2009

The effects of psychosocial stress and social support on memory

Dong T. H Nguyen
San Jose State University

Follow this and additional works at: https://scholarworks.sjsu.edu/etd_theses

Recommended Citation

Nguyen, Dong T. H, "The effects of psychosocial stress and social support on memory" (2009). *Master's Theses*. 3668.

DOI: <https://doi.org/10.31979/etd.cdudz-9z84>

https://scholarworks.sjsu.edu/etd_theses/3668

This Thesis is brought to you for free and open access by the Master's Theses and Graduate Research at SJSU ScholarWorks. It has been accepted for inclusion in Master's Theses by an authorized administrator of SJSU ScholarWorks. For more information, please contact scholarworks@sjsu.edu.

THE EFFECTS OF PSYCHOSOCIAL STRESS
AND SOCIAL SUPPORT ON MEMORY

A Thesis

Presented to

The Faculty of the Department of Psychology

San Jose State University

In Partial Fulfillment

of the Requirements for the Degree

Master of Arts

by

Dong T. H. Nguyen

May 2009

UMI Number: 1470974

Copyright 2009 by
Nguyen, Dong T. H.

INFORMATION TO USERS

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleed-through, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

UMI Microform 1470974
Copyright 2009 by ProQuest LLC
All rights reserved. This microform edition is protected against
unauthorized copying under Title 17, United States Code.

ProQuest LLC
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106-1346

© 2009

Dong T. H. Nguyen

ALL RIGHTS RESERVED

SAN JOSE STATE UNIVERSITY

The Undersigned Thesis Committee Approves the Thesis Titled

THE EFFECTS OF PSYCHOSOCIAL STRESS

AND SOCIAL SUPPORT ON MEMORY

by

Dong T. H. Nguyen

APPROVED FOR THE DEPARTMENT OF PSYCHOLOGY

Dr. Cheryl Chancellor-Freeland, Department of Psychology

4-3-09

Date

Dr. Megumi Hosoda, Department of Psychology

4-3-09

Date

Dr. Nancy Da Silva, Department of Organization and Management

4-6-09

Date

APPROVED FOR THE UNIVERSITY

Associate Dean Office of Graduate Studies and Research

5/5/09

Date

ABSTRACT

THE EFFECTS OF PSYCHOSOCIAL STRESS AND SOCIAL SUPPORT ON MEMORY

by Dong T. H. Nguyen

Stress is undeniably a part of everyday life, and the most profound types of stressors are psychosocial ones, producing a substantial rise in the stress hormone, cortisol. This rise in cortisol has been reported to impair memory. Social support is reported to buffer the effects of stress on overall well-being, and there are reasons to believe that men and women differentially benefit from social support. Therefore, the purpose of this study was to investigate the effects of stress on declarative memory and, in particular, how preexisting social support may mitigate this relationship. In addition, it was determined whether gender-specific effects were present. Seventy-two participants in the study were semi-randomly assigned either to the Trier Social Stress Test or to a control condition. Results showed no differences in declarative memory between the stress and control groups. Furthermore, preexisting social support did not buffer the effects of stress on memory. Gender differences and implications of the findings are discussed.

ACKNOWLEDGEMENTS

I'd like to thank my long-time mentor and thesis advisor, Dr. Cheryl Chancellor-Freeland, for her never-ending support and encouragement throughout my undergraduate and graduate education. She has always believed in me whenever I would doubt myself. She's taught me so much more than I can ever imagine. She's devoted so much time and effort in helping me achieve my goals that I can never say thank you enough. I'd also like to thank Dr. Megumi Hosoda for serving on my thesis committee, in addition to her guidance with statistical analyses. Furthermore, I'd also like to thank Dr. Nancy Da Silva for serving on my thesis committee. Finally, I'd like to give special thanks to colleagues who have been with me from the beginning of the project: Kerri Bayareddy, Anosha Shokrpour, and Anthony Holguin. Without the help of all these people, I never would have thought I would ever finish my thesis. Thank you everyone, for all your support.

TABLE OF CONTENTS

List of Figures.....	vii
Introduction.....	1
Stress and Cortisol	2
Cortisol and Memory	4
Inconsistencies in the Literature	5
Preexisting Social Support as a Mitigating Factor of Stress.....	8
Methods.....	11
Participants.....	11
Procedure	12
Cortisol Sample Collection	13
Stress Manipulation.....	15
Trier Social Stress Test	15
Coding.....	15
Gender	15
Measures	15
Preexisting Social Support.....	15
Perceived Stress	16
Memory Performance..	16
Results.....	17
Discussion	31
References	41

List of Figures

Figure 1. Mean salivary cortisol concentrations at each time point from a subset of control (n= 2) and stress (n=16) participants.	19
Figure 2. Mean delta cortisol for control and stress groups.....	21
Figure 3. Declarative memory performance for control and stress groups....	23
Figure 4. Effects of preexisting social support on stress and declarative memory performance.....	25
Figure 5. Delta salivary cortisol differences by gender.	27
Figure 6. Differences in perceived stress and preexisting social support in men.....	30

Introduction

Whether it comes from work, school, friends, or family, stress is undeniably a part of the fabric of modern life. Stress can be defined as strain caused by external demands on the organism, and it is evidenced by a substantial rise in the neurochemical, cortisol (Kalat, 2007). Psychological, physiological, as well as psychosocial factors (e.g., arising from one's social environment) can induce stress, and the negative consequences can be seen as a host of health problems including, but not limited to, a weakened immune response, major depression, cardiovascular diseases, and the exacerbation of existing diseases (Burg, 1992; Cohen, Doyle, Skoner, Rabin, & Gwaltney, 1997; Cohen, Hammen, Henry, & Daley, 2004; Hale, Hannum, & Espelage, 2005; Koh, Choe, Song, & Lee, 2006; Krantz & McCeney, 2002; Miyazaki et al., 2005). In terms of its effect on cognitive ability, stress is known to impair learning and memory (Wolf, 2003), presumably by way of cortisol's inhibitory effects on the formation of new memory and the retrieval of already existing memories.

Clearly, the effects of stress can be broad-reaching and deleterious, and the level of daily stress in modern life is ever-increasing. It is for these reasons that scientists have intensified efforts to determine factors that can buffer the negative effects of stress. One factor of interest is social support. Cohen and Wills (1985), in their seminal review, concluded that social support and, in particular, the perceived availability of social support, serve to buffer the effects of stress on health and well-being. The implication being that those individuals who have strong social support systems would be more robust and more resilient in the face of stressors. It also seems reasonable to expect that

this effect would translate beyond physical health to mental wellness and, in particular, to the preservation of cognitive integrity.

The aim of the present study was to test these ideas. We sought to determine whether or not social support would mitigate the negative effects of an acute stressor on declarative memory, the type of memory affected by stress-induced elevation in cortisol (Wolf, 2003). To the best of our knowledge, this has not been previously investigated. Furthermore, because some have reported that social support does not benefit women and men equally, we also sought to determine whether social support would have a gender-specific effect. Because women report seeking social support more than men (Ashton & Fuehrer, 1993), we expected women to have a more supportive social network compared to men and consequently to benefit more from social support in terms of being more protected from the memory-impairing effects of stress.

