al., 2003).

b. No specific predictions were made regarding whether working and declarative memory performance decrements are dependent upon an interaction between age, cortisol elevations, and time of day of testing.

6) Are working memory performance decrements dependent upon cortisol elevations, age, and sympathetic arousal, when controlling for education and hypertensive status?

Method

Participants

Participants were from the Boston oversample of the second phase of the Midlife Development in the United States (MIDUS II), a national survey of health and well-being funded by the National Institute on Aging. This sample included longitudinal participants (N = 104) from the original MIDUS I Boston subsample, a probability sample of the Greater Boston area (see Lachman & Firth, 2004), as well as an additional probability sample of non-longitudinal participants from the same Boston area (N = 309). Although longitudinal participants were assessed previously in the MIDUS I, data from the first wave were not utilized. Out of this total sample of 413 participants, the longitudinal participants completed all of the 7 cortisol assessments and 148 to 153 of the non-

longitudinal participants completed cortisol assessments 1-5 and 7 (see Appendix A). Age and education data were not available for participants who chose not to have their cortisol collected.

All data for the current study were collected between 2004 and 2007 and each participant was tested only once. Participants ranged in age from 34 to 88 (M = 59.93; SD = 12.80; N = 257) Those with a history of stroke, diabetes, neurological disorders, or who did not report English as their language spoken at home when growing up were excluded from the analyses.

Procedure

Participants were administered 15 cognitive tests while in their home (see Appendix A). Ten tests evaluating working memory, vocabulary, reasoning, and speed were completed prior to the assessment of immediate recall (i.e. logical memory immediate; Wechsler, 1997). Following this test, two more tests were completed prior to another assessment of working memory (i.e. letter-number sequencing; Wechsler, 1997). The test of briefly delayed recall (i.e. logical memory delayed; Wechsler, 1997) was administered immediately after this working memory task.

Salivary cortisol samples were collected over an average of 95.42 minutes (SD = 20.71 minutes) during cognitive testing. Samples were taken seven times from the longitudinal participants and six times from the non-longitudinal participants (sample #6 was not collected from the non-longitudinal participants).

Psychophysiological indicators of sympathetic arousal (e.g. heart rate and skin conductance level due to amount of perspiration on the finger) were measured using a MEDAC System/3^{TM} on the longitudinal participants only during the logical memory immediate, letter-number sequencing, and logical memory delayed tests. The instrument was manufactured by NeuroDyne Medical Corporation (Cambridge, MA) and is referred to as "Neurodyne" in Appendix A.

Following the completion of all cognitive tests, a diary, demographic questionnaire, photograph, and peak expiratory flow testing were taken or given to the participants.

The personality traits of extraversion, openness, and neuroticism were measured using the Revised Midlife Development Inventory (MIDI) Personality Scale (Lachman & Weaver, 1997; *Addendum for MIDI Personality Scales,* 2005) via a mail survey prior to the in-home cognitive testing procedure.

Measures

Salivary cortisol. Participants were instructed to avoid caffeinated products and to collect samples before they ate, drank, or brushed their teeth, since these activities may elevate cortisol levels. Salivary cortisol was collected via Sarstedt Salivette, a cotton-like swab that participants chewed on for about one minute. The saturated swab was then placed into a plastic container, which was stored in a Ziploc bag and refrigerated until transfer to an airtight freezer (-20.0^{o}C) for storage. Specimens were shipped to the

University of Dresden, Germany for analysis (cf. Kirschbaum et al. 1996). Cortisol was measured in nmol/L; higher values indicate more of the stress hormone.

During the in-home testing procedure, the first salivary cortisol sample was collected after the participant's informed consent and prior to the Spielberger State anxiety test (a measure that was not used in this study). Four cortisol specimens were collected prior to the assessment of immediate recall (i.e. logical memory immediate; Wechsler, 1997) and five were collected prior to the assessment of working memory (i.e. letter-number sequencing; Wechsler, 1997) and briefly delayed recall (i.e. logical memory delayed; Wechsler, 1997). Refer to Appendix A to determine when cortisol samples were collected during the in-home cognitive testing procedures.

Psychophysiological measures. Indicators of sympathetic arousal (i.e. increased heart rate and skin conductance) were measured on the longitudinal participants only and were determined using the MEDAC System/3^{TM}, referred to as NeuroDyne in Appendix A. This device collects data at a rate of 100 times per minute and was turned on just prior to the logical memory immediate and letter-number sequencing tests. Only data collected during the letter-number sequencing test was used in this study.

The MEDAC System/3^{TM} measured skin conductance level with a gold plated 2.5 cm^2 finger tip conductor. Greater skin conductivity occurs due to increased perspiration from the sweat glands, which can occur due to activation of the sympathetic nervous system (Gunnar & Quevedo, 2007). Increased skin conductivity produces larger values measured in micromhos. Values less than 2 micromhos reveal fairly low conductivity and arousal. Values between 5 and

10 micromhos indicate fairly high conductivity and arousal, and very high conductivity and arousal are reflected in values of 30 to 50 microhoms (NeuroDyne Medical Corporation, 1995). Assessment of skin conductance levels using a NeuroDyne monitor has been used in previous research (Auman, Bosworth, & Hess, 2005; Levy, Hausdorff, Hencke, & Wei, 2000).

The MEDAC System/3^{TM} calculated heart rate in beats per minute from measurements of pulse inter-beat-intervals using a finger tip photoplethysmograph sensor. Inter-beat intervals (IBI) were measured as the intervals between pulse wave peaks (i.e. pulse wave rise time and fall time). Heart rate was calculated from IBI using the following formula: (1/mean IBI) x 60.

Declarative memory. Logical memory immediate and delayed from the immediate memory and general memory test indexes of the Wechsler Memory Scale-3rd edition (WMS-III; Wechsler, 1997) was used to assess declarative memory. For logical memory immediate, a paragraph was read to participants who were then asked to recall as much of the material as possible immediately following paragraph presentation. For logical memory delayed, participants were asked to recall as much information as possible from the original paragraph after finishing three cognitive tests (i.e. task switching, trails, and letter-number sequencing). Participants were scored depending upon the accurate recall of phrases from the paragraph. The highest score possible was 25 (see Appendix B for scoring).

Whether the paragraph was emotionally arousing was not tested. The valence of some of the content in the paragraph appeared to range from neutral to somewhat negative (see Appendix B for paragraph content). Due to how the test was scored, it was not be possible to determine if valence influenced recall since neutral and negatively toned content were mixed.

Studies suggest that cortisol affects the immediate and briefly delayed recall of neutral and not negatively valenced material (see Wolf, 2008).

Working memory. Letter-number sequencing from the working memory test index of the WMS-III (Weschler, 1997) was used to measure working memory. Participants heard an alternating series of numbers and letters (e.g. 8 N 3 H) and then recalled the numbers in ascending order, followed by the letters in alphabetical order (e.g. 3 8 H N). There were seven levels ranging from two characters through eight characters with each level containing three trials. The total number of correct responses was graded and the highest possible score was 21.

Personality. The personality traits of extraversion, openness, and neuroticism were measured using the Revised Midlife Development Inventory (MIDI) Personality Scale (Lachman & Weaver, 1997, *Addendum for MIDI Personality Scales,* 2005). Participants rated a list of adjectives according to how well they thought the words described them (1 = a lot, 2 = some, 3 = a little, 4 = not at all; see Appendix C for scale). Adjectives for extraversion included: outgoing, friendly, lively, active, and talkative (α = .78; *Addendum for MIDI Personality Scales,* 2005). Adjectives for openness were as follows: creative, imaginative, intelligent, curious, broadminded, sophisticated, and adventurous (α = .74; *Addendum for MIDI Personality Scales,* 2005). And adjectives for neuroticism were: moody, worrying, nervous, and calm (α = .81; *Addendum for MIDI Personality Scales,* 2005). All descriptive ratings were reverse coded, except for calm. Participants from the Boston subsample completed the personality scale via a mail survey prior to the in-home cognitive testing procedure.

Covariates. Diagnoses or conditions reported by participants that could potentially be treated with corticosteroid therapy were considered as potential covariates, since corticosteroid medications (e.g. prednisone) disrupt the normal functioning of the HPA axis (see Gong, 2005). These diagnoses or conditions included: a) joint or bone disease, b) asthma or bronchitis or emphysema, c) other lung problems, d) sciatica or lumbago or backache, e) hemorrhoids, f) lupus or other autoimmune disorder, g) adrenal insufficiency (e.g. due to Addison's disease), and h) treated Cushing's disease (see Kasper et al., 2005; Koda-Kimble, Young, Kradjan, & Guglielmo, 2005). Prior to treatment, Cushing's disease causes hypercortisolism, often due to an ACTH secreting pituitary adenoma (Melmed & Jameson, 2005). Once this tumor is removed, many patients require low-dose corticicosteroid replacement therapy for about one year (Melmed & Jameson, 2005).

Conditions, medications other than corticosteroid therapy, and activities that can influence the HPA axis or concentration of salivary cortisol were also considered as covariates. These included a) pregnancy, b) smoking cigarettes, c) women taking hormone replacement therapy, d) women who were in the follicular phase of their menstrual cycle, e) women taking birth control, and f) drinking caffeinated beverages on the day of in-home testing.

Pregnancy and smoking cause an increase in salivary cortisol levels, and pregnancy is associated with an attenuated cortisol response to psychosocial stressors (Kirschbaum & Hellhammer, 1994, 1989). Estrogen replacement therapy (e.g. estradiol therapy) in postmenopausal women appears to have mixed effects upon the HPA axis's response to psychosocial stressors. Some studies have shown no significant differences between the

placebo and estradiol group in salivary cortisol elevations to a psychosocial stressor, while others have reported a significant increase in salivary cortisol in the estradiol group (Kajantie & Phillips, 2006).

When exposed to a laboratory based stressor, women who were in the follicular phase of their menstrual cycle or who were taking oral contraceptives experienced smaller rises in salivary cortisol compared to men. In contrast, women in the luteal phase (when estrogen levels are lower) produced salivary cortisol levels similar to men when exposed to the same type of stressor (Kajantie & Phillips, 2006).

Mixed results have been found regarding the association between caffeine and cortisol elevations. The intravenous administration of caffeine has been associated with increases in blood cortisol (Nickell & Uhde, 1994), while no association has been found between long-term (i.e. 7 days) oral caffeine administration and cortisol increase (MacKenzie et al., 2007).

Since smoking cigarettes and drinking caffeinated beverages may also affect the measures of sympathetic arousal, these activities were considered as potential covariates in analyses exploring whether working memory performance was dependent upon an interaction between sympathetic arousal and cortisol. Cigarette smoking has been related to the increased release of adrenaline and noradrenaline, resulting in elevations of heart rate and blood pressure (Cryer, Haymond, Santiago, & Shah, 1976). Caffeine administration at rest has been associated with increases in blood pressure and muscle sympathetic activity (Sudano et al., 2005). This increase, however, was not amplified further following administration of a laboratory-based stressor (Sudano et al., 2005).

Finally, since β-adrenergic receptor antagonists dampen sympathetic arousal by inhibiting activation of adrenoreceptors, and since this type of medication is commonly used to treat hypertension (Fisher & Williams, 2005), hypertensive status was used as a proxy for participants taking β-adrenergic receptor antagonists and was controlled in analyses where sympathetic arousal was examined as a moderator of working memory performance.

Analyses

Research questions and hypotheses 1-4

Multilevel modeling (MLM) was used to test the hypotheses and research questions relating to whether the cortisol response to cognitive challenge was dependent upon age, education, personality, and gender. MLM allowed for the analysis of repeated cortisol measures across the cognitive testing period, utilizing data that were not perfectly balanced. Repeated-measures analysis of variance requires balanced datasets, and thus was a less appropriate choice for the study. Further, MLM provided a means for determining interindividual differences in cortisol change over time. With this statistical technique, both initial baseline (i.e. level prior to testing) and slope across the testing period was examined and interindividual differences in cortisol trajectories was detected. Specifically tested was whether the within-person change in cortisol over time was dependent upon person-level traits of extraversion, openness, or neuroticism, or an interaction of the following characteristics:

a) age and education

b) personality (i.e. extraversion, openness, or neuroticism) and gender

c) personality (i.e. extraversion, openness, or neuroticism) and age

d) personality (i.e. extraversion, openness, or neurotcism) and education

All person-level variables used to create interaction terms were grand-mean centered prior to analysis to reduce non-esssential multicollinearity effects. To decompose significant interactions, graphs were plotted by calculating predicted points based upon the mean, and one standard deviation above and below the mean for specific variables in significant interaction terms. Simple slopes were determined utilizing computational tools established by Preacher, Curran, and Bauer (2006).

Research question and hypothesis 5 and research question 6

Moderated regression was employed to test whether elevated cortisol in response to completed cognitive tests impaired performance on subsequent assessments of declarative and working memory, and whether memory impairment was dependent upon specific moderators (i.e. age and time of day of testing). Unlike ANOVA, regression allowed for the use of continuous independent variable measures, such as cortisol.

Cortisol was operationalized in two ways: 1) the cortisol level (nmol/L) reached just prior to memory testing, and 2) the change in cortisol from baseline to the level just prior to memory testing. The cortisol level found in salivette # 4 (see Appendix A to reference salivette numbers) was used when logical memory immediate was the dependent variable. The cortisol level found in salivette # 5 was used when logical

memory delayed and working memory were the dependent variables. Cortisol change was calculated as follows:

- When logical memory immediate was the dependent variable: cortisol change = salivette # 4 – salivette # 1.
- When logical memory delayed and working memory were dependent variables: cortisol change = salivette # 5 – salivette # 1.

Separate moderated regression analyses were used to examine the association of cortisol level and cortisol change upon memory within models testing for moderator effects and controlling for the effects of education:

> Memory test performance = education + **cortisol level (or cortisol change)** + age + time of day of testing + cortisol level (or cortisol change) x age + cortisol level (or cortisol change) x time of day of testing + age x time of day of testing + cortisol level (or cortisol change) x age x time of day of testing

Moderated regression analyses were also utilized to explore whether working memory performance decrements were dependent upon cortisol elevations, age, and sympathetic arousal. Covariates included education, time of day of testing, smoking status, and hypertensive status. The mean and standard deviation of skin conductance and heart rate were used as measures of sympathetic arousal in separate moderated regression models where cortisol was operationalized as either cortisol level or cortisol change, as previously discussed:

> Working memory = education + time of day of testing + hypertensive status + smoking status + **cortisol level (or cortisol change)** + age + **heart rate mean (or standard deviation)** + cortisol level (or cortisol change) x age + cortisol level (or cortisol change) x heart rate mean (or standard deviation) + age x heart rate mean (or standard deviation) + cortisol level (or cortisol change) x age x heart rate mean (or standard deviation)

Working memory = education + time of day of testing + hypertensive status + smoking status + **cortisol level (or cortisol change)** + age + **skin conductance mean (or standard deviation)** + cortisol level (or cortisol change) x age + cortisol level (or cortisol change) x skin conductance mean (or standard deviation) + age x skin conductance mean (or standard deviation) + cortisol level (or cortisol change) x age x skin conductance mean (or standard deviation)

All variables used to create interaction terms were centered prior to analysis to reduce non-essential multicollinearity effects. To decompose significant interactions, graphs were plotted by calculating predicted points based upon the mean, and one standard deviation above and below the mean for specific variables in significant interaction terms. Simple slopes were determined utilizing computational tools established by Preacher, Curran, and Bauer (2006).

Results

Determining covariates

No significant correlations were found between the seven salivary cortisol measures and reports of joint or bone disease, other lung problems, sciatica or lumbago or backache, hemorrhoids, or lupus or other autoimmune disorder. Significant correlations were found between report of asthma or bronchitis or emphysema and salivary cortisol measure 1 [$r(218) = .15$, $p < .05$], 2 [$r(218) = .15$, $p < .05$] and 3 [$r(219) = .14$, $p < .05$]. Participants reporting the presence of asthma or bronchitis or emphysema had lower salivary cortisol at Time 1 through 3 than participants who did not report the presence of these conditions. No significant correlations were found between cortisol measures collected at Times 4 through 7 for this particular variable. When the cortisol average for

measurement Times 1 through 7 was correlated with participants' report of asthma or bronchitis or emphysema, no significant relationship was found [$r(222) = .12$, $p > .05$]. Thus the report of asthma or bronchitis or emphysema was not considered as a covariate in the analyses.

