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The effects of experimentally induced psychological stress on memory recall

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The effects of experimentally induced psychological stress on memory-recall

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Abstract:

Numerous studies have established the relationship between stress and memory, particularly stress and its effect on memory retrieval. When we experience stress, there is a cascade of hormonal responses which are critical to helping the body cope with the stressful event. When the stressful event persists, levels of the hormone cortisol--a type of glucocorticoid increases in response to the stress and have wide-ranging effects on the body. In particular, cortisol can have an effect on brain cells, particularly the hippocampus which is responsible for cognitive activities such as learning and memory. In this study, we utilized a public speaking task as a stress-inducing technique. We predicted that stress would negatively impact the ability of college students to retrieve a list of words. Preliminary analyses indicate no significant difference for word-recall as a function of condition, M=12.55, SD=3.57 and M=12.45, SD=3.80. Future studies should refine the methodology and take into consideration the time-dependence of this phenomenon.

Introduction:

The interplay between stress and memory can have a profound effect on our daily lives. In the demanding society that we live in today we are overwhelmed by a flood of stress stimulating phenomena, including deadlines for projects and assignments in school and work, demands from family and friends, and other trials and tribulations of modern life which can

further exacerbate our stress level. The scientific literature can attest to the fact that these influences modulate the psychobiological response.

Our bodies have the uncanny ability to respond to stressful events and restore homeostasis. When we perceive stress, the adrenal glands, the first line of defense in the stress response system, are activated. The brain transmits the perceived stress and instructs the adrenal glands to release the hormone adrenalin. This hormone than initiates a biological chain reaction that triggers the uptake of glucose for energy, increased beat of the heart, and tension in the muscles. All these biological activities are designed to cope with the perceived stress; the glands release the chemicals into the bloodstream, and these chemicals in turn propel the body organs into action. When the perceived stress persists the adrenal gland is further instructed to release another glucocorticoid hormone—cortisol. Cortisol can remain in the brain much longer than adrenalin and can have a greater effect on brain cells, particularly that of the hippocampus which is responsible for learning and memory. This prolonged elevation in cortisol level can become toxic to hippocampal cells (Sapolsky, 1999).

When the brain perceives stress it stimulates (**see figure 1**) the hypothalamus to synthesis and releases CRH (corticotrophin releasing hormone), in turn, the CRH stimulates pituitary glands which results in the secretions of another hormone: ACTH (Adrenocorticotropin). ACTH stimulates the production of cortisol via the adrenal glands. This pathway results in the elevation of blood cortisol level, however, the cortisol in an effort to down-regulate its secretion will inhibit the secretion of the corticotrophin releasing hormone (CRH). When this feedback system is altered, the excessive level of cortisol caused by the excess level of the corticotrophin releasing hormone impairs the hippocampus due to its high level of cortisol receptors (McEwen, 1998).