Stress and Cortisol

The original definition of stress by Hans Selye is “the non-specific response of the body to any demand for change” (as cited in Kalat, 2007, p. 366). In the face of a threat, there is a natural tendency for humans to exhibit the “fight or flight” response, and this serves to promote survival by activating the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis is a self-regulating system that allows the body to maintain homeostasis and mobilize energy when necessary. When activated, the HPA axis produces and releases a cascade of neurochemicals, including glucocorticoids that are secreted from the adrenal glands. One type of glucocorticoid, called cortisol, is released in regular cycles throughout the day, with high levels in the morning and low levels in the evening.

During a stress response, a substantial rise in cortisol helps mobilize glucose for muscle cells to utilize more efficiently, and, for this reason, cortisol is widely referred to as the “stress hormone” and is often used to quantify stress severity (Kalat, 2007).

The events that induce stress, in other words “stressors,” come in many different forms. In animal studies, environmental stressors such as restraint, cold water immersion, and electrical foot shocks have been shown to significantly increase cortisol levels (Harris et al., 2004; Retana-Marquez et al., 2003). However, more recent data suggest that the most profound type of stressor is a psychosocially-derived one, particularly one produced by interpersonal interactions (DeVries, Glasper, & Detillion, 2003). This is true for rodent, nonhuman primate, and human models. Nonhuman primates and humans exposed to psychosocial stress exhibit elevations in cortisol comparable to those seen in mice and rats. For example, in socially stable environments, dominant male baboons usually have lower cortisol baselines compared to those of subordinates in the group, who tend to have unusually high levels of basal cortisol due to their lower social status. Particularly interesting is the ease with which these findings can be generalized to the human condition. In the human world, lower social status, such as a reputation for engaging in illicit behaviors (Decker, 2000) or a lower grade job (Steptoe et al., 2003), are also associated with elevated basal cortisol levels. In experimental settings, the Trier Social Stress Test (TSST) has been shown to be a reliable psychosocial stressor for humans (Domes, Heinrichs, Reichwald, & Hautzinger, 2002; Kuhlmann, Piel, & Wolf, 2005; Wolf, Schommer, Hellhammer, McEwen, & Kirschbaum, 2001; Wolf, Schommer, Hellhammer, Reischies, & Kirschbaum, 2002). This stressor is comprised of

the most profound type of human psychosocial stressor, speech delivery and mental arithmetic tasks performed in front of an audience. These tasks can be very stressful because they lack predictability and controllability (DeVries et al., 2003). Thus, this experimental manipulation has been shown to reliably and effectively elevate cortisol levels (Domes et al., 2002; Kuhlmann et al., 2005; Wolf et al., 2001; Wolf et al., 2002). Following the TSST, salivary cortisol levels can remain elevated near the peak for 15 to 20 minutes before steadily returning to baseline 30 to 45 minutes following the cessation of stress.

Cortisol and Memory

As stated above, the stress-related rise in cortisol is thought to be an important adaptive response, mobilizing energy to specific tissues, thus enhancing our ability to confront or cope with the stressor. According to the literature, stress hormones divert blood glucose, or “fuel,” to exercising muscles, and it can also reduce the amount of fuel that reaches the brain's learning and memory center, the hippocampus. This ultimately compromises the ability of the hippocampus to create new memories (Wolf, 2003). This has been widely reported in animal studies, where various forms of stress have been shown to impair hippocampal-dependent spatial memory (Song, Che, Min-Wei, Murakami, & Matsumoto, 2006). In rodents, stress in the forms of learned helplessness and chronic mild stress have been shown to elevate corticosterone (i.e., mouse and rat form of cortisol) and impair spatial memory in the Morris Water Maze task. Both learned helplessness and chronic mild stress are stressors typically used in rodent studies. In the learned helplessness model, mice are subjected to a series of inescapable foot shocks for

15 seconds per day for three days and tested for memory. With the chronic mild stress model, mice are subjected to environmental stressors such as tilted cage, limited food, soiled cage, separated housing, overnight illumination, and reversed light/dark cycles for 5 weeks before they are tested for memory. Mice that have been exposed to either type of stressor showed hippocampal-dependent spatial memory impairment in the Morris Water Maze task, such that the time required for escape from the water-submerged maze is lengthened for stressed mice. In stress research involving animals, it is very common for animals to endure longer periods of stress compared to research involving humans, which would not be as practical. Human research involving stress typically does not require participants to complete more than a few sessions in order to obtain baseline and peak measurements for cortisol levels and memory performance. Nonetheless, similar to animal studies, stress in humans has also been reported to impair hippocampal-dependent memory. In research where stress is experimentally induced via exogenous cortisol administration such as hydrocortisone pills (Newcomer, Craft, Hershey, Askins, & Bardgett, 1994; Newcomer et al., 1999) or with a psychosocial stressor, such as speech delivery (Elzinga, Bakker, & Bremner, 2005; Kirschbaum, Wolf, May, Wippich, & Hellhammer, 1996; Kuhlmann et al. 2005; Takahashi et al., 2004), the stress response has been shown to impair declarative memory, another hippocampal-dependent form of memory dealing with factual information.

Inconsistencies in the Literature

While most studies report hippocampal-dependent memory impairment with elevations of cortisol (Elzinga et al., 2005; Kirschbaum et al., 1996; Kuhlmann et al.,

2005; Takahashi et al., 2004), it should be noted that some studies also report the enhancement of memory (Abercrombie, Speck, & Monticelli, 2006; Domes et al., 2002), or no change in memory at all (Wolf et al., 2002; Rohleder, Wolf, Kirschbaum, & Wolf, 2009). There are a number of explanations for these conflicting findings. One theory involves the time of day in which cortisol sampling took place. Cortisol secretion is high in the morning, lower in the afternoon, and at its lowest in the evening, and many reports recommend sampling cortisol during the afternoon to avoid the high baseline levels associated with the morning hours, and the low baseline levels associated with the evening hours (Kudielka, Schommer, Hellhammer, & Kirschbaum, 2004). Indeed, studies conducted during the afternoon hours (e.g., 2pm to 6pm) have more consistently documented elevated cortisol and hippocampal-dependent declarative memory impairment (Elzinga et al., 2005; Kirschbaum et al., 1996; Newcomer et al., 1999; Takahashi et al., 2004). Conversely, studies conducted during the morning hours have reported inconsistent results, which varied from stress-induced memory impairment (Kuhlmann et al., 2005; Maheu, Collicutt, Kornik, Moszkowski, & Lupien, 2005; Wolf et al., 2001), memory non impairment (i.e., no effect on memory) (Wolf et al., 2002), to memory enhancement (Domes et al., 2002). In sum, the time of day in which cortisol is sampled is a critical consideration when interpreting findings with stress and memory.