Participants were not asked questions regarding adrenal insufficiency or Addison's disease, or the diagnosis and treatment of Cushing's disease. The proportion of individuals with a diagnosis of Cushing's or Addison's disease in the American population is very small. The prevalence of Cushing's disease is estimated to be about 39 individuals per million (Jane & Laws, 2006) and the prevalence of Addison's disease is estimated to be 120 per million in Western countries (Ten, New, & Maclaren, 2001). Thus, it is unlikely that lack of information regarding these diagnoses was problematic for the current analyses.

Conditions, medications, and activities that can influence the HPA axis or concentration of salivary cortisol were also considered as covariates. These included a) pregnancy, b) women who were in the follicular phase of their menstrual cycle, c) women taking birth control, d) women taking hormone replacement therapy, e) smoking cigarettes, and f) drinking caffeinated beverages on the day of in-home testing. Data were insufficient to determine if there was a relationship between cortisol and menstrual cycle phase, since the women who reported the date of their last menstrual period ($N = 37$) were postmenopausal. No significant relationships were found between the seven cortisol measures and the following variables 1) pregnancy status, 2) women taking birth control,

3) women taking hormone replacement therapy, and 4) cigarette smoking status. The number of caffeinated beverages drank on the day of in-home testing demonstrated a significant negative relationship for cortisol measure 1 [$r(219) = -.14, p < .05$]. Correlation of this measure with the cortisol average, however, produced no significant association [$r(223) = -.10, p > .05$], and thus it was excluded as a potential covariate.

A significant negative association was found between cigarette smoking status and mean heart rate [$r_{pb}(82) = -.34, p < .05$], mean skin conductance [$r_{pb}(82) = -.37, p < .05$], and the standard deviation of skin conductance [$r_{pb}(82) = -.39, p < .05$]. Participants who reported being a smoker had higher mean heart rate and skin conductance and standard deviation of skin conductance levels than those who indicated not being a smoker. Thus cigarette smoking was utilized as a covariate in analyses examining the potential moderation effect of sympathetic arousal upon working memory performance. The number of caffeinated beverages drank on the day of in-home testing did not correlate significantly with the sympathetic arousal measures and consequently was not considered further as a covariate.

Participants did not provide information regarding whether they were taking β-adrenergic receptor antagonists, a medication that can reduce the sympathetic response. Since this is one type of medication that is used to treat high blood pressure (Fisher & Williams, 2005), hypertensive status, which was measured by participants recollection of the presence of high blood pressure in the last twelve months, was correlated with cortisol average, heart rate (mean and standard deviation), and skin conductance (mean

and standard deviation). There were no significant correlations between hypertensive status and any of these measures, except a trend towards significance with mean skin conductance [$r_{pb}(85) = .20$, $p = .06$]. This suggested that those reporting normal blood pressure may have had higher mean skin conductance levels than those indicating a history of high blood pressure. Because of this, hypertensive status was entered as a covariate in all analyses exploring the potential moderation effect of sympathetic arousal upon working memory.

Characteristics of excluded participants

Participants with a history of stroke, diabetes, neurological disorders, or who did not report English as their native language were excluded from the analyses. This resulted in a reduction of 31 participants from the dataset. To determine if there were any significant differences between the excluded and included participants, analyses were conducted according to proposed research questions and hypotheses, since the sample size varied depending upon the research question or hypothesis being addressed.

Research questions and hypotheses 1-4. To determine if cortisol values and time of cortisol assessment differed between the excluded and included participants, a between subjects multivariate analysis of variance (MANOVA) was conducted with all cortisol levels and times of collection entered as dependent variables, except the sixth cortisol level and related time of collection. These latter variables were excluded due to their smaller sample size ($N = 100$ for level, and $N = 104$ for time) compared to the average sample size of the other cortisol levels ($N = 247$) and times of collection ($N = 243$). The

overall MANOVA indicated no significant main effect for the excluded group when all cortisol values and times of collection (except number 6) were entered as dependent variables, $F(1, 214) = 1.43$, $p > .05$, $\eta^2 = .08$. Assumptions of homogeneity of variance-covariance matrices across design cells were maintained as indicated by a non-significant result for the Box's M test, F (5651.986) = 1.23, $p > .05$. An independent sample's t-test revealed no significant differences between the excluded and included groups for the sixth cortisol level, $t(98) = .30$, $p > .05$, and related time of collection, $t(102) = -.43$, $p > .05$.

To determine if the between person measures of personality, age and education varied between the excluded and included groups, a between subjects MANOVA was conducted. A significant main effect indicated that there was a significant difference between the two groups, $F(1, 235) = 2.38$, $p < .05$, $\eta^2 = .05$. Follow-up univariate analysis of variance (ANOVA) tests revealed that this effect was due to differences between the two groups in years of education, $F(1, 239) = 7.47$, $p < .05$, $\eta^2 = .03$. The excluded group on average had fewer years of education ($M = 14.02$, $SD = 3.07$) than the included group ($M = 15.9$, $SD = 3.27$). Assumptions of homogeneity of variance-covariance matrices across design cells was maintained as indicated by a non-significant result for the Box's M test, $F(6198.809) = .98$, $p > .05$. Levene's test also indicated homogeneity of error variances for years of education, $F(1,239) = 0.1$, $p > .05$. There were no significant differences at the univariate level between the two groups in the three measures of personality (i.e. neuroticism, extraversion, and openness) and age.

Chi-Square contingency tables were performed to determine if the excluded and included groups differed in number of male and female subjects. Results indicated that there were no significant differences between the numbers of males and females in the two groups, $X^2 = .36$, $p > .05$.

Research question and hypothesis 5 and research question 6. To determine if the excluded participants differed from the rest of the participants for variables addressing research question and hypothesis 5, three separate MANOVAs were conducted. The first overall MANOVA indicated no significant main effect for the two groups (included versus excluded) when the following were entered as dependent variables: logical memory immediate, age, years of education, cortisol change from time 1 to 4, cortisol level at time 4, and time of collection for cortisol sample number 4, $F(1, 119) = 1.72$, $p > .05$, $\eta^2 = .08$. The Box's M test was not significant, $F(1579.551) = .93$, $p > .05$. The second overall MANOVA also found no significant main effect for the two groups when the following were entered as dependent variables: logical memory delayed, age, years of education, cortisol change from time 1 to 5, cortisol level at time 5, and time of collection for cortisol sample number 5, $F(1, 117) = 2.00$, $p > .05$, $\eta^2 = .09$. The Box's M test was also not significant for this analysis, $F(1868.231) = 0.73$, $p > .05$.

The third overall MANOVA, however, did reveal a significant main effect for the two groups when the following were entered as dependent variables: letter-number sequencing, age, years of education, cortisol change from time 1 to 5, cortisol level at time 5, and time of collection for cortisol sample number 5, $F(1, 223) = 2.76$, $p < .05$, η^2

= .07. Follow-up ANOVAs revealed that this effect was due to differences between the two groups in years of education, $F(1, 228) = 8.08$, $p < .05$, $\eta^2 = .03$, and performance on the letter number sequencing test, $F(1, 228) = 10.68$, $p < .05$, $\eta^2 = .05$. A trend towards significance was also found for age, $F(1, 228) = 3.94$, $p = .05$, $\eta^2 = .02$. The excluded group on average had fewer years of education ($M = 13.98$, $SD = 2.97$) than the included group ($M = 15.90$, $SD = 3.27$) and performed less well on letter number sequencing ($M = 8.65$, $SD = 2.95$) than the included group ($M = 10.57$, $SD = 2.80$). Assumptions of homogeneity of variance-covariance matrices across design cells was maintained as indicated by a non-significant result for the Box's M test, $F(6927.141) = 1.05$, $p > .05$. Levene's test also indicated homogeneity of error variances for years of education, $F(1,228) = .01$, $p > .05$, and for letter number sequencing, $F(1,228) = .03$, $p > .05$. There were no significant differences at the univariate level between the two groups in the cortisol measures and time of collection.

To determine if the excluded participants differed from the rest of the participants for variables addressing research question 6, two separate MANOVAs were conducted. The first overall MANOVA indicated no significant main effect for the two groups when the following were entered as dependent variables: letter-number sequencing, heart rate mean, heart rate standard deviation, age, years of education, cortisol change from time 1 to 5, cortisol level at time 5, and time of collection for cortisol sample number 5, $F(1, 79) = 1.55$, $p > .05$, $\eta^2 = .14$. The Box's M test was not significant, $F(1273.204) = 0.73$, $p > .05$. The second overall MANOVA also demonstrated no significant main effects for the

two groups when skin conductance mean and standard deviation were substituted for heart rate mean and standard deviation, with the remaining dependent variables being the same as those entered for the first MANOVA, $F(1, 78) = 1.87$, $p > .05$, $\eta^2 = .16$. These results should be interpreted cautiously since the Box's M test was significant for this analysis, $F(1275.073) = 1.67$, $p < .05$.

Chi-Square contingency tables were performed to determine if the excluded and included groups differed in reports of hypertension and cigarette smoking status. Results indicated that there were no significant differences between the two groups in recollections of high blood pressure in the last twelve months, $X^2 = .87$, $p > .05$, and in number of cigarette smokers, $X^2 = .42$, $p > .05$.

Analyses addressing research questions and hypotheses 1-4

Exclusion of outliers. Any value that was greater than the mean level plus five times the standard deviation was omitted from analyses. Two single cortisol values were excluded (i.e. 38.89 nmol/L at time 3 and 100.01 nmol/L at time 7); the remaining cortisol data from these two participants were included. One participant who had cortisol values ranging from 49.12 to 106.76 nmol/L was excluded, since all cortisol data were outliers.

Demographic characteristics. The average age of the participants was 59.51 years ($SD = 12.65$; $N = 208$). Fifty-seven percent of the participants were female and the sample was highly educated with 15.98 average years of education ($SD = 3.30$).

Descriptive statistics. Descriptive statistics for the study variables can be found in Tables 1, 2, and 3. There was a slight increase in the average change in cortisol from baseline to trial 2 and from trial 2 to trial 3 (see Table 2). Following trial 3, the average trial to trial change in cortisol dropped slightly, and the standard deviation for the trial to trial change in cortisol ranged from 1.61 to 3.19. Only 21 participants out of 201 (i.e. 10.4%) experienced a 2.5 nmol/L increase in cortisol from baseline to the last cortisol assessment (trial 7), an increase considered to be a definitive cortisol response to a stressor (Kirschbaum et al., 1996). On average, participants took 95.42 minutes to complete the in-home testing (see Table 3), which is adequate time for an increased cortisol response and recovery to normal levels (de Kloet, Joels, & Holsboer, 2005).

Covariates. To control for the diurnal variation of cortisol, the time of day for the baseline cortisol measure was entered as a covariate. One hundred sixteen participants began the in-home testing procedures prior to 12:00 noon, while 92 started testing after this time.

Because previous research indicated that age and education were related to cortisol reactivity associated with cognitive testing (Neupert et al., 2006), these measures were controlled when they were not analyzed as independent variables of interest. There was a significant positive correlation between age and the average cortisol level [$r(206) = .24$, $p < .05$], thus older adults had higher average cortisol levels than younger adults. No significant relationship was found between years of education and average cortisol level, however.

Because there was a significant relationship between gender and average cortisol [r_{pb} (206) = -0.34, $p < .05$], gender was entered as a covariate when it was not tested as an independent variable. Women had a lower cortisol average than men. Longitudinal status was also significantly correlated with the average cortisol level. Longitudinal participants (N = 79) had a higher cortisol average than non-longitudinal participants (N = 129) [r_{pb}(206) = 0.14, $p < .05$]; thus, the variable indicating longitudinal status was used as a covariate in all analyses.

Multilevel models. Multilevel modeling was used to examine within person trajectories of cortisol associated with completing a cognitive testing battery (see Appendix A). Specifically tested was whether changes in cortisol over time were dependent upon person-level traits of extraversion, openness, or neuroticism, or an interaction of the following characteristics:

a) age and education

b) personality (i.e. extraversion, openness, or neuroticism) and gender

c) personality (i.e. extraversion, openness, or neuroticism) and age

d) personality (i.e. extraversion, openness, or neurotcism) and education

Effect of personality upon the within person cortisol slope was tested in models analyzing interaction effects. Since significant interactions only occurred between age and education and age and extraversion, the multilevel models used to obtain these results are described in detail on the next page:

Model for Age X Extraversion:

Level 1: $Cortisol_{it} = \beta_{0it} + \beta_{1it}\,(Trial) + r_{it}$

Level 2: $\beta_{0i} = \gamma_{00} + \gamma_{01}(\text{Time of day}) + \gamma_{02}(\text{Gender}) + \gamma_{03}(\text{Longitudinal status}) + \gamma_{04}\,(\text{Education}) + \gamma_{05}(\text{Age}) + \gamma_{06}(\text{Extraversion}) + \gamma_{07}(\text{ Age X Extraversion}) + u_{0i}$

$\beta_{1i} = \gamma_{10} + \gamma_{11}(\text{Age}) + \gamma_{12}(\text{Extraversion}) + \gamma_{13}(\text{Age X Extraversion})$

Model for Age X Education:

Level 1: $Cortisol_{it} = \beta_{0it} + \beta_{1it}(Trial) + r_{it}$

Level 2: $\beta_{0i} = \gamma_{00} + \gamma_{01}(\text{Time of day}) + \gamma_{02}(\text{Gender}) + \gamma_{03}(\text{Longitudinal status}) + \gamma_{04}\,(\text{Age}) + \gamma_{05}(\text{Education}) + \gamma_{06}(\text{Age X Education}) + u_{0i}$

$\beta_{1i} = \gamma_{10} + \gamma_{11}(\text{Age}) + \gamma_{12}(\text{Education}) + \gamma_{13}(\text{Age X Education})$

In the Level 1 equation, the dependent variable represented the cortisol level for each person at each time of collection. The intercept (β_{0it}) was defined as the expected level of cortisol for each person at baseline. The reactivity slope (β_{1it}) was the expected change in cortisol for each person associated with subsequent trials. The error term (r_{it}) represented how much each person's cortisol level varied over time and was a unique effect for each individual.

In the Level 2 equations, the dependent variable (β_{0i}) indicated the average cortisol level for each person across all trials. Each variable was centered around the grand sample mean, so γ_{00} was the mean cortisol level for a person of average age (i.e. 59 years) who was tested at the mean of the time of day of testing. Gamma coefficients (e.g. $\gamma_{01}, \gamma_{02}, \gamma_{03}, \gamma_{04}$ etc.) represented the effects of person-level characteristics such as time of

day of testing, gender, longitudinal status, and age upon average cortisol level. The intercept (γ_{10}) in the second Level 2 equation indicated the average change in cortisol over time for the whole sample controlling for age and extraversion (or age and education for Age X Education Model). The rest of the gamma coefficients (i.e. γ_{11}, γ_{12}, and γ_{13}) indicated the effects of each variable and interaction term upon the cortisol slope between trials for each person (β_{1i}).

Results from the fully unconditional model, a model that did not have any predictors, examined cortisol level and change over time for each person and indicated that 33% ($\sigma^2 = 8.74$, $z = 24.28$, $p < .05$) of the variance in cortisol was within-people while 67% ($\tau_{00} = 18.12$, $z = 9.78$, $p < .05$) was between people.

Model for Age X Extraversion. The average cortisol level for older adults was higher than for younger adults (γ_{05}); however, age did not predict within-person changes in cortisol over time (γ_{11}), whereas extraversion (γ_{12}) did (see Table 4 for unstandardized coefficients). Those scoring high in extraversion had a greater cortisol response over the cognitive testing occasions than those scoring low in extraversion. This effect was qualified by a significant Age X Extraversion interaction (γ_{13}). The average cortisol level was also significantly associated with two covariates: time of day of testing (γ_{01}) and gender (γ_{02}). Those tested in the morning had a higher cortisol level than those tested in the afternoon, and women had lower average cortisol levels than men (see Table 4). This model accounted for 12% of the within-person and 27% of the between-person variance

in cortisol, which was calculated by the equation for pseudo-R^2 from Raudenbush and Bryk (2002).

To decompose the Age X Extraversion interaction, slopes based upon the predicted age and extraversion differences in cortisol reactivity were plotted. Age was determined using the average age (59 years) for the middle, one standard deviation below the mean (47 years) for the younger, and one standard deviation above the mean (72 years) for the older adults. Low extraversion was indicated by one standard deviation below the mean (2.65) and high extraversion by one standard deviation above the mean (3.67).