Another factor that may have yielded inconsistent findings for stress and memory involves the time at which cortisol has been sampled following the stressor. The reported times for peak cortisol concentrations range anywhere from 10 to 20 minutes following a psychosocial stressor (Kirschbaum et al., 1996). However, Kirschbaum, Pirke, and

Hellhammer (1993) have demonstrated consistently that cortisol reliably peaks 10 minutes post-stressor. Indeed, although not uniformly adhered to, the consensus for the cortisol sampling protocol appears to be 10 minutes poststress for peak cortisol measurements (Domes et al., 2002; Elzinga et al., 2005; Kirschbaum et al., 1996; Wolf, et al., 2002).

Another timing issue in the stress and memory literature involves the time during which the delayed recall test takes place. Most tests are performed within one hour after the stressor; however, there are reports of days or even weeks of delayed recall testing following the stressor (Kirschbaum et al., 1996; Newcomer et al., 1994; Wolf et al., 2002). Some studies report that when delayed memory was tested within an hour following the stressor, both memory impairment (Kirschbaum et al., 1996) and memory enhancement (Nater et al., 2007) occurred. Other studies in which memory testing took place days or weeks later found memory impairment (Newcomer et al.), or no change in memory (Wolf et al.), respectively. There has also been reported variability in findings of stress-induced cortisol as a function of gender. Wolf et al. (2001) found a significant negative correlation between cortisol and memory for men only, such that the larger the increase in cortisol, the lower the number of words correctly recalled. This relationship was not significant for women. Men exhibited a 1.5 to 2 fold higher cortisol responses following psychosocial stress compared to women even when both men and women began testing with comparable baselines (Kirschbaum, Wust, & Hellhammer, 1992). One possible explanation for men having a more robust stress response than women is that men naturally have higher levels of testosterone, a hormone that stimulates the HPA

axis on a regular basis (i.e., produce cortisol) (Kirschbaum, Kudielka, Gaab, Schommer, & Hellhammer, 1999). If the HPA axis is already primed to produce cortisol by naturally occurring higher testosterone levels, the more testosterone one has, the more primed they are. Adding stress would undoubtedly contribute to even higher levels of cortisol. This priming effect in addition to the stress-induced elevation in cortisol may explain why men show such a robust stress response compared to women. Although women produce testosterone, the amount produced is extremely low compared to the amount produced in men.

Another gender-specific factor involves a woman's menstrual cycle fluctuations (Elzinga et al., 2005; Kirschbaum et al., 1996; Newcomer et al., 1999), a factor that has been reported to affect cortisol output, and one that is rarely controlled (Kirschbaum et al., 1999). Kirschbaum et al. (1999) examined salivary cortisol differences among women in the luteal phase, women in the follicular phase, and healthy men. In this study, poststress salivary cortisol levels were comparable between men and women in the luteal phase. Women in the follicular phase showed substantially lower peak cortisol levels compared to women in the luteal phase or to men. Indeed, most studies investigating stress-induced memory impairment utilize only men to circumvent the hormonal fluctuations associated with the menstrual cycle in women (Abercrombie et al., 2006; Kuhlmann et al., 2005; Nater et al., 2007; Takahashi et al., 2004).

Preexisting Social Support as a Mitigating Factor of Stress

Social support is a well-accepted mitigating factor of stress on overall health and well-being (Cohen et al., 1985). There is no single definition for social support, and it

may be defined in many ways depending on how researchers conceptualize the construct. Social support has been measured as the various types (e.g., instrumental, informational, emotional), sources (e.g., friends, family), and quality and quantity of social support perceived and/or received. Regardless of how it is assessed, the vast majority of the literature on social support strongly supports its buffering effects on the negative effects of stress on overall well-being (Cohen & Hoberman, 1983; Cohen & Wills, 1985; Gerin, Milner, Chawla, & Pickering, 1995; Nezlek & Allen, 2006). Even in some species of rodent and nonhuman primates, affiliative behaviors have been linked to overall health and well-being (DeVries et al., 2003). Indeed, basal cortisol levels of female tufted ear marmosets remain stable when separated from their mother and housed with a sibling compared to separation from its mother and housed alone. Affiliative behaviors such as grooming can decrease sympathetic activity and increase oxytocin release, a hormone linked to suppressing the negative adrenocortical effects of stress on the body (Heinrichs, Baumgartner, Kirschbaum, & Ehlert, 2003). Similar beneficial effects of social support have also been observed in humans.

Kirschbaum, Klauer, Filipp, and Hellhammer (1995) showed that men receiving verbally supportive statements from their girlfriend in preparation of a stress task showed an attenuated cortisol response compared to men who received social support from a stranger or no social support at all. In fact, Hale et al. (2005) reported that a sense of belonging, one specific domain of social support, predicted better perceptions of health in women and fewer physical symptoms in men.

There is ample support for the mitigating effects of social support on immune function, disease prognosis, and overall well-being (Cohen et al., 1997; Cohen et al., 2004; Hale et al., 2005; Koh et al., 2006; Miyazaki et al., 2005). However, to our knowledge, whether or not social support mitigates the effects of stress on memory loss has not yet been investigated. Therefore, the aim of the present study was to examine the relationship between stress and social support, controlling for previously stated potential confounds; we also sought to determine whether or not gender plays a role in this relationship. Based on previous reports, it was predicted that acute stress would negatively affect hippocampal-dependent memory, and we expected that social support would buffer this effect. Furthermore, because women tend to seek out social support more than men (Ashton & Fuehrer, 1993), we predicted social support to differ by gender, and that this difference would offset the negative effects of stress on memory. That is, we expected women to have high levels of social support, to be less affected by stress, and to perform better on the memory task than men or women with low levels of social support. As an experimental check, we wanted to ensure any differences in cortisol responses following stress was not due to perceived stress levels at the start of the study. We expected no differences in perceived stress levels for the control and stress groups.

Methods

Participants

One-hundred thirty-four undergraduate college students participated in the study for course participation credit. Participants were semi-randomly assigned to one of two groups: stress or control. Assignment to either group was based on the timeslot in which the participant signed up for the study. Because more women than men signed up for the study and assignment was based on students' as well as the researchers' availability, we could not completely randomize participants to balance for condition or gender. Sixty-two participants were excluded from statistical analyses a priori due to non-compliance with experimental constraints. Participants were asked to refrain from specific behaviors to qualify for participation. We asked that participants refrain from any strenuous physical exercise, smoking, and consuming large meals or acidic beverages at least one hour prior to the study. These restrictions were made explicit because these behaviors can artificially alter cortisol levels. Part of the demographic questionnaire involved a check for adherence to these experimental restrictions, and the results from these were that twenty-eight participants violated these restrictions and were excluded from the study. Twenty-three participants were "non-responders" and were excluded from the study. In other words, they did not show a minimum 10% increase in saliva cortisol following the TSST, which is the conventional criteria for inclusion in stress research (Kirschbaum et al., 1996). Eleven participants were also excluded from the study due to chronic inflammatory illnesses (e.g., hyperthyroidism) or having irregular or no menstrual cycles, known confounding variables for cortisol sampling (Kirschbaum et al.,

1999). The menstrual cycle has an average of 28 days. The first half (i.e., days 1 through 14) of the cycle is the follicular phase and the second half of the cycle (i.e., days 15 through 28) is the luteal phase. Only women who reported in the demographics questionnaire to be in the follicular or luteal phase was considered to have a normal menstrual cycle (i.e., menstruate monthly) and thus were included in the study. The final participant pool included 72 students (44 females and 28 males) with a mean age of 20.86 years ($SD = 5.53$). Sixty-one percent were women and 39% were men. Participants were 34% Asian, 27% Hispanic, 22% Caucasian, 4% African American, and 13% mixed. The control group was comprised of 26 women and 16 men, and the stress group was comprised of 18 women and 12 men. It should also be noted that this study was approved by the Institutional Review Board Ethics Committee at San Jose State University.