Predicted slopes revealed that younger and middle-aged adults scoring high in extraversion experienced less of a decline in cortisol over time than same aged adults scoring low in extraversion (see Figure 2). The simple slope for younger adults scoring high in extraversion was not significant (simple slope = -0.14, $p > .05$), suggesting that these participants experienced little change in cortisol over time. The simple slope for middle-aged adults scoring high in extraversion was significant (simple slope = -0.24, $p < .05$) and smaller than older adults scoring high in extraversion (simple slope = -0.33, $p < 05$). Within-person change in cortisol appeared to be the same for older adults scoring high and low in extraversion. Thus, extraversion scores did not appear to make a difference in older adults' cortisol response during the cognitive challenge, while it may have played a role in the cortisol response of middle-aged and younger adult participants.

Model for Age X Education. The average cortisol level for older adults was higher than for younger adults (γ_{04}); however, neither age (γ_{11}) nor education (γ_{12}) alone predicted within-person changes in cortisol over time (see Table 5 for unstandardized coefficients). There was an Age x Education interaction (γ_{06}) predicting cortisol level. Time of day of testing (γ_{01}) and gender (γ_{02}) were also significantly related to cortisol level, just as in the model for Age X Extraversion. Results revealed that within-person changes in cortisol also depended upon an Age X Education interaction (γ_{13}). This model accounted for 5% of the within-person and 29% of the between-person variance in cortisol.

In order to interpret the Age X Education interaction, two additional models were conducted as indicated by Neupert et al. (2006). One model examined those with low education (operationalized as two years of college or less) and another examined those with high education (operationalized as college degree or higher). Slopes for within-person cortisol change were then plotted using the average age (59 years) for the middle, one standard deviation below the mean (47 years) for the younger, and one standard deviation above the mean (72 years) for the older adults. Younger adults with low education appeared to experience a steeper decline in cortisol over time (simple slope = -0.48, $p < .05$) compared to middle-aged (simple slope = -0.36, $p < .05$) and older adults (simple slope = -0.23, $p < .05$) with similar educational backgrounds (see Figure 3). In contrast, older adults with higher education seemed to experience the greatest decline in cortisol over time (simple slope = -0.47, $p < .05$) compared to middle-aged (simple slope

= -0.36, $p < .05$) and younger adults (simple slope = -0.26, $p < .05$) with similar educational backgrounds.

Remaining models. As mentioned previously, openness and neuroticism were not significant predictors of the within-person cortisol slope, and neither personality characteristic interacted with age to predict cortisol reactivity (see Tables 6 and 7). Both models explained 11% of the within-person and 28% of the between-person variance in cortisol. No significant interactions were found for personality and education or personality and gender (see Tables 8-13). Congruent with previous models, time of day of testing and gender remained significantly related to cortisol level. Age also continued to be associated with cortisol level, except in the Age X Neuroticism model. Extraversion also continued to be a significant predictor of the cortisol reactivity slope in the Extraversion X Education and Extraversion X Gender models.

The model for Extraversion X Education accounted for 12% of the within-person and 25% of the between person variance in cortisol, while the model for Extraversion X Gender accounted for 12% of the within-person and 28% of the between-person variance. Models for Openness X Gender, Neuroticism X Education, and Neuroticism X Gender accounted for 11% of the within-person and 28% of the between-person variance in cortisol. The model for Openness X Education accounted for the same degree of within-person variance as these previous models and 27% of the between-person variance in cortisol.

Analyses addressing research question and hypothesis 5

Demographic characteristics. Since some of the participants did not complete all of the cognitive tests, demographic characteristics varied slightly depending upon the cognitive test employed as the dependent variable. For logical memory immediate (N = 113), 53% of the participants were male, while for logical memory delayed (N = 110), 55% were male. For letter-number sequencing (N = 204), 55% were female. The average age of the participants was 59.04 (SD = 12.52) and the mean years of education was 16.18 (SD = 3.62) when logical memory immediate or delayed was the dependent variable. Similar descriptive statistics for age and years of education were found when analyses employed letter-number sequencing as the dependent variable (see Table 14).

Descriptive statistics. Descriptive statistics for the study variables can be found in Tables 14 and 15. Cortisol change from baseline to Trial 4 and cortisol level at Trial 4 were analyzed to determine whether cortisol increase was associated with performance decrements in logical memory immediate. Cortisol collection at Trial 4 occurred after participants had completed the Spielberger State exam, potentially 8 cognitive tests, and an interviewer report (see Appendix A). Following the collection of Trial 4 cortisol, participants were given a short break, and then were asked to complete four more cognitive tests (i.e. backwards counting, dual task, logical memory immediate, and task switching) before the collection of trial 5 cortisol. The average change from baseline to Trial 4 was -0.26 (SD = 5.95). Twenty-four participants out of 113 (i.e. 21%) experienced a 2.5 nmol/L increase in cortisol from baseline to Trial 4, an elevation which has been

defined as a definitive cortisol response to a stressor (Kirschbaum et al., 1996). The average cortisol level at Trial 4 was 7.82 (*SD* = 6.31).

Cortisol change from baseline to Trial 5 and cortisol level at Trial 5 were analyzed to determine whether cortisol increase was related to performance decrements in logical memory delayed and letter-number sequencing. Since the sample sizes for logical memory delayed and letter-number sequencing were different, cortisol change and level will be discussed according to cognitive test analyzed as the dependent variable.

The average cortisol change from baseline to Trial 5 for logical memory delayed was -1.32 (*SD* = 5.67). Eighteen participants out of 110 (i.e. 16%) experienced a 2.5 nmol/L increase in cortisol from baseline to Trial 5, which is considered an indication of reactivity (Kirschbaum et al., 1996). The average cortisol level at Trial 5 was 6.87 (*SD* = 5.30).

The average cortisol change from baseline to Trial 5 for letter-number sequencing was -1.19 (*SD* = 5.05). Twenty-nine out of 204 (i.e. 14%) participants experienced a 2.5 nmol/L increase in cortisol from baseline to Trial 5. Fourteen out of these 29 participants had an increase in the morning, while 15 out of these 29 experienced a cortisol increase in the afternoon. The average cortisol level at Trial 5 for letter-number sequencing was 6.35 (*SD* = 4.61).

Covariates. All analyses controlled for years of education. Time of day of testing was controlled unless considered as a specific moderator. Trial 4 cortisol levels were collected in 47% of the participants prior to 12 noon. Trial 5 cortisol levels for logical

memory delayed and letter-number sequencing were collected in 44% and 43% of the participants, respectively, prior to 12 noon. Age was also controlled unless considered as a specific moderator.

Moderated regression. Moderated regression was employed to determine whether elevated cortisol, associated with completed cognitive tests, impaired performance on subsequent assessments of declarative and working memory, and whether cortisol associated memory impairment was dependent upon the specific moderators of age and time of day of testing. To examine the effects of cortisol upon cognitive testing, the level of cortisol and the change in cortisol from baseline were tested in separate analyses. There were no main effects for cortisol level or cortisol change from baseline when logical memory immediate, logical memory delayed, and letter-number sequencing were entered as dependent variables. Thus, neither cortisol level nor cortisol change alone had independent effects upon declarative or working memory (see Tables 16, 17 and 18). Significant main effects were found for age and education in each of these analyses indicating that younger adults and those with increased years of education performed better than older adults and those with decreased years of education. Time of day was also a significant main effect when logical memory immediate was a dependent variable and cortisol change (not level) was entered as a predictor. Those participants who were tested earlier performed better than those who were tested later.

When age and cortisol change (or level) were examined as moderators of declarative and working memory performance, a significant Age X Cortisol Change

interaction was found for letter-number sequencing. This 2-way interaction was qualified by a significant 3-way interaction of Age X Cortisol Change X Time of Day (see Table 18). These same effects were not found for logical memory immediate or delayed. Interactions involving cortisol level were not significant for any of the cognitive tests. Two-way interactions involving time of day (e.g. Cortisol Change X Time of Day, or Age X Time of Day) were also not significant for any of the cognitive tests.

To decompose the 3-way interaction, simple slope analyses were conducted to determine how predicted letter-number sequencing scores varied according to age, cortisol response, and time of day of testing. In each of these analyses, age was entered using the average age (59 years) for the middle, one standard deviation below the mean (47 years) for the younger, and one standard deviation above the mean (72 years) for the older adults. One standard deviation below the mean change in cortisol from baseline to Trial 5 represented cortisol non-responders (-6.24 nmol/L), while one standard deviation above that mean indicated cortisol responders (3.86 nmol/L). Time in the AM represented one standard deviation below the mean for time at Trial 5 (637.72 minutes or 10:38 AM) and time in the PM represented one standard deviation above the mean for the same time point (1001.32 minutes or 4:41 PM). Predicted letter-number sequencing scores were plotted as a function of this data while controlling for the effects of education. Three column graphs were plotted to explore the Age X Cortisol Change X Time of Day interaction. The first graph compared the predicted performance of letter-number sequencing scores of cortisol responders and non-responders, plotted as a function of age

and time of day. The second compared the predicted scores in the morning and afternoon, plotted as a function of age and cortisol response, and the third compared the predicted working memory performance of young, middle, and older adults, plotted as a function of cortisol response and time of day.

When comparing predicted letter-number sequencing scores of cortisol responders and non-responders, simple slopes indicated that scores of younger, middle-aged, and older adult cortisol responders were not significantly different than same-aged non-responders in the morning (younger: simple slope = 0.01, $p > .05$; middle: simple slope = -0.05, $p > .05$; older: simple slope = -0.11, $p > .05$). In the afternoon, however, the predicted working memory performance for older adult cortisol non-responders was lower than the predicted score for older adult cortisol responders (simple slope = 0.38, $p < .05$) (see Figure 4). This same result was not found for younger and middle aged participants tested at this time. Simple slopes revealed that the predicted working memory of younger and middle-aged cortisol responders did not differ from same aged non-responders in the afternoon (younger: simple slope = -0.12, $p > .05$; middle: simple slope = 0.13, $p > .05$).

The predicted scores of cortisol responders tested in the morning were compared to the scores of responders tested in the afternoon. The same morning and afternoon comparisons were also made for cortisol non-responders. Simple slopes revealed predicted performance differences for older adults only (see Figure 5). Older adult cortisol non-responders tested in the morning had better scores than same-aged cortisol

non-responders tested in the afternoon (simple slope = - 0.007, $p < .05$). Conversely, older adult cortisol responders tested in the morning had lower test scores than same-aged cortisol responders tested in the afternoon (simple slope = 0.006, $p < .05$).

Finally, age differences in predicted working memory performance was explored according to cortisol response and time of day of testing. Simple slope analyses indicated that there were no performance differences between the young, middle, and older adult cortisol responders tested in the afternoon (simple slope = 0.01, $p > .05$) (see Figure 6). Letter-number sequencing scores appeared to vary by age for the cortisol responders tested in the morning (simple slope = -0.12, $p < .05$) and for the cortisol non-responders tested at both times of the day (AM: simple slope = -0.07, $p < .05$; PM: simple slope = -0.19, $p < .05$). Younger adult cortisol non-responders seemed to perform better than middle-aged and older adult cortisol non-responders in the morning and afternoon. Younger adult cortisol responders also had better predicted scores on letter-number sequencing than middle-aged and older adult cortisol responders tested in the morning.

Since there appeared to be no age differences in the letter number sequencing performance of cortisol responders tested in the afternoon and since cortisol response has been associated with increased education in middle aged and older adults (Neupert et al., 2006), it seemed important to explore the degree that education may have contributed to the increase in cortisol from baseline to Trial 5 at this time. Another moderated regression analysis was performed with cortisol change from baseline to Trial 5 tested as a dependent variable, and education, age, and time of day entered as moderators.

Controlling for gender and longitudinal status, a significant main effect was found for education and this effect was qualified by a significant Age X Education X Time of Day interaction (see Table 19).

To decompose this interaction, predicted change in cortisol from baseline to trial 5 was plotted as a function of education, time of day, and age, controlling for gender and longitudinal status (see Figure 7). Columns for age were graphed using the average age (59 years) for the middle, one standard deviation below the mean (47 years) for the younger, and one standard deviation above the mean (72 years) for the older adults. One standard deviation below the mean education in years represented low education (12.72 years) and one standard deviation above the mean education in years indicated high education (19.18 years). Time in the AM represented one standard deviation below the mean for time at Trial 5 (637.72 minutes or 10:38 AM) and time in the PM was one standard deviation above that same mean (1001.32 minutes or 4:41 PM).

Examination of this graph suggested that older adults with high education appeared to have the greatest cortisol increase in the afternoon. Simple slopes analysis revealed that the difference in cortisol change between older adults with high education in the morning and same aged and similarly educated adults in the afternoon was close to significance (simple slope = 0.011, $p = 0.06$). None of the other simple slopes for younger and middle-aged adults, and older adults with low education were significant.

Analyses addressing research question 6

Exclusion of outliers. Any value greater than the mean level plus five times the standard deviation was omitted from analyses. One skin conductance level that was 55.27 micromhos met this exclusion criterion and was deleted.

Demographic characteristics. Indicators of sympathetic arousal (e.g. heart rate and skin conductance) were measured on the longitudinal participants only. Fifty-five percent of the participants were male and the total sample size was 75 for analyses employing skin conductance measures and 76 for analyses utilizing heart rate measures. The average age of the participants was 59.55 (*SD* = 12.34) and the mean years of education was 16.26 (*SD* = 3.82).

Descriptive statistics. Descriptive statistics can be found in Table 20. In the overall sample, there was variability in skin conductance (*M* = 3.90, *SD* = 2.17) and heart rate (*M* = 69.68, *SD* = 9.57). Fifteen out of 75 participants (i.e. 20%) had skin conductance values greater than 5 micromhos, a value indicating fairly high conductivity and arousal (NeuroDyne Medical Corporation, 1995). Thirteen out of 75 participants (i.e. 17%) experienced a cortisol increase greater than 2.5 nmol/L.

Covariates. All analyses controlled for years of education, smoking status, the time of day of testing, and hypertensive status, which was utilized as a proxy for beta-adrenergic receptor blockers.

Measures of sympathetic arousal. Measures of sympathetic arousal included heart rate and skin conductance level mean and standard deviation. Since baseline data for

these measures were not available, the standard deviation was analyzed to provide another means for examining sympathetic arousal. It was assumed that the standard deviation would be increased if the testing situation provoked a sympathetic response; however, it is also possible that a large standard deviation could reflect a change from a high score to a low score. To test this, the mean level and standard deviation for each measure was correlated to determine whether there was a positive association between increased level and variability. Results suggested that those with high mean skin conductance level had high standard deviations of skin conductance [r (73) = .57, $p <$ .05], while there was no relationship between mean heart rate and standard deviation [r (73) = -.08, $p >$ .05].

Moderated regression. Moderated regression was employed to determine whether working memory performance, as measured by letter-number sequencing, was dependent upon cortisol elevations, age and sympathetic arousal. Cortisol level and cortisol change were both tested independently. No specific main or interaction effects for heart rate were found (see Tables 21 and 22). Significant main effects for age and education were found in all analyses investigating heart rate as a predictor. Older adults and those with less education had lower letter-number sequencing scores than younger adults and those with higher education.

There was a main effect for skin conductance standard deviation when cortisol level (and not cortisol change) was entered as a predictor (see Table 23). Those participants who had increased skin conductance variability demonstrated better scores

on letter-number-sequencing than those who had decreased skin conductance variability. Just as with the analyses employing heart rate as a predictor, age and education remained significant predictors in analyses where skin conductance was tested with cortisol level. Additionally, in the analysis where skin conductance standard deviation and cortisol level was tested, there was a significant main effect for smoking status. Smokers had higher working memory test scores than non-smokers. There were no interaction effects between skin conductance, age, and cortisol level.

When cortisol change was entered as a predictor in two separate analyses where skin conductance mean was a predictor in one analysis and skin conductance standard deviation was a predictor in the other, significant Cortisol Change X Skin Conductance Mean and Cortisol Change X Skin Conductance Standard Deviation interactions were found (see Table 24). Education was a significant main effect in each analysis. Age was only a significant predictor, however, in the analysis where cortisol change and skin conductance mean were entered as predictors. Age ceased to be a main effect in the analysis where cortisol change and skin conductance standard deviation were employed as predictors.

To decompose these two interactions, separate moderated regression analyses with Cortisol Change X Skin Conductance Mean and Cortisol Change X Skin Conductance Standard Deviation were analyzed controlling for age, education, time of day of testing, smoking status, and hypertensive status. Both of these 2-way interactions

remained significant (Cortisol Change X Skin Conductance mean: $\beta = 0.24$, $t = 2.20$, $p < .05$; Cortisol Change X Skin Conductance standard deviation: $\beta = 0.35$, $t = 2.92$, $p < .05$).

To decompose the Cortisol Change X Skin Conductance Mean interaction, columns were plotted based upon the predicted performance on letter-number sequencing as a function of cortisol response and mean skin conductance level, controlling for age, education, time of day of testing, smoking status, and hypertensive status (see Figure 8). Columns for mean skin conductance were graphed using one standard deviation below the mean for the low level (1.7 micromhos), and one standard deviation above the mean for the high level (6.02 micromhos). One standard deviation below the mean change in cortisol from baseline to Trial 5 represented cortisol non-responders (-6.87 nmol/L) and one standard deviation above that mean indicated cortisol responders (4.29 nmol/L). Simple slope analyses revealed that cortisol non-responders with a low skin conductance mean had similar predicted letter-number sequencing scores as cortisol responders with a similar skin conductance level (simple slope = -0.10, $p > .05$). Cortisol non-responders with a high skin conductance mean appeared to have lower scores than cortisol responders with a similar skin conductance level. Simple slope analysis, however, only suggested a trend in this direction (simple slope = 0.15, $p = .06$).

To decompose the Cortisol Change X Skin Conductance Standard Deviation interaction, columns were plotted based upon the predicted performance on letter-number sequencing as a function of cortisol response and standard deviation of skin conductance level, controlling for age, education, time of day of testing, smoking status, and

hypertensive status (see Figure 9). Columns for the standard deviation of skin conductance were graphed using one standard deviation below the skin conductance standard deviation mean for the low level (0), and one standard deviation above the skin conductance standard deviation mean for the high level (0.66). One standard deviation below the mean change in cortisol from baseline to Trial 5 represented cortisol non-responders (-6.87 nmol/L) and one standard deviation above that mean indicated cortisol responders (4.29 nmol/L). Simple slope analyses revealed that cortisol non-responders with a low skin conductance standard deviation had higher predicted letter-number sequencing scores than cortisol responders with a similar skin conductance level (simple slope = -0.15, $p < .05$). Conversely, cortisol non-responders with a high skin conductance standard deviation had a lower predicted working memory score than cortisol responders with a similar skin conductance level (simple slope = 0.21, $p < .05$).

Discussion

This study explored whether between-person differences, such as personality, age, education, and gender, were associated with differences in cortisol reactivity to a series of challenging cognitive tasks. Although it was anticipated that highly educated older and middle-aged adults would express an increased cortisol response over time compared to less educated participants, this result was not found. Of the personality variables tested, extraversion was associated with an increased cortisol response. This was further qualified by an Age X Extraversion interaction indicating that younger and middle-aged extraverted adults expressed the least change in cortisol throughout the cognitive testing

period compared to older adults and middle-aged and younger adults scoring low in extraversion.

This study was also interested in determining whether cortisol elevations prior to declarative and working memory testing were associated with less optimal performance on those tests and whether such effects were dependent upon age and the time of day of testing, as well as sympathetic arousal in the case of working memory. Contrary to expectations, cortisol elevations alone were not significantly associated with performance on either test. Working memory, however, was associated with an Age X Cortisol Change X Time of Day interaction. Results suggested that older adult cortisol responders performed better on letter-number sequencing in the afternoon than 1) same-aged participants who did not experience a cortisol increase in the afternoon and 2) same aged-participants who did experience a cortisol increase in the morning. Older adults who experienced a cortisol response in the afternoon tended to be highly educated.

Further analysis also suggested that working memory performance depended upon an interaction between cortisol response and sympathetic arousal, as measured by skin conductance level and standard deviation. Those participants with increased cortisol and sympathetic arousal appeared to perform as well on letter-number sequencing as those without a cortisol or sympathetic response. Age differences were not found in these analyses.

Research question and hypotheses 1-4

Contrary to hypothesis 1a and the report from Neupert et al. (2006), older and middle-aged adult participants who were highly educated did not experience a greater cortisol response - as demonstrated by a more positive cortisol slope - than the younger educated and less educated older and middle-aged participants. The predicted cortisol levels at baseline for older and middle-aged adults with more education did appear to be higher than same aged participants with less education and younger educated subjects. This higher level at baseline may have reflected anticipation of social evaluative threat (Dickerson & Kemeny, 2004). Unfortunately, measures indicating the degree that the cognitive testing situation was threatening to participants' social self-esteem were not investigated prior to commencement of testing or throughout the testing period in this study. The negative cortisol reactivity slope for highly educated middle aged and older adult participants suggested that if a possible perception of threat existed initially, it may not have persisted throughout the entire cognitive testing situation.

That the present study found a negative cortisol reactivity slope in contrast to the positive slope reported by Neupert et al. (2006) may be due to the differences in the number of cognitive tests employed by each study. Neupert et al. (2006) examined cortisol reactivity during and following 8 cognitive tests, while the present study investigated responses during and after 15 tests. The increased number of tasks may have allowed for the more educated older and middle-aged adult participants, who initially had high cortisol levels at Trial 1, to become more comfortable with the testing over time,

resulting in declining cortisol levels towards the end of the testing period. This assumption seems somewhat supported by the average trial to trial change in cortisol (see Table 2), which indicated that average cortisol levels did not begin to drop until after Trial 3 and the completion of 7 cognitive tests (see Appendix A). A multilevel model examining quadratic rather than linear changes in cortisol over time may have captured this effect. Quadratic analyses should be considered for future investigations of the relationship between cortisol reactivity and the completion of a long-series of cognitive tests.

Predictions regarding increased cortisol reactivity in participants scoring high in the trait of openness were also not found. There were no significant differences in cortisol response between those scoring high and low in openness. It is possible that the cognitive testing situation was not as strong of a stressor as the TSST employed by Oswald et al. (2004, 2006), and thus was too insensitive to detect differences in the physiological responses of these two groups. Cognitive testing was associated with a physiological stress response in a minority of the participants. Only 10.4% experienced what is considered by Kirschbaum et al. (1996) to be a definitive cortisol response to a stressor (i.e. an increase of 2.5 nmol/L) from Trial 1 to Trial 7.

There were also no significant differences in the cortisol responses of those scoring high and low in trait neuroticism. This was consistent with Oswald et al. (2004), but not with Oswald et al. (2006), where women who scored high in neuroticism experienced a blunted cortisol response to the TSST. Again, the stress-inducing nature of

the cognitive testing situation in the present study may have been too mild to isolate differences in the physiological stress responses of men and women scoring high and low in neuroticism.

Participants scoring high in extraversion, however, did experience a greater cortisol response over time than those scoring low in extraversion. This result was qualified by an Age X Extraversion interaction revealing that middle aged and younger adult participants scoring high in the trait of extraversion appeared to have less negative cortisol slopes over time than same-aged participants scoring low in this personality dimension. There were no significant differences between predicted cortisol levels at Trial 1 and Trial 7 for younger extraverts, indicating that there was perhaps little, if any, decline in cortisol over time for these participants. Although there was a difference in predicted cortisol levels at Trial 1 and Trial 7 for middle-aged extraverts, with Trial 7 level being lower than the Trial 1 level, their cortisol slope was less negative than the slope for middle-aged introverts. Middle-aged and younger adult introverts had more sharply negative cortisol reactivity slopes, suggesting a greater decline in cortisol over time.

These findings were somewhat similar to those found by Oswald et al. (2004) who reported that extraverts had a greater cortisol response to the TSST than introverts. In the present study, however, there were no significant differences between extraverts and introverts in the level of cortisol, and because the extraverts did not express positive cortisol slopes, it would be difficult to conclude that these participants experienced a

physiological stress response during the cognitive testing. Differences occurred in the degree of cortisol reactivity over time, which was not measured by Oswald et al.

More research would be needed to explain why the cortisol reactivity slopes of middle-aged and younger adult extraverts did not decline as much as same-aged introverts. Although speculative, it is possible that extraverts due to their more lively, outgoing, and active personalities may have been more engaged than the introverts in the cognitive testing procedure. This engagement may have resulted in a mild physiological reactivity or arousal that caused little decline in cortisol throughout the testing period. Future work would need to include measures of interest and arousal to test this assumption. To further understand why extraversion seems associated with differences in cortisol reactivity compared to introversion, a closer examination of the association of different dimensions of extraversion to cortisol reactivity would be required.

It is also possible that the diurnal cortisol rhythm of extraverts is different than introverts, and that the present findings are reflective of that difference and not a response to the cognitive testing situation. LeBlanc and Ducharme (2005) found that the cortisol levels of extraverts were higher than introverts in the early afternoon, suggesting differences in daily cortisol rhythm. To investigate the possibility that the findings in the present study are due to differences in cortisol patterns between the two groups, future testing would need to compare the cortisol reactivity of extraverts and introverts at rest with reactivity slopes during a cognitive testing occasion similar to the one employed in this study.

Unlike the middle-aged and younger adult extraverts, older adult extraverts appeared to have a similar cortisol reactivity slope as same aged introverts. Both older adult groups had higher cortisol levels than the younger aged groups, and this initial high level may have had a more pronounced influence on cortisol reactivity over time than the degree of extraversion for these older adult participants.

Increased cortisol reactivity associated with extraversion was not dependent upon gender as reported by Oswald et al. (2006), who found that only extraverted men had an increased cortisol response to the TSST. Oswald et al. studied women in the follicular phase of their menstrual cycle, when cortisol responses to psychosocial stressors tend to be lower than those for men (Kajantie & Phillips, 2006). In all of the multilevel models analyzed in the present study where gender was entered as a predictor, there were no gender differences in cortisol reactivity during the cognitive testing procedure. Thus, the cortisol response of men and women were similar.

Lack of information regarding the number of women in the follicular and luteal phase of their menstrual cycle and lack of information regarding the number of post-menopausal women make interpretation of this finding difficult from a physiological perspective. Few women reported taking oral contraceptives (N = 3), which can dampen the cortisol response to a psychosocial stressor, thus this may have contributed to the similar cortisol responses of men and women. As discussed in previous sections, women in the luteal phase tend to have similar psychosocial stress-induced cortisol responses as

men (Kajantie & Phillips, 2006). How many of these women were in the sample, however, was not known.

On average, women had lower salivary cortisol levels than men during the cognitive testing procedure. Premenopausal women have been found to have slightly lower cortisol levels than men in the same age range (Van Cauter, Leproult, & Kupfer, 1996). A cross-sectional study has reported that cortisol levels did not change with age in a sample of women aged 17 to 71 years (Murakami et al., 1999). Previous research has also reported that older women had lower morning (or 8 A.M.) salivary cortisol than men (Brandtstädter, Baltes-Götz, Kirschbaum, & Hellhammer, 1991). In a sample of participants aged 35 to 65 years, morning cortisol levels declined linearly according to increased age in women but not in men. The occupational status of women was suggested as a possible moderator of this effect, with declines in cortisol associated with increased age being more pronounced in housewives than in employed women. Further studies are needed to explore why women may have lower levels of cortisol than men.

Exploratory analyses investigating whether cortisol reactivity was related to interactions between personality and education were not significant. Interactions between age and openness or neuroticism, and interactions between gender and openness or neuroticism were also not associated with cortisol reactivity. Further research exploring these issues may need to employ a psychosocial stressor, such as the TSST, to ensure a physiological stress response that is great enough, and thus sensitive enough, to detect

whether potential personality differences in cortisol reactivity are dependent upon gender and education, and age in the case of openness and neuroticism.

Research question and hypothesis 5

Contrary to hypothesis 5, participants who experienced the highest cortisol elevations measured by cortisol level or cortisol change did not perform more poorly on the declarative and working memory tasks than those demonstrating the least cortisol response. Lack of significant findings may have been due to an inadequate cortisol response by the sample as a whole. Average cortisol levels, which ranged from 6.35 to 7.82 nmol/L depending upon cognitive test evaluated as the dependent variable, were not as high as those reported in the Kirschbaum et al. (1996) and Oei et al. (2006) studies (17.7 nmol/L and 34.4 nmol/L, respectively). Average cortisol change declined on average from baseline, and thus was much lower in the present study compared to Kirschbaum et al., where 9 out of 13 participants experienced an increase ≥ 2.5 nmol/L. Participants in the stress condition of the Oei et al. study had an average increase of 13.6 nmol/L. For the cortisol responders in the Lupien et al. (1997) study, the cortisol level increased 6.1 nmol/L and this resulted in an average level of 17.7 nmol/L.

The cognitive challenge in the present study may have been a milder stressor than in these previous studies where the TSST, or a variation of it, was employed; thus the current study may not have had enough cortisol responders to find a significant main effect using moderated regression. Dichotomizing the cortisol change variable into cortisol responders (i.e. those with an increase ≥ 2.5 nmol/L from baseline) and cortisol

non-responders (i.e. those with a decrease < 2.5 nmol/L from baseline) may be an alternative method for increasing power to detect significant main effects for cortisol increase on declarative and working memory in future work.

Additionally, the absence of significant main effects for cortisol change or cortisol level when a test of declarative memory was the dependent variable may have resulted from the use of an assessment tool with emotional content. Previous research indicates that acute cortisol increase appears to hinder memory for neutral items rather than emotionally arousing ones (Tops et al., 2003, Wolf, 2008). Thus, the emotional content of the story to be recalled, which mentions a robbery and a family that had not eaten for two days, may have facilitated participant's recall capabilities despite slight to moderate cortisol elevations.

Although there were no main effects for cortisol level or cortisol change, performance on the working memory test was associated with an Age X Cortisol Change X Time of Day interaction. When controlling for education, older adult's performance on letter-number sequencing appeared to be dependent upon time of day of testing and cortisol change from baseline (Trial 1) to collection prior to the letter-number sequencing test (Trial 5). Significant effects were found primarily for older adults. Older adult cortisol responders tested in the afternoon had better predicted letter-number sequencing scores than same-aged cortisol non-responders tested at this same time. Older adult non-cortisol responders tested in the morning had higher predicted scores on the working memory task than same-aged cortisol non-responders tested in the afternoon. Conversely,

older adult cortisol responders tested in the morning had lower predicted letter-number sequencing scores than same-aged cortisol responders tested in the afternoon. These same associations were not found in the younger and middle aged participants. Finally, predicted working memory performance did not vary by age for cortisol responders who completed testing in the afternoon.

Although speculative, the association between cortisol and memory found primarily in the older adult participants may have been due to an increased sensitivity to cortisol not present in middle-aged and younger adults. One possible explanation for this increased sensitivity may relate to lower levels of dehydroepiandrosterone (DHEA) generally found in older adults compared to younger adults (Herbert et al., 2006). DHEA is thought to modulate the action of glucocorticoids in the brain (Herbert et al.), thus potentially making the brain more sensitive to the actions of cortisol when DHEA levels are low. This assertion deserves further study. DHEA supplementation has not been shown to enhance cognitive performance in a randomized clinical trial involving 235 older adults (M = 68 years, SD = 8) (Kritz-Silverstein, von Mühlen, Laughlin, & Bettencourt, 2008). Thus, the role of DHEA in older adult cognitive performance is not clear. Additional research would be needed to investigate whether cognitive performance (improvements or deficits) associated with stress-induced cortisol elevations are moderated by DHEA levels.

Lack of significant effects in the younger and middle-aged cortisol responders may have also been due to the use of a working memory task that was not sufficiently

difficult enough to differentiate performance differences according to cortisol increase and time of day of testing. Oei et al. (2006) found that cortisol only impaired performance when working memory loads were high. Perhaps for older adults, the letter-number sequencing task was more taxing on their working memory than in the younger and middle-aged participants. Subsequently, significant performance differences were detected between the morning and afternoon for the older adult cortisol responders.