Procedure

This experiment was conducted in two separate research suites. One suite served as the meeting place and data collection for the stress group, and the second suite was reserved for the control group. Each participant was individually tested for a maximum of one and a half hour in one of the two suites. Data were collected between 1pm to 5:30pm to minimize the effects of cortisol fluctuations throughout the day. After participants provided informed consent, they recalled a wordlist (see below for a detailed description), and then participated in the TSST (experimental group) or viewed a travel video (control group). Following the stressor or travel video, participants filled out a survey packet assessing preexisting social support, perceived stress, and demographic

information. Perceived stress was also assessed to ensure that perceived existing stress did not affect variability in baseline cortisol levels. Experimenters were gender-balanced for both the stress and control conditions to control for experimenter and gender bias. The timeline for the experimental protocol is presented below in Table 1.

Cortisol Sample Collection

Saliva samples were collected using the Salivette (Sarstedt, Inc., Newton, NC), a saliva collection device in which participants chewed on a small cotton cylinder designed to absorb saliva. Once collected, samples were stored in a -80 Celsius freezer. Cortisol from the saliva samples was measured via enzyme-immunoassays (Salimetrics, LLC, State College, PA), a technique that is able to detect and quantify salivary cortisol, and has been used in other studies (Abercrombie et al., 2006). Five samples were collected for each condition (see Table 1).

Table 1. Protocol timeline for control and stress conditions in 10 minute intervals.

Group	Social Support							
	Free	Experimental	Perceived Stress		Delayed			
	Recall	Manipulation	Survey		Recall	Debrief		
Control	C1	C2	Travel video		C3	C4	C5	
	0	10	20	30	40	50	60	70
Stress	C1	C2	TSST		C3	C4	C5	

Note. C = Cortisol sample, TSST = Trier Social Stress Test.

Stress Manipulation

Trier Social Stress Test (TSST). The TSST (Kirschbaum et al., 1993) is a moderate laboratory inducer of the stress response (i.e., increase in cortisol levels). This stress challenge consisted of a 5 minute speech preparation for a job talk, a 5 minute speech about the job talk, and a 5 minute mental arithmetic task (i.e., serially subtract 13 from 2,083) in front of two women judges.

Coding

Gender. Undergraduate students (44 women, 28 men) were recruited and included for data analyses in the present study. Women were coded as “1” and men were coded as “2.”

Measures

Preexisting Social Support. Social support was assessed using the College Student Social Support Scale (CSSSS) (McGrath, Gutierrez, & Valadez, 2000). The CSSSS is a 26-item, self-report, 5-point, Likert-type scale assessing preexisting availability, helpfulness, and frequency of social support received from friends and from family. Internal consistency of 0.96 for the overall scale was based on a sample of college students from a mid-sized public university in the Midwest. Internal consistency for the present study’s sample is 0.95. A sample item includes “My friends listen to me talk about issues related to school.” The questionnaire took approximately 10 minutes to complete. A median split was utilized to separate global social support into a low and high group for data analyses.

Perceived Stress. Perceived stress was assessed with the Hassles and Uplifts Scale (Delongis, Folkman, & Lazarus, 1988), a 53-item, self-report, 4-point Likert type scale (0 = *None or not applicable*, 3 = *A great deal*). Each item assessed how much of a hassle an item was the previous day. A sample item included “Your parents or parents-in-law.” The questionnaire has a Cronbach’s alpha of 0.91 (as reported in Mayoral, 2006). The questionnaire took about 5 minutes for completion.

Memory Performance. Using the Max R. Colheart Psycholinguistic Database (Wilson, 1988), a commonly used online dictionary for research, 20 words were randomly selected and used for assessment of declarative memory. Parameters for the words were as follow: between four and eight letters, having one to two syllables, and contained moderate familiarity and concreteness in the English language. Memory performance was defined as difference scores for words forgotten (i.e., number of words correctly remembered at immediate recall minus number of words correctly remembered at delayed recall).

Results

It should first be reiterated that of the 134 participants solicited and tested, 62 were excluded from this study due to putative confounding variables. As previously stated, these included various violations in experimental restrictions and non-cortisol responsivity. With these exclusions in mind, it is important to note that the relatively small sample may produce attenuated results.

Previous research conducted by Kirschbaum et al. (1999) have shown a difference in poststress cortisol levels for women in the follicular and luteal phases; however in our study, no differences were observed in poststress difference scores (i.e., peak cortisol concentration minus baseline) for women in the follicular ($M = 4.50$, $SD = 4.32$) compared to women in the luteal phase ($M = 3.22$, $SD = 2.30$), $t(16) = 0.62$, $p = .54$. Therefore, for this study, both groups of women were combined and included for data analyses. All analyses were conducted with Type I error at the rate of .05.

We tested the efficacy of the stress challenge, and we also tested that peak cortisol would be evidenced at 10 minutes poststress, as previously reported (Kirschbaum et al., 1993). To test these, a subset of 18 randomly chosen participants' samples was assayed to develop a cortisol profile to determine baseline and peak collection time. A subset rather than the entire sample was assayed due to financial constraints. Figure 1 shows mean salivary cortisol concentrations at the five time points for the control and stress conditions. Sample 1 was collected 10 minutes before the stress challenge, shortly after arrival at the testing suite (i.e., baseline). Samples 2 and 3 were collected immediately before and after the stress challenge, respectively. Sample 4 was collected 10 minutes

poststress challenge (i.e., peak). Finally, Sample 5 was collected 30 minutes poststress challenge. This subset of data originally utilized four control participants, but only when they were analyzed was it realized that one was an outlier and the other could not be included due to non-compliance with study criteria. We analyzed a larger subset of stress participants relative to controls because of an anticipated, yet unknown portion of non-responders we may need to exclude from data analyses. Fortunately, the 16 stress participants we included to test baseline and peak levels showed the expected trend for stress-induced cortisol and so were included for further data analyses. Again, the purpose of this subset of data analysis was to establish baseline and peak time points for further analyses. As can be seen, samples obtained approximately 10 minutes before stress and 10 minutes after stress were determined as cortisol baseline and peak, respectively. Consequently, for the remaining participants' samples, only baseline and peak collection time points were analyzed.

All subsequent analyses regarding cortisol were conducted using the averaged difference score between baseline and peak cortisol concentrations (i.e., delta cortisol). We took the difference score (i.e., peak minus baseline) for each participant and averaged them into one score for the control group and one score for the stress group to rule out individual differences in baseline variability.