The pattern of results for older adult cortisol responders and non-responders tested in the afternoon appears to reflect somewhat the findings reported by Het et al. (2005), where pharmacological administration of cortisol in the afternoon was related to a slight memory enhancement compared to a placebo group. Unlike the Het et al. (2005) findings, however, where cortisol given in the morning was associated with impaired recall, older adult cortisol responders in the morning of the present study did not seem to perform worse than same-aged cortisol non-responders tested during this same time. Although speculative, cortisol elevations in the afternoon may have had some beneficial effects upon working memory in these older participants, since the afternoon and evening hours are times when older adults report being less alert and perform less well on working memory tasks than in the morning (Monk & Kupfer, 2007; Yoon, May & Hasher, 2000). On the other hand, slight to moderate increases in the morning, when older adults report being more alert, may not have had an influence on working memory, at least when compared to same-aged cortisol non-responders tested at this same time.

When older adult cortisol responders tested in the morning are compared to same-aged responders tested in the afternoon, performance differences are suggested. Older adult cortisol responders completing the letter-number sequencing test in the afternoon appeared to have better working memory scores than same-aged responders tested in the morning. This implies that cortisol response in the morning when cortisol levels are usually higher may not be beneficial to older adults' working memory compared to elevations in the afternoon when levels tend to be lower.

These assumptions are based upon older adults having a normal diurnal cortisol rhythm. In the present study, information regarding basal cortisol pattern was not available for any of the participants, so it is not known whether older adults possessed a normal diurnal rhythm. Further studies are needed to compare basal levels at specific time points throughout several days to levels expressed on a day containing a cognitive challenge. This would clarify somewhat whether increases are associated with a cognitive challenge and would provide a better means to test whether stress-induced cortisol increases in the morning when levels are normally higher have a less beneficial effect upon working memory than stress-induced increases in the afternoon when levels typically are lower. This investigation would be important since there is variability in basal cortisol levels and diurnal patterns within older adult groups (Herbert et al., 2006).

Basal cortisol levels tend to increase with age, although there is individual variability in this trend (Herbert et al., 2006). Some research has indicated that older adults have decreased morning levels and increased evening levels, resulting in a

flattened diurnal pattern (Herbert et al., 2006). Others have reported the presence of typical, as well as inconsistent patterns, in a small sample (N = 48) of older adults aged 70 to 82 years (Ice, Katz-Stein, Himes, & Kane, 2004). Fifty percent of the Ice et al. (2004) sample expressed normal rhythms, while 48% had inconsistent patterns indicating that the cortisol slopes from the two analysis days varied in size. The remaining 2% had a flattened rhythm. Variability in cortisol patterns has also been found in a sample of community dwelling adults with a mean age of 36.7 years (SD = 12.01) (Smyth et al. 1997). Thus, variability in cortisol rhythm is not isolated to older adults, although the potential mechanisms responsible for variability in older adults may be different than in younger adults (Herbert et al., 2006). Whether stress-induced cortisol increases have different effects upon memory depending upon time of day of testing, as well as type of basal cortisol pattern, deserves further investigation in older and younger adults.

The pattern of effects for older adult cortisol non-responders appears to reflect past research examining time of day effects upon memory span tasks and specific tests of inhibition in younger and older adults (West, Murphy, Armilio, Craik, & Stuss, 2002; Yoon, May & Hasher, 2000). When tested with an object working memory task that included distractors (i.e. 1-back distractor), older adults (M = 72.6 years) committed more errors in the evening (i.e. 5:00 PM) than similarly aged older adults tested in the morning (i.e. 9:00 AM), while younger adults (M = 25.1 years) tested in the morning exhibited more errors than same aged adults tested in the evening (West et al., 2002). In the West et al. study, older adults reported being more alert in the morning than in the

evening, and younger adults indicated being more alert in the evening than in the morning. In the present study, older adult cortisol non-responders tested in the morning had better letter-number sequencing scores than same aged-cortisol non-responders tested in the evening. Measures of alertness, however, were not available so it is not known whether these performance differences were related to feelings of arousal at the different times of day.

Unlike the younger adults in the West et al. (2002) study, time of day did not seem to play a role in the working memory performance of the younger and middle-aged cortisol non-responders in the current study. This finding may have been due to the use of a working memory task that was not sufficiently sensitive enough to detect any potential arousal-related performance differences within these age groups.

Cortisol responders in the afternoon seemed to have the same predicted letter-number sequencing score regardless of age. Although education was controlled in this analysis, the degree that education may be associated with cortisol change from baseline to Trial 5 and therefore was indirectly accountable for this result due to its influence upon cortisol increase was tested. Education was a significant main effect for cortisol change from baseline to Trial 5 and this was qualified by an Age X Education X Time of Day interaction when controlling for gender and longitudinal status. Highly educated older adults who were tested in the afternoon appeared to have a greater increase in cortisol than similarly aged highly educated participants tested in the morning. The difference between these two predicted cortisol change values, however, did not reach significance

(p = .06) most probably because the range in cortisol change from baseline to Trial 5 was large (-18.46 nmol/L to 16.67 nmol/L). Thus, results remain suggestive that older adult cortisol responders in the afternoon were more likely to be highly educated and that this characteristic may have contributed to their predicted letter-number sequencing score. Thus, the possible higher education level of the older adult cortisol responders tested in the afternoon may have also contributed to the previously described performance differences between these older adults and: 1) older adult non-cortisol responders tested in the afternoon, and 2) older adult cortisol responders tested in the morning. Whether the letter-number sequencing performance of highly educated older adults tested in the afternoon was mediated by cortisol elevations was not tested.

Further examination of Figure 7 suggests that highly educated older adults appeared to be more relaxed (i.e. predicted cortisol change scores were negative) when tested in the morning than similarly educated and same aged adults tested in the afternoon. Although speculative, this may have been due to greater perceived alertness in the morning than in the afternoon. When faced with a cognitive challenge in the afternoon when feelings of alertness may be low, highly educated older adults may become more aroused in order to face the demands of the cognitive tasks. The impetus for this arousal could be based in a desire to maintain the social esteem that is associated with good performance on cognitive tests (Dickerson & Kemeny, 2004). Investigation of these assumptions would require a self-report measure of alertness prior to and

throughout the cognitive testing period, as well as a measure assessing whether success on the cognitive tasks was important to the participants' social esteem.

Whether the elevation in cortisol contributed to improved working memory in these older adult participants is not known since this was a correlational study. Few studies other than the Het et al. (2005) meta-analysis have investigated whether cortisol administration produces beneficial or detrimental effects on memory depending upon time of day of dosing. Het et al. did not examine effects upon working memory, however, and little is known regarding whether time of day of cortisol increase may affect memory in older adults, since most studies have used younger adults as subjects. No participant was older than 40 years in the studies reviewed by Het et al.

The current results suggest that highly educated older adult participants may experience an increased cortisol response in the afternoon when faced with a cognitive challenge. This is a potentially important finding since acute elevations in cortisol that initially may be adaptive could become maladaptive if such elevations occur repeatedly (Herbert et al., 2006). Annual increases in plasma cortisol over 3 to 6 years was associated with impaired declarative memory function in older adults (aged 60 to 90 years at baseline) who had currently high cortisol levels at the time of testing (Lupien, Fiocco, et al., 2005).

Unlike cortisol change, there were no significant interactions involving cortisol level. Cortisol change reflects an absolute response to a stressor (Kirschbaum et al.,

1996), whereas cortisol level may not. High levels at baseline, for example, may remain high and unchanged throughout the stressor period.

Declarative memory performance was not significantly related to any interactions involving cortisol. A smaller number of participants took the logical memory immediate ($N = 113$) and the logical memory delayed ($N = 111$) tests compared to the number of subjects who completed letter-number sequencing ($N = 204$). Thus, there may not have been enough power to detect interaction effects. As mentioned previously, the content of the story to be recalled may have been emotionally arousing and this could have facilitated participants' memory regardless of the time of day that increases in cortisol levels occurred.

Research question 6

Cortisol change, and not cortisol level, interacted with skin conductance level and standard deviation in separate moderated regression equations where letter-number sequencing was the dependent variable. The pattern of results for both skin conductance level and standard deviation were similar. Significant slopes, however, were only found for skin conductance standard deviation. Results based on predicted points suggested that cortisol responders who had increased skin conductance standard deviation performed better on the working memory test than cortisol non-responders who had equally high skin conductance variability. On the other hand, cortisol responders who had low skin conductance standard deviation had lower scores than cortisol non-responders who had equally low skin conductance variability.

These findings were not congruent with the Elzinga and Roelofs (2005) study, which indicated that working memory performance was worse in cortisol responders with increased sympathetic arousal compared to cortisol non-responders who also experienced increased sympathetic activity. Further, when measures of sympathetic activity of the cortisol responder group returned to baseline, performance on the working memory task improved.

Elzinga and Roelofs (2005) determined the presence of a sympathetic response by comparing heart rate and blood pressure readings at baseline prior to the TSST with those achieved during working memory testing directly following the TSST. The present study did not have baseline measures; hence, the determination of arousal was less clear. Skin conductance variability, however, was related to skin conductance level, suggesting the presence of potential sympathetic response in those with increased level and variability. Thus, it seems that some degree of arousal occurred in the participants of the present study. The degree or intensity of that arousal, however, cannot be compared between the two studies since different measures of sympathetic activity were used.

It is possible that differences in arousal levels may be responsible for the disparate results. On average, cortisol increased in the Elzinga and Roelofs responder group by approximately 8.0 nmol/L; the predicted point for cortisol-responders in the present study was 4.29 nmol/L (i.e. one standard deviation above the mean change in cortisol from baseline to Trial 5). Thus, it is possible that the participants in the Elzinga and Roelofs study had a greater physiological response than participants in the present study, and as a

result experienced working memory impairment due to that greater response. Differences in the age of the participants (M = 21.33 vs. M = 59.55 years) may also have played a role, although it is unclear whether the sympathetic response of older adults to psychosocial stressors varies from younger adults, due to the limited number of studies that have addressed this issue (Epel, Burke, & Wolkowitz, 2007; Lau, Edelstein, & Larkin, 2001). Some studies have reported greater sympathetic responses in older adults, while others have found no differences between the two different age groups (Epel, Burke, & Wolkowitz, 2007).

It is possible that the mild to moderate increase in cortisol prior to the letter-number sequencing test and increased skin conductance variability during testing may have indicated a constant level of vigilance that was cognitively supportive for some. Those participants who were aroused during testing and who did not have a past cortisol response may have perceived the working memory test as more challenging than the previous tests, and as a result performed less well than those who had possibly maintained attention throughout the testing period. Those participants who had a past cortisol response but did not demonstrate a sympathetic response during the working memory testing may not have been able to maintain the same level of attention and as a result performed more poorly than those who were perhaps more vigilant. Again, these conclusions are speculative and difficult to justify considering that the participants with the least arousal, that is the cortisol non-responders without skin conductance variability, did not appear to perform any worse than those who seemed to be most aroused. One

could assume that these participants were calmer due to increased confidence in their abilities but such assumptions, like the previous speculations, require much further testing with at least baseline measures of skin conductance and self-report measures of arousal and confidence during the testing period.

The mean and standard deviation of heart rate were not significantly associated with letter-number sequencing performance. Heart rate is not a pure measure of sympathetic arousal, since the heart is innervated by both the parasympathetic and sympathetic nervous system (Berntson, Quigley, & Lozano, 2007). Sweat glands are innervated by sympathetic nerves alone, and thus are a better reflection of sympathetic response (Dawson, Schell, & Filion, 2007). Further, heart rate appears to be dominated by parasympathetic influence, and thus is not the most optimal measure for sympathetic arousal (Berntson, Quigley,& Lozano, 2007).

Limitations

Not all of the participants in the Boston subsample completed the cortisol assessments, thus the results obtained may be influenced by selection effects. Data regarding the age and education of those who chose not to participate in the in-home testing were not provided so it is not known whether differences in sample characteristics existed.

Only a small percentage of participants within this study experienced a 2.5 nmol/L increase in cortisol from baseline, an elevation that has been defined as indicative of a definitive cortisol response to a stressor (Kirschbaum et al., 1996). This may have decreased

power to detect some of the potential differences in cortisol reactivity within people based upon between-person differences in personality, age, education, and gender.

Fewer numbers of participants completed the declarative memory tests. This small sample size resulted in a smaller number of participants who experienced a definitive cortisol increase and this may have limited power to detect whether cortisol elevations were associated with declarative memory performance. Further, the declarative memory test included emotional material that may have been arousing and thus supported immediate and briefly delayed recall processes.

The collection of Trial 4 and 5 cortisol samples did not occur immediately prior to the cognitive tests of interest. Between the Trial 4 collection and logical memory immediate, there was a brief break and 2 cognitive tests. Collection of Trial 5 cortisol was followed by 1 cognitive task before the administration of letter-number sequencing and logical memory delayed. Thus, the actual degree of cortisol change from baseline to declarative or working memory testing is not known. It is possible that cortisol levels may have changed further during the interim period between collection and testing. Further, this study employed a correlational design, thus a cause and effect relationship between cortisol change and memory performance cannot be established.

Due to the cross-sectional nature of the study, differences between age groups that were found cannot be differentiated from cohort effects (Schaie & Caskie, 2004). Information regarding ethnicity was not available, so generalizations according to ethnic group cannot be

made. The majority of the participants had from 12 to 20 years of education, thus generalizations to a sample of adults having less than 12 years of education cannot be made.

Conclusions

The present study reports that elevations of cortisol in the afternoon were not associated with impairments in older adults' working memory performance compared to same-aged adults who did not experience a cortisol increase at this same time. Arousal during the afternoon when cortisol levels are lower and when older adults report being less alert may have beneficial effects upon older adults' memory. Further, older adult cortisol responders tested in the afternoon were shown to have better predicted scores on letter-number sequencing than same-aged cortisol responders tested in the morning. Thus, arousal in the morning when cortisol elevations are higher than in the afternoon may not be beneficial to older adults' memory.

Older adults who had cortisol elevations during the afternoon were also highly educated. Thus, arousal processes alone were probably not solely related to the performance differences demonstrated in this study. Cortisol increases in these adults may have been due to social evaluative threat or may have been the result of mechanisms compensating for lower feelings of arousal in the afternoon compared to the morning. Further research is required to test these assumptions and to determine whether acute cortisol increase in the afternoon is cognitively adaptive for memory functioning in older adults.

The present study also reports that younger and middle-aged extraverts maintained a constant level of cortisol while completing a battery of cognitive tests. Further work is needed

to identify why extraverts may respond in this way and whether such a response is cognitively and biologically adaptive.

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Appendices

Appendix A

List of In-Home Procedures

Informed Consent

Salivette #1

Assessment #1

Spielberger State
Forward Digit Span
Backward Digit Span
WAIS Vocab

Salivette #2

Assessment #2

Serial 7
Letter Comparison
Digit Symbol Substitution/Baseline
Letter Series

Salivette #3

Assessment #3

Ravens Advanced Progressive Matrices
Interviewer Report

Salivette #4

Assessment #4

Break
Backwards Counting
Dual Task

Neurodyne ON

Logical Memory Immediate
|
Neurodyne OFF

Task Switching

Salivette #5

Assessment #5

Trails

Neurodyne ON

Letter-Number Sequencing
|
Logical Memory Delayed
|
Neurodyne OFF

Salivette #6

Assessment #6

Diary
Demographic Questionnaire

Salivette #7

Assessment #7

Photograph
PEF

Appendix B

Declarative Memory Testing

"Participant # _____. For the next section, I am going to record your responses for scoring later. Do I have your permission to tape the session? I am now going to read a short story to you. Listen carefully and try to remember it just the way I say it, as close to the same words as you can remember. When I am through, I want you to tell me everything I read to you. You should tell me all you can remember even if you are not sure. Are you ready?"

Anna Thompson of South Boston, employed as a cook in a school cafeteria, reported at the police station that she had been held up on State Street the night before and robbed of fifty-six dollars. She had four small children, the rent was due, and they had not eaten for two days. The police, touched by the woman's story, took up a collection for her.

"Tell me everything you can remember about this story. Start at the beginning."