Figure 1. Mean salivary cortisol concentrations at each time point from a subset of control (n= 2) and stress (n=16) participants.

As expected, there were no differences in baseline cortisol levels between the control ($M = 5.21$ $SD = 4.30$) and stress ($M = 4.75$ $SD = 5.94$) groups, $t(70) = 0.24$, $p = .70$. In other words, both groups had comparable cortisol levels at the start of the study. Results from the total sample were that cortisol concentrations became elevated 10 minutes following stress. As depicted in Figure 2, the stress group ($M = 5.89$, $SD = 6.05$) showed substantially higher delta cortisol levels compared to the control group ($M = -1.22$, $SD = 1.60$) following psychosocial stress, which is consistent with the literature. This finding confirmed the effectiveness of the TSST, as evidenced by the outcome of a one-way analysis of variance (ANOVA). The delta cortisol scores were significantly different for the control and stress groups, $F(1,70) = 53.23$, $p < .001$.

Figure 2. Mean delta cortisol for control and stress groups. Vertical lines represent standard error of the means. * $p < .001$.

Given our findings with cortisol elevations in the stress group, we would expect to find memory to be impaired in the stress relative to the control groups. However, our results did not show a significant difference in declarative memory impairment between stress and control groups, $F(1,70) = 2.46, p = .12$. In fact, there was a slight trend for enhanced memory in the stress group. In other words, the control group ($M = 1.90, SD = 1.54$) forgot slightly more words than the stress group ($M = 1.40, SD = 1.00$), although this difference was not statistically significant (see Figure 3).

Figure 3. Declarative memory performance for control and stress groups. Vertical lines represent standard error of the means. $p = .12$.

We also predicted that preexisting social support would attenuate the stress response. We utilized a median split method for stress participants scoring in the lower ($n=15$) and upper ($n=15$) half of the global social support continuum to examine high and low social support effects on stress and to also maximize the number of participants per group. Results showed no differences in the stress response (i.e., delta cortisol) for participants with low ($M = 5.82, SD = 6.77$) or high ($M = 5.97, SD = 5.47$) levels of preexisting social support, $F(1,28) = 0.004, p = .95$. We also expected preexisting social support to buffer the negative effects of stress on memory. However, stressed participants with low ($M = 1.40, SD = 0.99$) or high ($M = 1.40, SD = 1.06$) levels of preexisting social support did not differ significantly in memory performance. That is, preexisting social support did not improve memory, $F(1,70) = 0.41, p = .52$. (see Figure 4), and this would be expected given the finding that social support did not moderate the cortisol response.

Figure 4. Effects of preexisting social support on stress and declarative memory performance. Vertical lines represent standard error of the means. $p = .52$.

Baseline cortisol levels did not differ between men ($M = 5.97, SD = 8.16$) and women ($M = 3.94, SD = 3.92$) in the stress condition, $t(28) = .91, p = .37$. However, peak levels did differ between men and women following psychosocial stress. Consistent with the literature, stressed men showed approximately twice the amount of cortisol elevation ($M = 8.52, SD = 7.82$) compared to stressed women ($M = 4.15, SD = 3.85$), and this was statistically significant, $F(1,28) = 4.16, p < .05$ (see Figure 5).

Figure 5. Delta salivary cortisol differences by gender. Vertical lines represent standard error of the means. * $p < .05$.

Consistent with predictions, women ($M = 3.84$, $SD = 0.50$) had significantly higher levels of preexisting social support compared to men ($M = 3.54$, $SD = 0.10$); $F(1,70) = 5.93$, $p < .05$, but this factor did not benefit women more in their declarative memory performance, $F(1,42) = 1.46$, $p = .24$. This finding is not consistent with our original prediction; however, it is consistent with our results for social support for the group at large.

Finally, perceived stress was assessed to ensure that preexisting stress did not contribute to variability in cortisol levels. The logic being that if levels of perceived stress were elevated at the experiment's onset, participants might exhibit a more robust cortisol response following the acute stressor. To this end, we assessed the role of perceived stress in the cortisol responses. Results confirm that cortisol reactivity following the stress challenge did not differ significantly for participants with either low ($M = 6.53$, $SD = 6.81$) or high levels of perceived stress ($M = 5.16$, $SD = 5.20$), $F(1,28) = 0.38$, $p = .54$.

Furthermore, just as social support was expected to mitigate a stress response (i.e., cortisol elevation) to the TSST, it seemed reasonable to expect that higher levels of social support would buffer self-perceived, daily stress. However, results did not support differences between daily stress levels in those with high levels of social support ($M = 1.71$, $SD = 0.38$) versus those with lower levels of social support ($M = 1.68$, $SD = 0.33$), $F(1,28) = 0.10$, $p = .75$. However, differences did emerge when social support and gender were both taken into consideration. Contrary to our predictions, men were more affected than women by social support. For example, men with high levels of preexisting

social support reported higher levels of perceived stress ($M=1.90$ $SD = 0.36$) compared to men with low levels of preexisting social support ($M=1.60$, $SD = 0.28$). Unexpectedly, there were only significant effects for social support in men, $F(1,27) = 4.99$, $p < .05$ (see Figure 6).

Figure 6. Differences in perceived stress and preexisting social support in men.

Vertical lines represent standard error of the means. * $p < .05$.

Discussion

Stress is the body's natural response to a threat or change. Often termed "fight or flight," this is an adaptive mechanism that mobilizes glucose to muscle cells so that a rapid retreat is possible when necessary. Elevations in the neurochemical, cortisol, make this response possible, but cortisol may also contribute to reduced energy flow to the brain, impeding the processing and storage of information. Thus, although the stress may be an adaptive response to an acute stressor, the rise in cortisol may also be detrimental to the hippocampus, the learning and memory center of the brain. More specifically, the stress response may impair declarative memory and the consolidation of short-term memory into long-term memory. Numerous factors have been suggested to mitigate the negative effect of stress on memory, of which social support has gained considerable and sustained attention. The purpose of this study was to show the effects of stress on hippocampal-dependent declarative memory, and to determine the role preexisting social support plays in mitigating this relationship. The type of stress examined in our study was psychosocially derived. We utilized a psychosocial stressor because it is reported to be particularly effective. For the most part, research shows that psychosocial stress impairs declarative memory (Wolf, 2003), and the most commonly used psychosocial stress paradigm is the TSST because it reliably elevates cortisol.

Numerous studies have reported that social support buffers the deleterious effects of stress (Nezlek & Allen, 2006). However, whether and to what extent social support buffers the effects of stress on memory had not been previously assessed. The present study was to determine whether the memory of stressed individuals with high levels of

social support would be less affected compared to stressed individuals with low levels of social support. Furthermore, because women are reported to differentially receive and benefit from social support (Kirschbaum et al., 1995), we predicted that women would be less affected by the psychosocial stressor.

The present study has meticulously controlled for potentially, confounding variables, and we reasoned that by doing so, our study would clarify inconsistencies in the literature. We expected to report that stress reliably impairs declarative memory performance. For example, we controlled for cortisol sampling concerns. Samples were collected during the afternoon period to control for aberrant cortisol fluctuations. Peak cortisol samples were collected at the optimal time point following stress (i.e., 10 minutes poststress). Participants violating the exclusion criteria prior to participation in the study were excluded. Participants with medical conditions that would undoubtedly affect cortisol levels (e.g., Cushing's Disease) were also excluded from the study. Stress participants who failed to show a conventional 10% increase in cortisol levels following the stress challenge were also excluded. Finally, women having irregular or no menstrual cycles were also excluded from the study. In other words, we identified these variables and corrected for them by excluding these participants' data from statistical analyses. We also utilized the TSST to induce stress because it is reported to effectively impair memory. Nonetheless, and despite our efforts, the results in this study were inconsistent with the majority of the literature that shows stress impairs declarative memory performance. Although we were able to demonstrate successful induction of stress with the TSST, as evidenced by significant cortisol elevations, we did not observe

corresponding impaired declarative memory performance. Our results did not support the predicted relation between stress and memory. One explanation for this finding may have been that cortisol levels in this study, although significantly elevated, were not sufficiently elevated to impair memory. The mean baseline ($M = 4.75$ nmol/L, $SD = 5.94$) and peak ($M = 10.65$ nmol/L, $SD = 9.87$) cortisol concentrations in the present study were considerably lower relative to those reported previously by Kirschbaum et al. (1996), whose average baseline ($M = 8.46$ nmol/L, $SEM = 1.02$) and peak ($M = 17.65$ nmol/L, $SEM = 2.17$) cortisol values were nearly twice as high using the TSST. Other studies using the TSST reporting impaired declarative memory also showed cortisol elevations that were at least two times higher than those we observed (e.g., between 18-22 nmol/L) (Kuhlmann et al., 2005; Takahashi et al., 2005). Unexpectedly, our relatively low cortisol levels not only yielded nonsignificant increases in the words forgotten during our memory task, but these levels appeared to slightly facilitate declarative memory. That is, the stressed group remembered slightly more, albeit not significantly, words compared to the control group (i.e., $p = .12$).

One explanation for our findings with cortisol and memory could well be that the absolute levels of peak cortisol were not elevated enough. Although inconsistent with our predictions, stress-induced memory enhancement is not inconsistent with other studies that have reported cortisol elevations to improve declarative memory performance (Abercrombie et al., 2006; Domes et al., 2002). Although not intuitive at first glance, this outcome makes sense from both adaptive and evolutionary perspectives. It seems reasonable that a situation that creates a stress response should be well remembered, lest

the organism becomes doomed to revisit the stress-creating circumstance. For example, when an organism confronts an environment with predators, it would benefit the organism, and promote survivorship, if it were to remember and avoid the dangerous location. However, it also seems reasonable that excessive cortisol could make it difficult to process or retrieve information, as evidenced when people become confused in a severe crisis. It may be that only extremely high levels of stress-induced cortisol impair memory. In the present study, it may have been that cortisol was not elevated enough to interfere with memory. With that said, it is important to note other factors may have also contributed to our unexpected findings.

One factor may have been the difficulty of the memory task utilized. In the present study, the words were randomly selected from an online generated list with a set of parameters. Words generated and randomly selected were between four and eight letters, having one to two syllables, and contained moderate familiarity and concreteness in the English language. The method for random selection of words to assess declarative memory, to our knowledge, has not been fully described in detail in past research. Thus, we only have an idea for the nature of words that past studies has utilized (e.g., rainbow). Other studies utilized between 24 and 30 words and participants remembered anywhere between 5 and 17 words, or 21 to 57% following the TSST (Kirschbaum et al., 1996; Kuhlmann et al., 2005; Takahashi et al., 2004; Wolf et al., 2001). In the present study, the absolute number of words recalled following stress was approximately six out of 20 possible words, a 32% rate well within the range of reported percentages. Based on the reported percentages for words recalled in the literature, it appears that the number of

words used in our study was a comparable number to test for recall. However, it could be that the words contained in the memory task in the present study may have been too difficult (e.g., verse, salute) such that it was harder to remember, creating a floor effect for both groups, such that no one group performed better than the other. It may also be that different tests assessing declarative memory may have differential effects on memory. For example, Kirschbaum et al. used a cued recall declarative memory test in which researchers cued participants with the first two letters of 24 nouns previously presented to them. In their study, higher cortisol response following stress was related to more memory impairment. In the present study, participants were presented the list of 20 words and were assessed for immediate free recall. Approximately one hour later, participants were asked to recall the words (i.e., delayed recall) without any cues. Another reason may be that the time elapsed between the stress challenge and the delayed recall test (i.e., 30 minutes) was too long of an interval for the effects of cortisol to take effect. Indeed, by this time, cortisol levels may likely have returned to baseline levels and stress-induced cortisol effects on memory could no longer be detected. In fact, Kirschbaum et al. (1996) reported declarative memory impairment when memory was assessed during peak cortisol levels (i.e., 10 minutes poststress). Thus, stress-induced changes in memory may also be sensitive to the time at which delayed recall testing took place.

An unexpected finding in the present study involved the effect of social support on stress. It now seems to be common knowledge that preexisting social support is both preventative and curative. Indeed, from the Mayo Clinic web site that reads, “The

bottom line: More friends, less stress” (Mayo Clinic, 2008, last paragraph) to the editorial in the prestigious American Journal of Medicine, “A friend, not an apple, a day will help keep the doctor away” (Eisenberg, 1979), the message of social support is that the more you have, the better it is. Therefore, we predicted that this study would reveal the benefits of social support to be transferable to the effects of stress on memory. We predicted that preexisting social support would buffer the effects of the TSST-induced stress on memory, and that women would benefit more than would men. Unexpectedly, we found no overall effect for social support on memory. More surprisingly, however, were our results that social support did not offset the effects of stress at all, neither those assessed by cortisol levels (i.e., TSST-induced) nor those self-perceived (i.e., daily stress). Also, women did not benefit more from social support than did men.

There are many different ways to assess social support. In the present study, we used a measure that quantified social support. We selected this measure because of its high reliability compared to others (McGrath et al., 2000). It may be that the social support scale used in the present study did not represent the different kinds of social support that has been reported to be beneficial. More specifically, the social support questionnaire utilized in the present study did not measure specific forms of social support known to buffer the effects of stress on overall health (Hale et al., 2005). This measure of preexisting social support may undermine the buffering effects of stress because men and women differentially benefit from tangible and emotional forms of social support, respectively (Reevy, & Maslach, 2001). While it may be true that having a strong, preexisting social support network is beneficial to overall health and well-being

as widely reported, it may not necessarily be a function of its effect on cortisol, but rather on other elements in the neurochemical cascade during stress.