Story A	**Story Unit**	**Scoring Criteria**
Anna		*Anna* or variant of the name
Thompson		*Thompson* is required
of South		*South* (in any context)
Boston		*Boston* (in any context)
employed		indication that she held a job
as a cook		*cook* or some form of the word is required
in a school		*school* is required
cafeteria		*cafeteria* is required
reported		indication that a formal statement was made to someone in authority
at the police		*police* in any context
station		*station* (in any context) or a word or phrase denoting a police station
that she had been held up		indication that she had been held up (i.e. gunpoint or knife)
on State Street		*State Street* in any context
the night before		indication that the hold-up occurred previous night
and robbed		indication that a robbery took place
of fifty-six dollars		indication that an amount of money greater than $49 but less than $60 was taken from her
She had four		*four* is required along with an indication that the children were hers
small children,		*children* or a synonym is required
the rent was due		a phrase indicating that the rent was due
and they had not eaten		indication that her children or the family were without food
for two days.		*two days* is required or phrase meaning about 2 days
The police		a word or phrase signifying one or more members of the police department (in any context)
touched by her story		indication that her story evoked sympathy
took up a collection		a phrase indicating that money was collected
for her.		indication that the money collected was for her or her children.

Story A Recall Unit Score (Range = 0 – 25)

Appendix C

Revised MIDI Personality Scales – MIDUS II

(For scoring instructions, please see original MIDI personality scales)

Please indicate how well each of the following describes you.

		A lot	Some	A little	Not at all
a)	Outgoing	1	2	3	4
b)	Helpful	1	2	3	4
c)	Moody	1	2	3	4
d)	Organized	1	2	3	4
e)	Self-Confident	1	2	3	4
f)	Friendly	1	2	3	4
g)	Warm	1	2	3	4
h)	Worrying	1	2	3	4
i)	Responsible	1	2	3	4
j)	Forceful	1	2	3	4
k)	Lively	1	2	3	4
l)	Caring	1	2	3	4
m)	Nervous	1	2	3	4
n)	Creative	1	2	3	4
o)	Assertive	1	2	3	4
p)	Hardworking	1	2	3	4
q)	Imaginative	1	2	3	4
r)	Softhearted	1	2	3	4
s)	Calm	1	2	3	4
t)	Outspoken	1	2	3	4
u)	Intelligent	1	2	3	4
v)	Curious	1	2	3	4
w)	Active	1	2	3	4
x)	Careless	1	2	3	4
y)	Broad-minded	1	2	3	4
z)	Sympathetic	1	2	3	4
aa)	Talkative	1	2	3	4
bb)	Sophisticated	1	2	3	4
cc)	Adventurous	1	2	3	4
dd)	Dominant	1	2	3	4
ee)	Thorough	1	2	3	4

Table 1

Descriptive Statistics for Research Questions and Hypotheses 1-4

	N	Mean	Standard deviation	Minimum	Maximum
Age	208	59.51	12.65	35	88
Education (years)	208	15.98	3.30	7.0	26.0
Extraversion	208	3.16	.51	1.80	4.00
Neuroticism	208	2.06	.63	1.00	3.75
Openness	206	3.07	.47	1.86	4.00
Cortisol Trial 1	206	7.54	5.52	.06	34.90
Cortisol Trial 2	206	7.56	5.81	.69	31.60
Cortisol Trial 3	208	7.64	5.45	.52	34.14
Cortisol Trial 4	206	7.21	5.51	.51	33.33
Cortisol Trial 5	203	6.45	4.76	.40	31.66
Cortisol Trial 6	79	6.75	5.08	.33	28.70
Cortisol Trial 7	203	5.58	3.57	.18	20.81
Time at Trial 1	208	754.26	184.89	444.00	1223.00

Note: Cortisol levels are reported in nmol/L and time is reported as minutes from 12 midnight.

Table 2

Descriptive Statistics for Trial to Trial Change in Cortisol Level (nmol/L)

	N	Mean	Standard deviation	Minimum	Maximum
Trial 2 minus Trial 1	205	.02	2.97	-11.58	17.03
Trial 3 minus Trial 2	206	.10	3.19	-18.51	11.26
Trial 4 minus Trial 3	206	-.47	2.92	-11.89	13.91
Trial 5 minus Trial 4	201	-.68	1.61	-8.40	3.56
Trial 6 minus Trial 5	78	-.61	2.68	-14.80	14.39
Trial 7 minus Trial 6	77	-.65	2.09	-7.09	5.27
Trial 7 minus Trial 1	201	-1.9	4.77	-24.21	10.17

Table 3

Descriptive Statistics for Time in Minutes between each Cortisol Collection

	N	Mean	Standard Deviation	Minimum	Maximum
Trial 2 minus Trial 1	208	11.50	3.18	6.00	26.00
Trial 3 minus Trial 2	207	21.00	3.19	13.00	43.00
Trial 4 minus Trial 3	207	21.17	4.91	9.00	41.00
Trial 5 minus Trial 4	205	15.30	5.00	3.00	37.00
Trial 6 minus Trial 5	80	11.24	2.54	5.00	21.00
Trial 7 minus Trial 6	80	25.78	11.52	10.00	99.00
Trial 7 minus Trial 1	202	95.42	20.71	55.00	203.00

Table 4

Unstandardized Coefficients (and Standard Errors) of Multilevel Model of Age and Extraversion Differences in Cortisol Reactivity

Fixed Effects	Unstandardized coefficients (and Standard errors)
Cortisol level, β_{0i}	
Intercept, γ_{00}	18.05* (1.41)
Time of day, γ_{01}	-0.01* (0.001)
Gender, γ_{02}	-2.34* (0.57)
Longitudinal status, γ_{03}	0.75 (0.56)
Education, γ_{04}	0.02 (0.08)
Age, γ_{05}	0.05* (0.02)
Extraversion, γ_{06}	-0.41 (0.57)
Age X Extraversion, γ_{07}	0.03 (0.05)
Reactivity slope, β_{1i}	
Intercept, γ_{10}	-0.33* (0.04)
Age, γ_{11}	0.0004 (0.003)
Extraversion, γ_{12}	0.18* (0.08)
Age X Extraversion, γ_{13}	-0.02* (0.006)

* $p < .05$

Table 5

Unstandardized Coefficients (and Standard Errors) of Multilevel Model of Age and Education Differences in Cortisol Reactivity

Fixed Effects	Unstandardized coefficients (and Standard errors)
Cortisol level, β_{0i}	
Intercept, γ_{00}	18.19* (1.35)
Time of day, γ_{01}	-0.01* (0.001)
Gender, γ_{02}	-2.50* (0.54)
Longitudinal Status, γ_{03}	0.77 (0.54)
Age, γ_{04}	0.06* (0.02)
Education, γ_{05}	0.04 (0.09)
Age X Education, γ_{06}	0.02* (0.01)
Reactivity slope, β_{1i}	
Intercept, γ_{10}	-0.37* (0.04)
Age, γ_{11}	-0.00003 (0.003)
Education, γ_{12}	-0.003 (0.01)
Age X Education, γ_{13}	-0.002* (0.001)

* $p < .05$

Table 6

Unstandardized Coefficients (and Standard Errors) of Multilevel Model of Age and Openness Differences in Cortisol Reactivity

Fixed effects	Unstandardized coefficients (and Standard errors)
Cortisol level, β_{0i}	
Intercept, γ_{00}	17.52* (1.99)
Time of day, γ_{01}	-0.009* (0.0015)
Gender, γ_{02}	-2.26* (0.55)
Longitudinal status, γ_{03}	0.77 (0.56)
Education, γ_{04}	0.02 (0.09)
Age, γ_{05}	0.05* (0.02)
Openness, γ_{06}	-0.20 (0.65)
Age X Openness, γ_{07}	-0.02 (0.05)
Reactivity slope, β_{1i}	
Intercept, γ_{10}	-0.35* (0.04)
Age, γ_{11}	0.0007 (0.003)
Openness, γ_{12}	0.12 (0.08)
Age X Openness, γ_{13}	-0.01 (0.007)

* $p < .05$

Table 7

Unstandardized Coefficients (and Standard Errors) of Multilevel Model of Age and Neuroticism Differences in Cortisol Reactivity

Fixed effects	Unstandardized coefficients (and Standard errors)
Cortisol level, β_{0i}	
Intercept, γ_{00}	17.71* (1.97)
Time of day, γ_{01}	-0.009* (0.001)
Gender, γ_{02}	-2.31* (0.55)
Longitudinal status, γ_{03}	0.71 (0.56)
Education, γ_{04}	0.02 (0.08)
Age, γ_{05}	0.04 (0.02)
Neuroticism, γ_{06}	-0.28 (0.46)
Age X Neuroticism, γ_{07}	0.06 (0.04)
Reactivity slope, β_{1i}	
Intercept, γ_{10}	-0.35* (0.04)
Age, γ_{11}	0.002 (0.003)
Neuroticism, γ_{12}	0.04 (0.06)
Age X Neuroticism, γ_{13}	-0.008 (0.005)

* $p < .05$

Table 8

Unstandardized Coefficients (and Standard Errors) of Multilevel Model of Extraversion and Education Differences in Cortisol Reactivity

Fixed effects	Unstandardized coefficients (and Standard errors)
Cortisol level, β_{0i}	
Intercept, γ_{00}	14.82* (2.07)
Time of day, γ_{01}	-0.009* (0.001)
Gender, γ_{02}	-2.31* (0.56)
Longitudinal status, γ_{03}	0.81 (0.56)
Age, γ_{04}	0.05* (0.02)
Extraversion, γ_{05}	-0.45 (0.57)
Education, γ_{06}	0.07 (0.09)
Extraversion X Education, γ_{07}	0.15 (0.17)
Reactivity slope, β_{1i}	
Intercept, γ_{10}	-0.34* (0.04)
Extraversion, γ_{11}	0.19* (0.08)
Education, γ_{12}	-0.01 (0.01)
Extraversion X Education, γ_{13}	0.001 (0.02)

* $p < .05$

Table 9

Unstandardized Coefficients (and Standard Errors) of Multilevel Model of Extraversion and Gender Differences in Cortisol Reactivity

Fixed effects	Unstandardized coefficients (and Standard errors)
Cortisol level, β_{0i}	
Intercept, γ_{00}	14.47* (2.13)
Time of day, γ_{01}	-0.009* (0.001)
Age, γ_{02}	0.06* (0.02)
Longitudinal status, γ_{03}	0.70 (0.56)
Education, γ_{04}	0.03 (0.08)
Extraversion, γ_{05}	-3.59 (1.84)
Gender, γ_{06}	-2.36* (0.60)
Extraversion X Gender, γ_{07}	2.08 (1.15)
Reactivity slope, β_{1i}	
Intercept, γ_{10}	-0.37* (0.13)
Extraversion, γ_{11}	0.60* (0.24)
Gender, γ_{12}	0.02 (0.08)
Extraversion X Gender, γ_{13}	-0.27 (0.16)

* $p < .05$

Table 10

Unstandardized Coefficients (and Standard Errors) of Multilevel Model of Openness and Education Differences in Cortisol Reactivity

Fixed Effects	Unstandardized coefficients (and Standard errors)
Cortisol level, β_{0i}	
Intercept, γ_{00}	14.82* (2.06)
Time of day, γ_{01}	-0.009* (0.001)
Age, γ_{02}	0.05* (0.02)
Longitudinal status, γ_{03}	0.75 (0.56)
Gender, γ_{04}	-2.32* (0.55)
Openness, γ_{05}	-0.35 (0.65)
Education, γ_{06}	0.05 (0.09)
Openness X Education, γ_{07}	0.27 (0.19)
Reactivity slope, β_{1i}	
Intercept, γ_{10}	-0.33* (0.04)
Openness, γ_{11}	0.16 (0.09)
Education, γ_{12}	-0.017 (0.01)
Openness X Education, γ_{13}	-0.03 (0.03)

* $p < .05$

Table 11

Unstandardized Coefficients (and Standard Errors) of Multilevel Model of Openness and Gender Differences in Cortisol Reactivity

Fixed Effects	Unstandardized coefficients (and Standard errors)
Cortisol level, β_{0i}	
Intercept, γ_{00}	14.69* (2.63)
Time of day, γ_{01}	-0.009* (0.001)
Age, γ_{02}	0.05* (0.02)
Longitudinal status, γ_{03}	0.74 (0.56)
Education, γ_{04}	0.02 (0.09)
Openness, γ_{05}	2.19 (2.01)
Gender, γ_{06}	-2.42* (0.60)
Openness X Gender, γ_{07}	-1.55 (1.24)
Reactivity slope, β_{1i}	
Intercept, γ_{10}	-0.42* (0.13)
Openness, γ_{11}	-0.12 (0.27)
Gender, γ_{12}	0.05 (0.08)
Openness X Gender, γ_{13}	0.15 (0.17)

* $p < .05$

Table 12

Unstandardized Coefficients (and Standard Errors) of Multilevel Model of Neuroticism and Education Differences in Cortisol Reactivity

Fixed effects	Unstandardized coefficients (and Standard errors)
Cortisol level, β_{0i}	
Intercept, γ_{00}	14.97* (2.06)
Time of day, γ_{01}	-0.009* (0.001)
Gender, γ_{02}	-2.28* (0.55)
Longitudinal status, γ_{03}	0.71 (0.57)
Age, γ_{04}	0.05* (0.02)
Neuroticism, γ_{05}	-0.29* (0.57)
Education, γ_{06}	0.06 (0.09)
Neuroticism X Education, γ_{07}	-0.09 (0.14)
Reactivity slope, β_{1i}	
Intercept, γ_{10}	-0.34* (0.04)
Neuroticism, γ_{11}	0.04 (0.06)
Education, γ_{12}	-0.01 (0.01)
Neuroticism X Education, γ_{13}	-0.01 (0.02)

* $p < .05$

Table 13

Unstandardized Coefficients (and Standard Errors) of Multilevel Model of Neuroticism and Gender Differences in Cortisol Reactivity

Fixed Effects	Unstandardized Coefficients (and Standard Errors)
Cortisol level, β_{0i}	
Intercept, γ_{00}	14.59* (2.63)
Time of day, γ_{01}	-0.009* (0.001)
Age, γ_{02}	0.05* (0.02)
Longitudinal status, γ_{03}	0.72 (0.57)
Education, γ_{04}	0.03 (0.08)
Neuroticism, γ_{05}	0.94 (1.44)
Gender, γ_{06}	-2.42* (0.59)
Neuroticism X Gender, γ_{07}	-0.85 (0.91)
Reactivity slope, β_{1i}	
Intercept, γ_{10}	-0.42* (0.13)
Neuroticism, γ_{11}	0.05 (0.20)
Gender, γ_{12}	0.05 (0.08)
Neuroticism X Gender, γ_{13}	-0.006 (0.13)

* $p < .05$

Table 14

Descriptive Statistics for Research Question and Hypothesis 5 where Letter-Number Sequencing was the Dependent Variable

	N	Mean	Standard deviation	Minimum	Maximum
Age	204	59.48	12.40	35	88
Education (years)	204	15.90	3.27	7.0	26.0
Letter-number sequencing	204	10.57	2.80	2	19
Cortisol Trial 5	204	6.35	4.61	.40	31.66
Cortisol change from baseline to Trial 5	204	-1.19	5.05	-18.46	16.67
Time at Trial 5	204	819.52	181.80	524.00	1285.00
Trial 5 time minus Trial 1 time	204	69.17	9.43	50.00	104.00

Note: Cortisol levels are reported in nmol/L and time is reported as minutes from 12 midnight.

Table 15

Descriptive Statistics for Research Question and Hypothesis 5 where a Test of Declarative Memory was the Dependent Variable

	N	Mean	Standard deviation	Minimum	Maximum
Age	113	59.04	12.52	35	84
Education (years)	113	16.18	3.62	10.0	26.0
Logical memory immediate	113	13.11	3.52	6	22
Cortisol Trial 4	113	7.82	6.31	.51	33.33
Cortisol change from baseline to Trial 4	113	-.26	5.95	-15.67	19.30
Time at Trial 4	113	806.60	191.79	508.00	1257.00
Trial 4 time minus Trial 1 time	113	53.98	8.04	36.00	91.00
Logical memory delayed	110	11.67	4.23	0	21
Cortisol Trial 5	110	6.87	5.30	.40	31.66
Cortisol change from baseline to Trial 5	110	-1.32	5.67	-18.46	16.67
Time at Trial 5	110	816.45	186.94	524.00	1272.00
Trial 5 time minus Trial 1 time	110	72.05	8.54	56.00	104.00

Note: Cortisol levels are reported in nmol/L and time is reported as minutes from 12 midnight.

Table 16

Results from Moderated Regression Analyses where Cortisol Level was a Predictor and Logical Memory Immediate and Delayed were Dependent Variables

	Logical memory immediate:		Logical memory delayed:	
Predictors:	Standardized coefficient	*t*-value	Standardized coefficient	*t*-value
Education	0.29*	3.14*	0.26*	2.73*
Age	-0.26*	-2.61*	-0.26*	-2.20*
Cortisol level	-0.10	-0.91	-0.13	-1.06
Time of day	-0.23	-1.91	-0.12	-0.95
Age X Cortisol Level	0.003	0.03	-0.08	-0.58
Age X Time of Day	0.004	0.03	-0.06	-0.38
Cortisol Level X Time of Day	0.07	0.58	-0.009	-0.07
Age X Cortisol Level X Time of Day	0.006	0.05	-0.05	-0.33

Note. $R^2 = 0.19$ for logical memory immediate; $R^2 = 0.14$ for logical memory delayed.
*$p < .05$

Table 17

Results from Moderated Regression Analyses where Cortisol Change was a Predictor and Logical Memory Immediate and Delayed were Dependent Variables

	Logical memory immediate:		Logical memory delayed:	
Predictors:	Standardized coefficient	*t*-value	Standardized coefficient	*t*-value
Education	0.28*	3.13*	0.24*	2.58*
Age	-0.27*	-2.89*	-0.26*	-2.68*
Cortisol change	-0.01	-0.13	-0.10	-0.90
Time of day	-0.22*	-2.27*	-0.06	-0.52
Age X Cortisol Change	0.05	0.45	-0.12	-0.86
Age X Time of Day	-0.003	-0.03	0.01	0.14
Cortisol Change X Time of Day	0.09	0.77	-0.10	-0.83
Age X Cortisol Change X Time of Day	0.05	0.40	-0.10	-0.73

Note. $R^2 = 0.19$ for logical memory immediate; $R^2 = 0.14$ for logical memory delayed.
*$p < .05$

Table 18

Moderated Regression Analyses where Letter-Number Sequencing was the Dependent Variable

	Cortisol level:		Cortisol change:	
Predictors:	Standardized coefficient	*t*-value	Standardized coefficient	*t*-value
Education	0.20*	3.14*	0.19*	2.92*
Age	-0.38*	-5.02*	-0.40*	-6.25*
Cortisol	0.02	0.26	0.07	1.04
Time of day	-0.05	-0.60	-0.02	-0.36
Age X Cortisol	0.04	0.49	0.17*	2.14*
Age X Time of Day	0.06	0.64	0.005	0.08
Cortisol X Time of Day	-0.04	-0.57	0.13	1.85
Age X Cortisol X Time of Day	0.08	0.75	0.21*	2.55*

Note. $R^2 = 0.24$ when cortisol level was a predictor; $R^2 = 0.27$ when cortisol change was a predictor. Pattern of results remained the same when baseline cortisol was entered as a covariate.
*$p < .05$

Table 19

Moderated Regression Analysis where Cortisol Change (from Baseline to Trial 5) was the Dependent Variable

Predictors:	Standardized coefficient	*t*-value
Gender	-0.09	-1.18
Longitudinal status	-0.10	-1.36
Education	0.15*	2.03*
Age	0.06	0.77
Time of day	0.13	1.70
Age X Education	-0.03	-0.40
Age X Time of Day	-0.0002	< -0.001
Education X Time of Day	0.08	1.07
Age X Education X Time of Day	0.16*	2.01*

Note. R^2= .06. Pattern of results remained the same when baseline cortisol was entered as a covariate.
*$p < .05$

Table 20

Descriptive Statistics for Research Question 6

	N	Mean	Standard deviation	Minimum	Maximum
Age	75	59.55	12.34	35	84
Education	75	16.26	3.82	10.0	26.0
Letter-number sequencing	75	10.95	2.60	6	19
Cortisol Trial 5	75	6.74	5.25	.40	31.66
Cortisol change from baseline to Trial 5	75	-1.29	5.58	-18.46	12.78
Time at Trial 5	75	815.89	184.27	587.00	1226.00
Skin conductance standard deviation	75	.34	.33	.01	2.07
Skin conductance level	75	3.90	2.17	1.29	12.24
Heart rate standard deviation	76	9.39	5.65	1.48	33.90
Heart rate level	76	69.68	9.57	44.93	98.78

Note: Cortisol levels are reported in nmol/L, skin conductance in micromhos, heart rate is beats per minute, and time is reported as minutes from 12 midnight.

Table 21

Moderated Regression Analyses where Heart Rate and Cortisol Level were Predictors of Letter-Number Sequencing

	Heart rate *M*:		Heart rate *SD*:	
Predictors:	Standardized coefficient	*t*-value	Standardized coefficient	*t*-value
Education	0.36*	2.79*	0.35*	2.55*
Time of day	0.05	0.35	-0.003	-0.02
Smoking status	-0.22	-1.80	-0.13	-1.06
Hypertensive status	0.01	0.09	-0.006	-0.05
Age	-0.30*	-2.23*	-0.30*	-2.27*
Cortisol level	-0.03	-0.17	0.03	0.16
Heart rate	-0.09	-0.66	-0.13	-0.91
Age X Cortisol Level	-0.11	-0.80	-0.15	-0.71
Age X Heart Rate	0.005	0.04	0.05	0.28
Cortisol Level X Heart Rate	0.15	1.13	-0.14	-0.58
Age X Cortisol Level X Heart Rate	0.03	0.22	0.11	0.35

Note. $R^2 = 0.30$ when heart rate *M* was a predictor; $R^2 = 0.28$ when heart rate *SD* was a predictor.
* $p < .05$

Table 22

Moderated Regression Analyses where Heart Rate and Cortisol Change were Predictors of Letter-Number Sequencing

	Heart rate *M*:		Heart rate *SD*:	
Predictors:	Standardized coefficient	*t*-value	Standardized coefficient	*t*-value
Education	0.36*	3.01*	0.32*	2.67*
Time of day	0.03	0.20	-0.03	-0.27
Smoking status	-0.22	-1.90	-0.16	-1.38
Hypertensive status	0.01	0.08	-0.004	-0.03
Age	-0.31*	-2.32*	-0.26*	-2.13*
Cortisol change	0.08	0.65	0.06	0.51
Heart rate	-0.13	-1.05	-0.12	-0.89
Age X Cortisol Change	-0.03	-0.23	0.06	0.38
Age X Heart Rate	0.03	0.30	0.02	0.15
Cortisol Change X Heart Rate	0.18	1.38	0.06	0.32
Age X Cortisol Change X Heart Rate	0.12	0.93	-0.22	-0.93

Note. R^2= 0.33 when heart rate *M* was a predictor; R^2 = 0.29 when heart rate *SD* was a predictor.
* $p < .05$

Table 23

Moderated Regression Analyses where Skin Conductance and Cortisol Level were Predictors of Letter-Number Sequencing

	Skin conductance *M*:		Skin conductance *SD*:	
Predictors:	Standardized coefficient	*t*-value	Standardized coefficient	*t*-value
Education	0.33*	2.43*	0.32*	2.60*
Time of day	0.06	0.41	0.02	0.17
Smoking status	-0.20	-1.80	-0.22*	-2.05*
Hypertensive status	-0.03	-0.29	-0.03	-0.27
Age	-0.30*	-2.37*	-0.28*	-2.27*
Cortisol level	0.11	0.67	0.01	0.09
Skin conductance	0.20	1.65	0.34*	2.16*
Age X Cortisol Level	-0.19	-1.30	-0.11	-0.90
Age X Skin Conductance	-0.006	-0.05	0.04	0.20
Cortisol Level X Skin Conductance	0.17	1.23	0.25	1.87
Age X Cortisol Level X Skin Conductance	-0.05	-0.34	-0.11	-0.52

Note. $R^2 = 0.33$ when skin conductance *M* was a predictor; $R^2 = 0.40$ when skin conductance *SD* was a predictor.
* $p < .05$

Table 24

Moderated Regression Analyses where Skin Conductance and Cortisol Change were Predictors of Letter-Number Sequencing

	Skin Conductance *M*:		Skin Conductance *SD*:	
Predictors:	Standardized coefficient	*t*-value	Standardized coefficient	*t*-value
Education	0.33*	2.80*	0.30*	2.64*
Time of day	-0.008	-0.07	0.07	0.64
Smoking status	-0.22	-1.95	-0.18	-1.74
Hypertensive status	0.01	0.09	0.03	0.30
Age	-0.24*	-2.03*	-0.23	-1.97
Cortisol change	0.10	0.87	0.07	0.64
Skin conductance	0.19	1.61	0.15	0.96
Age X Cortisol Change	-0.09	-0.73	-0.07	-0.63
Age X Skin Conductance	0.04	0.28	0.25	1.88
Cortisol Change X Skin Conductance	0.27*	2.38*	0.31*	2.58*
Age X Cortisol Change X Skin Conductance	-0.09	-0.69	-0.009	-0.07

Note. $R^2 = 0.37$ when skin conductance *M* was a predictor; $R^2 = 0.43$ when skin conductance *SD* was a predictor.
* $p < .05$

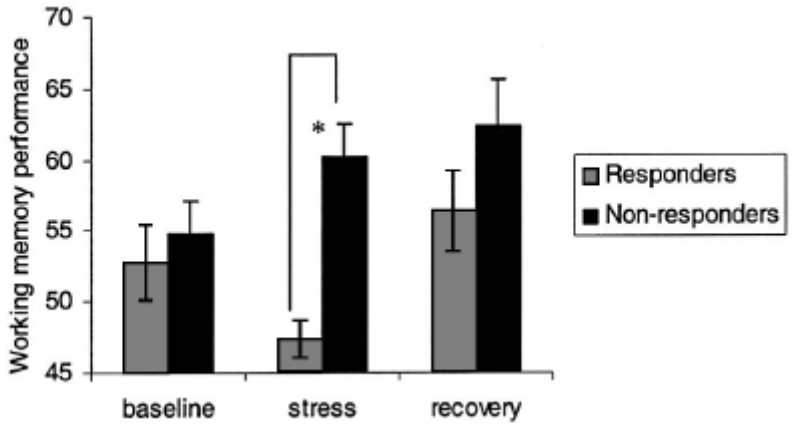

Figure 1. Working memory performance at baseline, and during the stress and recovery periods of the TSST for cortisol responders and non-responders from Elzinga and Roelofs (2005; p. 101). * indicates a significant difference at $p < .05$.

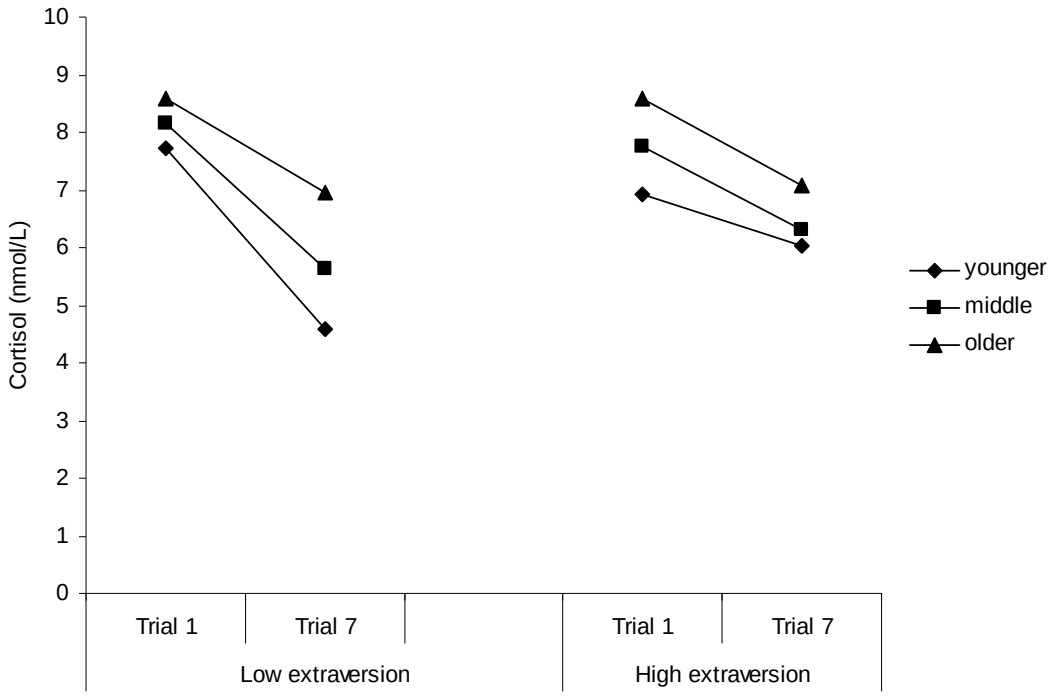

Figure 2. Predicted age and extraversion differences in cortisol reactivity, controlling for testing time, gender, longitudinal group status and education. The lines for age were plotted using the average age (59 years) for the middle, one standard deviation below the mean (47 years) for the younger, and one standard deviation above the mean (72 years) for the older adults. One standard deviation below mean extraversion indicates low extraversion (2.65) and one standard deviation above the mean represents high extraversion (3.67).

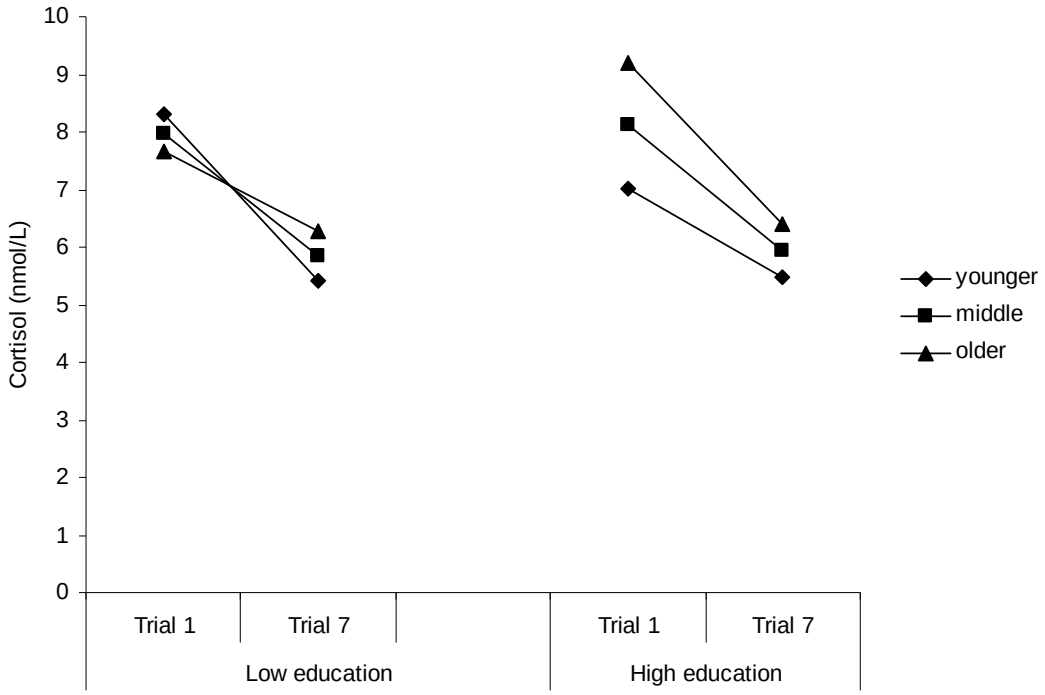

Figure 3. Predicted age and education differences in cortisol reactivity across trials, controlling for testing time, gender, and longitudinal group status. The lines for age were plotted using the average age (59 years) for the middle, one standard deviation below the mean (47 years) for the younger, and one standard deviation above the mean (72 years) for the older adults. Low education represents participants who had a 2-year degree or less (N = 81) and high education represents participants who had a bachelor's or more (N = 127).