On the other hand, it may be that if we had measured social support, in specific forms, can mitigate the effects of stress, but that it has to be directly manipulated social support. For example, after the TSST, men who received verbally supportive statements from their girlfriend showed attenuated cortisol responses compared to men who were not supported at all, or men who were supported by a stranger (Kirschbaum et al., 1995). In a more recent study by Quas and Lench (2007), the effects of directly manipulated social support on declarative memory were tested in 5 and 6 year old children. Researchers used heart rate as an indicator of stress while children individually watched a 2 minute fear-eliciting video and tested for declarative memory the following week. Direct manipulation of social support involved the manner in which the person assessing declarative memory (i.e., interviewer) carried out the assessment. They either built rapport, provided supportive comments and feedback, and expressed welcoming nonverbal cues (i.e., eye contact, smiles) while assessing memory, or interviewers assessed for declarative memory in the complete opposite manner for the non-supportive condition. They reported that children with increased heart rate during the video clip the previous week show impaired declarative memory retrieval in the non-supportive condition. Thus, it may be that preexisting social support does not buffer the effects of stress on memory, but that directly manipulated and specific forms of social support may do so.

We also found that social support differed by gender. It was not surprising that women tended to have higher levels of social support compared to men. So in other words, consistent with the literature, women sought more social support, but inconsistent with the literature, women did not benefit more from social support than did men. Again, a notable concern here is the social support measure utilized. It makes sense intuitively that women would have an overall higher level of social support compared to men. But similar to the non supportive results for the buffering hypothesis of social support on stress and memory, specific forms of social support and or its direct manipulation, may be more sensitive to stress-induced memory impairment than preexisting social support alone.

Because men showed twice the amount of cortisol elevation compared to women, and women had higher levels of preexisting social support, we would have expected men to have higher levels of perceived stress compared to women, especially if men had low levels of preexisting social support. The literature shows that social support is beneficial to the overall health and well-being of an individual. However, what we found is contrary to what the literature reports. In the present study, the more social support men had, the higher the perceived levels of stress. It could be that men perceiving higher levels of stress were seeking out more social support or that having higher levels of social support led to higher levels of perceived stress. In the present study, social support appears to buffer the effects of the stress response for women, but not for men. It could be that in different cultures, it is perfectly acceptable and expected for women to seek social support; but for men it is a sign of weakness so that the more men seek out social

support, the more they perceive it to be stressful because of what society deem that behavior to indicate. Thus, social support may not always serve to be beneficial as the research consensus reports, but as shown in the present study as unfavorable, especially in men. Finally, the method used to classify high and low preexisting social support (i.e., median split) inadvertently gave rise to this finding that may or may not be meaningful at all.

In sum, the present study is the first to examine preexisting social support and its effect on the relation between stress and memory. Regretfully, preexisting social support did not buffer the effects of stress on memory in the present study. In addition, no memory impairment was observed following the stressor. Several reasons may explain these findings. First of all, elevations in cortisol may not have been elevated enough to fully detect any memory-impairing effects of stress. Contrary to our prediction, results showed a trend toward the facilitation of memory in the stress group, which is not unheard of (Het, Ramlow, & Wolf, 2005). Second, the memory task used was not sensitive enough to detect stress-induced memory changes. Therefore, future investigations need to use a more robust memory task, or use multiple reliable measures of declarative memory. Third, the sample size was insufficient to detect differences in these relationships, and need to be increased in future studies while taking into consideration the fact that an unknown portion of participants will be excluded due to non compliance among other reasons (e.g., chronic inflammatory illnesses, irregular menstrual cycles). Fourth, the measure of social support used, although with superb reliability, was not used optimally to delineate the types of social support (e.g.,

instrumental, emotional) beneficial to men and women as research has reported (Reevy et al., 2001). Finally, future research should consider using multiple reliable measures of social support assessing general as well as specific types of social support known to be beneficial. These future efforts would not only support that the maintenance of a strong social support network is beneficial for overall health and well-being, but also for the preservation of cognitive integrity, especially when faced with an acute stressor.

References

- Abercrombie, H. C., Speck, N. S., & Monticelli, R. M. (2006). Endogenous cortisol elevations are related to memory facilitation only in individuals who are emotionally aroused. *Psychoneuroendocrinology*, 31(2), 187-196.
- Ashton, W. A., & Fuehrer, A. (1993). Effect of gender and gender role identification of participant and type of social support resource on support seeking. *Sex Roles*, 28(7/8), 461-476.
- Burg, M. M. (1992). Stress, behavior, and heart disease. In B. L. Zaret, M. Moser, & L. S. Cohen (Eds.), *Yale University School of Medicine Heart Book* (pp. 94-104). New York, New York: Hearst Books.
- Cohen, J. (1992). A power primer. *Psychological Bulletin*, 112(1), 155-159.
- Cohen, S., Doyle, W. J., Skoner, D. P., Rabin, B. S., & Gwaltney, J. M. (1997). Social ties and susceptibility to the common cold. *Journal of American Medical Association*, 277(24), 1940-1944.
- Cohen, A., Hammen, C., Henry, R. M., & Daley, S. E. (2004). Effects of stress and social support on recurrence in bipolar disorder. *Journal of Affective Disorders*, 82(1), 143-147.
- Cohen, S., & Hoberman, H. M. (1983). Positive events and social support as buffers of life change stress. *Journal of Applied Social Psychology*, 13(2), 99-125.
- Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, 98(2), 310-357.
- Decker, S. A. (2000). Salivary cortisol and social status among Dominican men. *Hormones and Behavior*, 38(1), 29-38.
- Delongis, A., Folkman, S., & Lazarus, R. (1988). The impact of daily stress on health and mood: Psychological social resources as mediators. *Journal of Personality and Social Psychology*, 54(3), 486-495.
- DeVries, A. C., Glasper, E. R., Detillion, C. E. (2003). Social modulation of stress responses. *Physiology and Behavior*, 79(3), 399-407.
- Domes, G., Heinrichs, M., Reichwald, U., & Hautzinger, M. (2002). Hypothalamic-pituitary-adrenal axis reactivity to psychosocial stress and memory in middle-aged women: High responders exhibit enhanced declarative memory performance. *Psychoneuroendocrinology*, 27(7), 843-853.

- Eisenberg, L. (1979). A friend, not an apple, a day will help keep the doctor away. *The American Journal of Medicine*, 66(4), 551-553.
- Elzinga, B. M., Bakker, A., & Bremner, J. D. (2005). Stress-induced cortisol elevations are associated with impaired delayed, but not immediate recall. *Psychiatry Research*, 134(3), 211-223.
- Gerin, W., Milner, D., Chawla, S., & Pickering, T. (1995). Social support as a moderator of cardiovascular reactivity in women: A test of the direct effects and buffering hypotheses. *Psychosomatic Medicine*, 57(1), 16-22.
- Hale, C. J., Hannum, J. W., & Espelage, D. L. (2005). Social support and physical health: The importance of belonging. *Journal of American College Health*, 53(6), 276-284.
- Harris, R. B. S., Gu, H., Mitchell, T. D., Endale, L., Russo, M., & Ryan, D. H. (2004). Increased glucocorticoid response to a novel stress in rats that have been restrained. *Physiology and Behavior*, 81(4), 557-568.
- Heinrichs, M., Baumgartner, T., Kirschbaum, C., & Ehlert, U. (2003). Social support and oxytocin interact to suppress cortisol and subjective responses to psychological stress. *Society of Biological Psychiatry*, 54(12), 1389-1398.
- Het, S., Ramlow, G., & Wolf, O. T. (2005). A meta-analytic review of the effects of acute cortisol administration on human memory. *Psychoneuroendocrinology*, 30(8), 771-784.
- Kalat, J. W. (2007). *Biological Psychology*. (Rev. ed.). Australia: Wadsworth.
- Kirschbaum, C., Klauer, T., Filipp, S. H., & Hellhammer, D. H. (1995). Sex-specific effects of social support on cortisol and subjective responses to acute psychological stress. *Psychosomatic Medicine*, 57(1), 23-31.
- Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. *Psychosomatic Medicine*, 61(2), 154-162.
- Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The "Trier Social Stress Test": A tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, 28(1), 76-81.