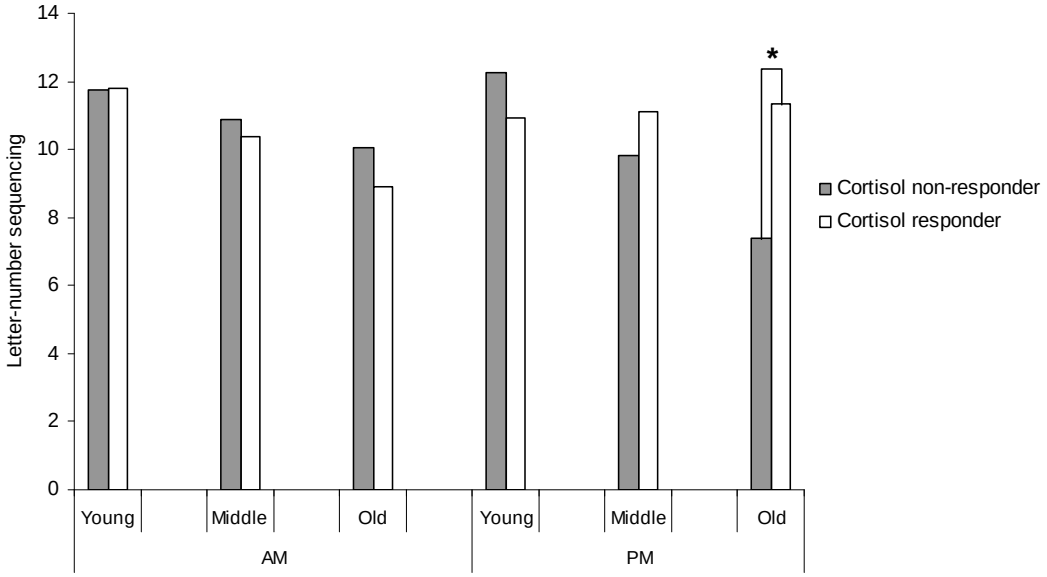

Figure 4. Conceptualization 1 of Age X Cortisol Change X Time of Day interaction. Comparison of predicted letter-number sequencing scores of cortisol responders and non-responders graphed as a function of age and time of day, controlling for education. Age was entered using the average age (59 years) for the middle, one standard deviation below the mean (47 years) for the younger, and one standard deviation above the mean (72 years) for the older adults. One standard deviation below the mean change in cortisol from baseline to Trial 5 represented cortisol non-responders (-6.24 nmol/L), while one standard deviation above that mean indicated cortisol responders (3.86 nmol/L). Time in the AM represented one standard deviation below the mean for time at Trial 5 (637.72 minutes or 10:38 AM) and time in the PM represented one standard deviation above the mean for the same time point (1001.32 minutes or 4:41 PM).
* indicates a significant difference at $p < .05$.

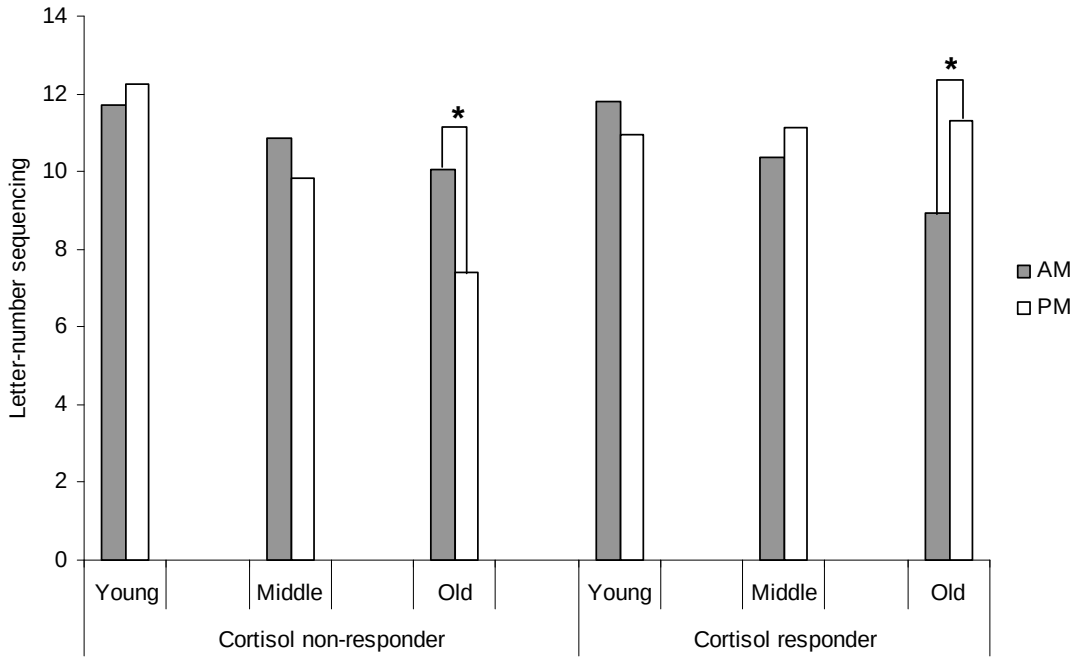

Figure 5. Conceptualization 2 of Age X Cortisol Change X Time of Day interaction. Comparison of predicted letter-number sequencing scores in the morning and afternoon graphed as a function of age and cortisol response, controlling for education. Age was entered using the average age (59 years) for the middle, one standard deviation below the mean (47 years) for the younger, and one standard deviation above the mean (72 years) for the older adults. One standard deviation below the mean change in cortisol from baseline to Trial 5 represented cortisol non-responders (-6.24 nmol/L), while one standard deviation above that mean indicated cortisol responders (3.86 nmol/L). Time in the AM represented one standard deviation below the mean for time at Trial 5 (637.72 minutes or 10:38 AM) and time in the PM represented one standard deviation above the mean for the same time point (1001.32 minutes or 4:41 PM).
* indicates a significant difference at $p < .05$.

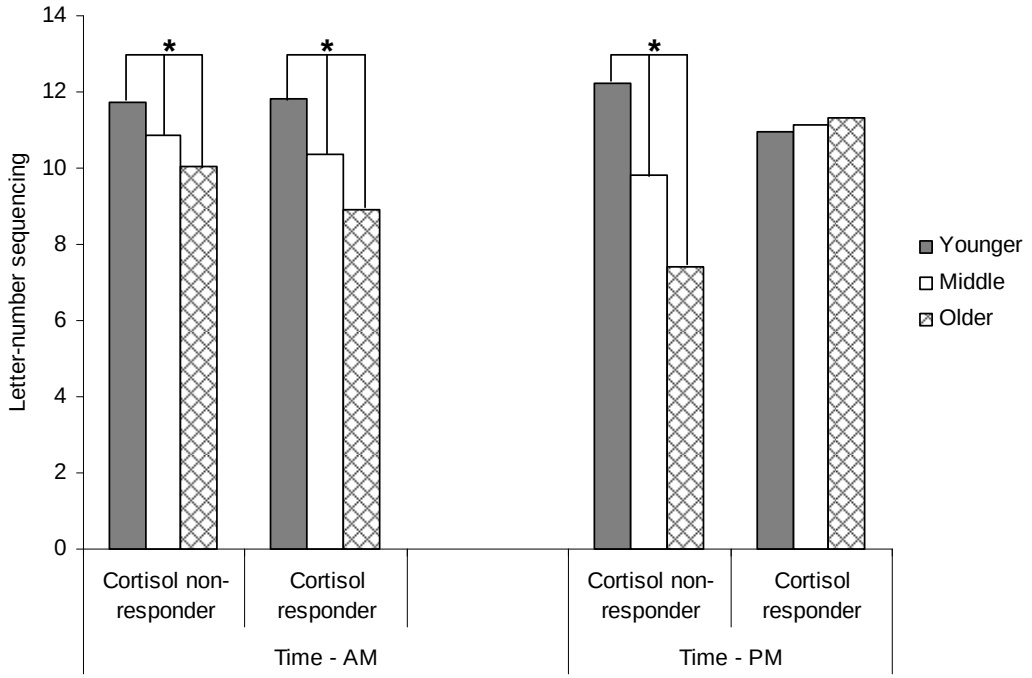

Figure 6. Conceptualization 3 of Age X Cortisol Change X Time of Day interaction. Comparison of predicted letter-number sequencing scores of young, middle-aged and older adults graphed as a function of cortisol response and time of day, controlling for education. Age was entered using the average age (59 years) for the middle, one standard deviation below the mean (47 years) for the younger, and one standard deviation above the mean (72 years) for the older adults. One standard deviation below the mean change in cortisol from baseline to Trial 5 represented cortisol non-responders (-6.24 nmol/L), while one standard deviation above that mean indicated cortisol responders (3.86 nmol/L). Time in the AM represented one standard deviation below the mean for time at Trial 5 (637.72 minutes or 10:38 AM) and time in the PM represented one standard deviation above the mean for the same time point (1001.32 minutes or 4:41 PM).
* indicates a significant difference at $p < .05$.

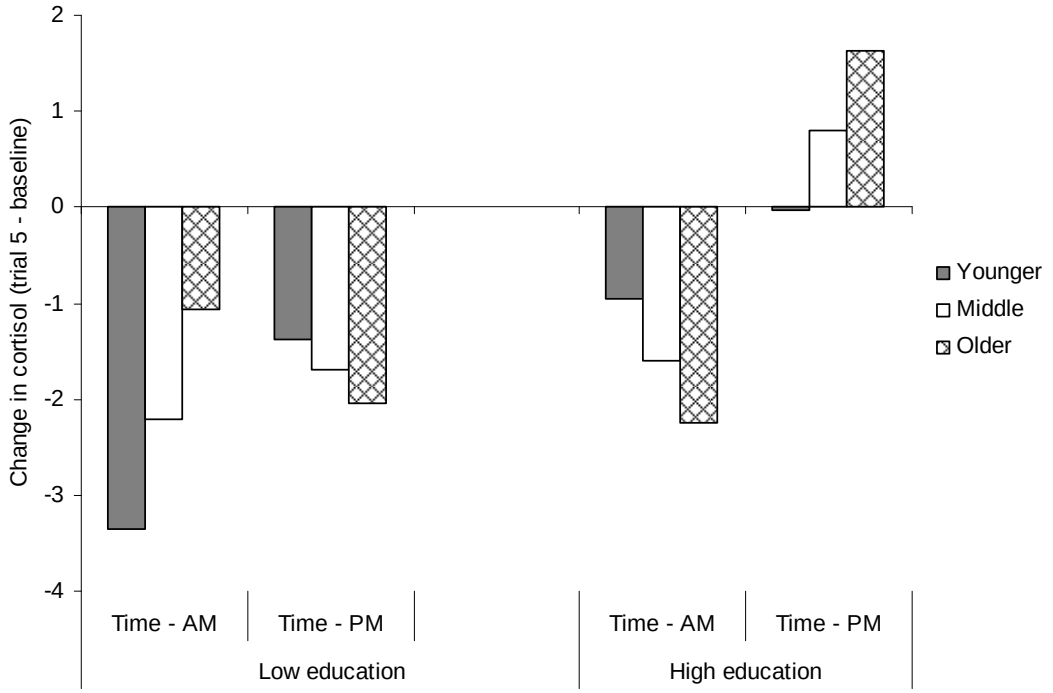

Figure 7. Predicted change in cortisol from baseline to trial 5 as a function of education, time of day, and age, controlling for gender and longitudinal status. Columns for age were graphed using the average age (59 years) for the middle, one standard deviation below the mean (47 years) for the younger, and one standard deviation above the mean (72 years) for the older adults. One standard deviation below the mean education in years represents low education (12.72 years) and one standard deviation above the mean education in years indicates high education (19.18 years). Time in the AM represents one standard deviation below the mean for time at Trial 5 (637.72 minutes or 10:38 AM) and time in the PM represents one standard deviation above the mean for time at Trial 5 (1001.32 minutes or 4:41 PM).

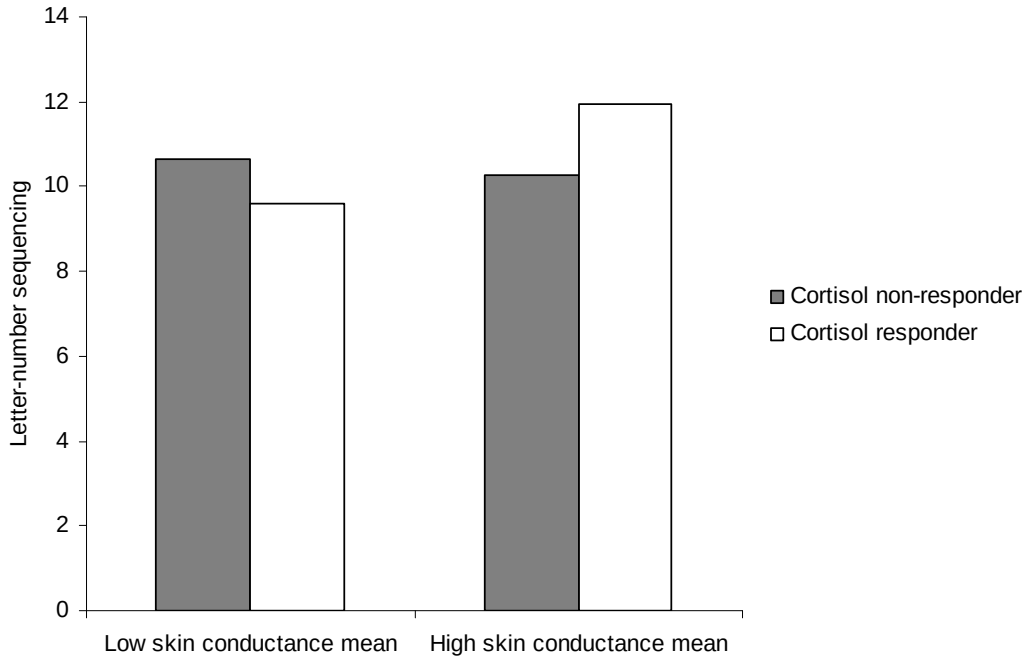

Figure 8. Predicted performance on letter-number sequencing as a function of cortisol response and mean skin conductance level, controlling for age, education, time of day of testing, smoking status, and hypertensive status. Columns for mean skin conductance were graphed using one standard deviation below the mean for the low level (1.7 micromhos), and one standard deviation above the mean for the high level (6.02 micromhos). One standard deviation below the mean change in cortisol from baseline to trial 5 represents cortisol non-responders (-6.87) and one standard deviation above that mean indicates cortisol responders (4.29).

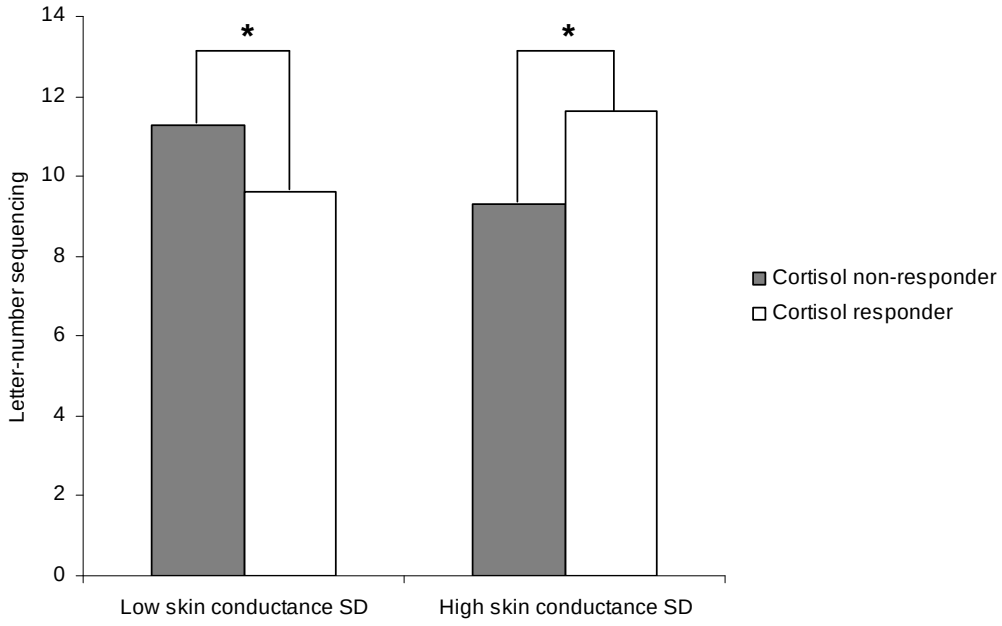

Figure 9. Predicted performance on letter-number sequencing as a function of cortisol response and standard deviation of skin conductance level, controlling for age, education, time of day of testing, smoking status, and hypertensive status. Columns for the standard deviation of skin conductance were graphed using one standard deviation below the skin conductance standard deviation mean for the low level (0), and one standard deviation above the skin conductance standard deviation mean for the high level (0.66). One standard deviation below the mean change in cortisol from baseline to trial 5 represents cortisol non-responders (-6.87) and one standard deviation above that mean indicates cortisol responders (4.29). * indicates a significant difference at $p < .05$.