- Kirschbaum, C., Wolf, O. T., May, M., Wippich, W., & Hellhammer, D. H. (1996). Stress-and treatment-induced elevations of cortisol levels associated with impaired declarative memory in healthy adults. *Life Sciences*, 58(17), 1475-1483.
- Kirschbaum, C., Wust, S., & Hellhammer, D. (1992). Consistent sex differences in cortisol responses to psychological stress. *Psychosomatic Medicine* 54(6), 648-657.
- Koh, K. B, Choe, E., Song, J. E., & Lee, E. H. (2006). Effect of coping on endocrinoimmune functions in different situations. *Psychiatry Research*, 143(2-3), 223-234.
- Krantz, D. S., & McCeney, M. K. (2002). Effects of psychological and social factors on organic disease: A critical assessment of research on coronary heart disease. *Annual Review of Psychology*, 53, 341-369.
- Kudielka, B. M., Schommer, N. C., Hellhammer, D. H., & Kirschbaum, C. (2004). Acute HPA axis responses, heart rate, and mood changes to psychosocial stress (TSST) in humans at different times of day. *Psychoneuroendocrinology*, 29(8), 983-992.
- Kuhlmann, S., Piel, M., & Wolf, O. T. (2005). Impaired memory retrieval after psychological stress in healthy young men. *Journal of Neuroscience*, 25(11), 2977-2982.
- Maheu, F. S., Collicutt, P., Kornik, R., Moszkowski, R., & Lupien, S. J. (2005). The perfect time to be stressed: A differential modulation of human memory by stress applied in the morning or in the afternoon. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 29(8), 1281-1288.
- Mayo Clinic. (2008, July 23). Social support: Tap this tool to reduce stress. Retrieved May 31, 2009, from <http://www.mayoclinic.com/health/social-support/SR00033/NSECTIONGROUP=2>
- Mayoral, L. (2006). Exam stress, depression, social support, and sleep disturbances. *Masters Abstracts International*, San Jose State University, San Jose, California, United States.
- McGrath, P. B., Gutierrez, P. M., & Valadez, I. M. (2000). Introduction of the College Student Social Support Scale: Factor structure and reliability assessment. *Journal of College Student Development*, 41(4), 415-426.
- Miyazaki, T., Ishikawa, T., Nakata, A., Sakurai, T., Miki, A., Fujita, O., et al. (2005). Association between perceived social support and Th1 dominance. *Biological Psychology*, 70(1), 30-37.

- Nater, U. M., Moor, C., Okere, U., Stallkamp, R., Martin, M., Ehlert, U., et al. (2007). Performance on a declarative memory task is better in high than low cortisol responders to psychosocial stress. *Psychoneuroendocrinology*, 32(6), 758-763.
- Newcomer, J. W., Craft, S., Hershey, T., Askins, K., & Bardgett, M. E. (1994). Glucocorticoid-induced impairment in declarative memory performance in adult humans. *Journal of Neuroscience*, 14(4), 2047-2053.
- Newcomer, J. W., Selke, G., Melson, A. K., Hershey, T., Craft, S., Richards, K., et al. (1999). Decreased memory performance in healthy humans induced by stress-level cortisol treatment. *Archives of General Psychiatry*, 56(6), 527-533.
- Nezlek, J. B., & Allen, M. R. (2006). Social support as a moderator of day-to-day relationships between daily negative events and daily psychological well-being. *European Journal of Personality*, 20(1), 53-68.
- Quas, J. A., & Lench, H. C. (2007). Arousal at encoding, arousal at retrieval, interviewer support, and children's memory for a mild stressor. *Applied Journal of Psychology*, 21(3), 289-305.
- Reevy, G. M., & Maslach, C. (2001). Use of social support: Gender and personality differences. *Sex Roles*, 44(7/8), 437-459.
- Retana-Marquez, S., Bonilla-Jaime, H., Vazquez-Palacios, G., Dominguez-Salazar, E., Martinez-Garcia, R., & Velazquez-Moctezuma, J. (2003). Body weight gain and diurnal differences of corticosterone changes in response to acute and chronic stress in rats. *Psychoneuroendocrinology*, 28(2), 207-227.
- Rohleder, N., Wolf, J. M., Kirschbaum, C., & Wolf, O. T. (2009). Effects of cortisol on emotional but not on neutral memory are correlated with peripheral glucocorticoid sensitivity of inflammatory cytokine production. *International Journal of Psychophysiology*, 72(1), 74-80.
- Song, L., Che, W., Min-Wei, W., Murakami, Y., & Matsumoto, K. (2006). Impairment of the spatial learning and memory induced by learned helplessness and chronic mild stress. *Pharmacology Biochemistry and Behavior*, 83(2), 186-193.
- Steptoe, A., Kunz-Ebrecht, S., Owen, N., Feldman, P. J., Willemsen, G., Kirschbaum, C., et al. (2003). Socioeconomic status and stress-related biological responses over the working day. *Psychosomatic Medicine*, 65(3), 461-470.
- Takahashi, T., Ikeda, K., Ishikawa, M., Tsukasaki, T., Nakama, D., Tanida, S., et al. (2004). Social stress-induced cortisol elevation acutely impairs social memory in humans. *Neuroscience Letters*, 363(2), 125-130.

- Wilson, M. D. (1988). The MRC psycholinguistic database: Machine readable dictionary, Version 2. *Behavioural Research Methods, Instruments and Computers*, 20(1), 6-11.
- Wolf, O. T. (2003). HPA axis and memory. *Best Practice and Research Clinical Endocrinology and Metabolism*, 17(2), 287-299.
- Wolf, O. T., Schommer, N. C., Hellhammer, D. H., McEwen, B. S., & Kirschbaum, C. (2001). The relationship between stress induced cortisol levels and memory differs between men and women. *Psychoneuroendocrinology*, 26(7), 711-720.
- Wolf, O. T., Schommer, N. C., Hellhammer, D. H., Reischies, F. M., & Kirschbaum, C. (2002). Moderate psychosocial stress appears not to impair recall of words learned 4 weeks prior to stress exposure. *Stress*, 5(1), 59-64.