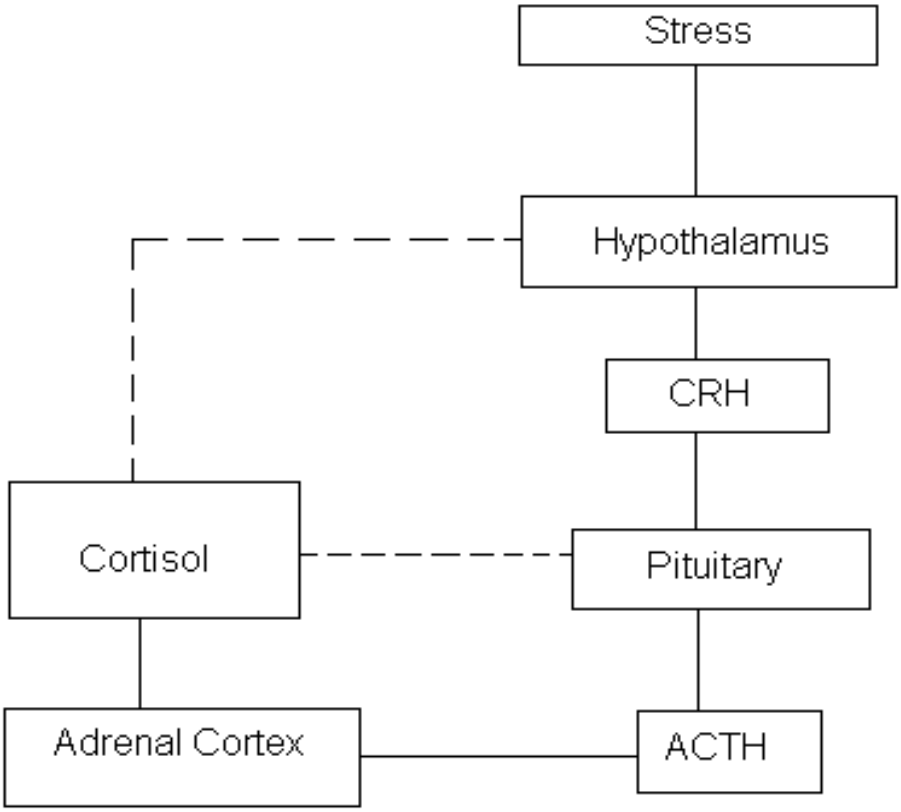

Figure 1: Negative Feedback system of the HPA-The feedback system depicts the secretion and down-regulation of cortisol as well as the regulation of secretion of CRH and ACTH. Cortisol can suppress the secretion of CRH from the hypothalamus and ACTH from the pituitary.

Experimental method utilizing Golgi-impregnated tissue (**see figure 2**) established that excess glucocorticoid can alter dentritic morphology in hippocampal neurons of adult rats (Woolley et al, 1990). Twenty-one days of daily injection of 10 mg of corticosterone resulted in decreased numbers of apical dentritic branch points and decreased total apical dentritic length relative to baseline. Elevation in corticosterone resulted in the structural alteration of the adult rat. Because the physiology of a rat is similar to our own, this suggests that humans might also suffer from the neurological effects of prolonged stress.

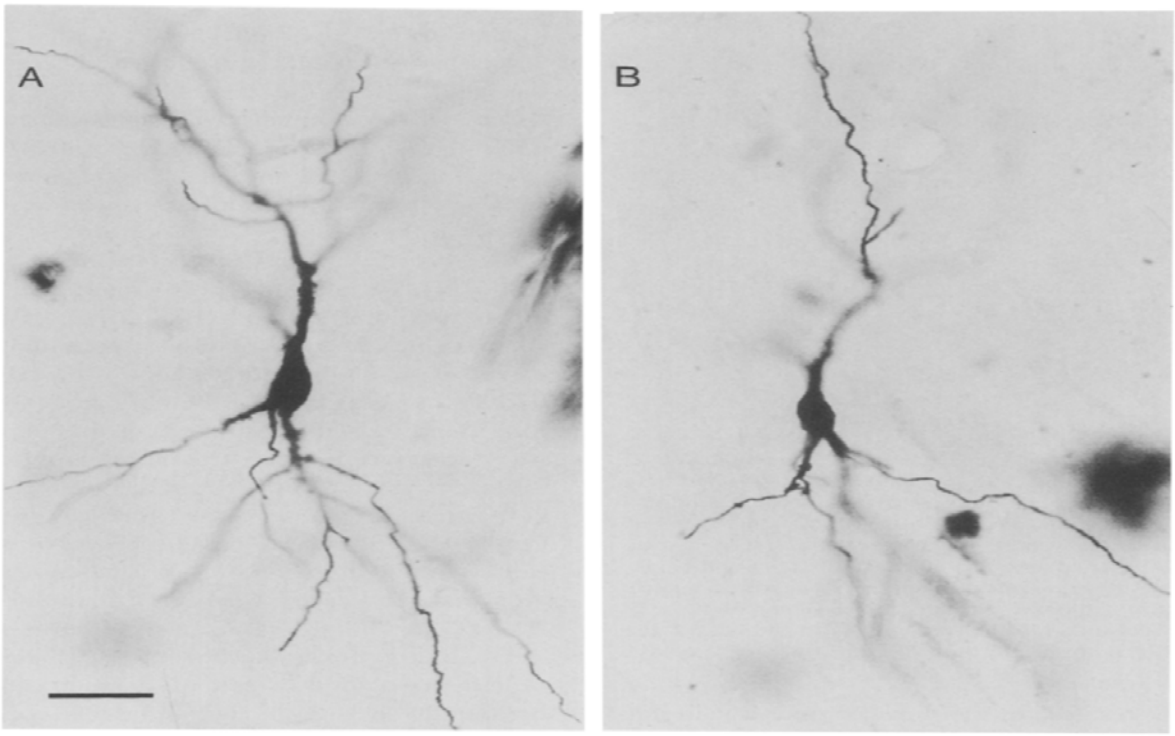

Figure 2: The images depict the two different brains of Golgi-impregnated cells. **Image A** depicts the brain of the control, whereas, **Image B** depicts the corticosterone injected. There is decrease in dendtritic number of branch points and apical dendritic length in Image B compared to Image A. Source: (Woolley et al, 1990)

In support of the extension of the animal research towards humans, research on Cushing's syndrome exemplifies the disruptive nature in which prolonged elevation in cortisol (hypercortisolism) can play in the brain. In a study conducted by Starkman and colleagues, patients with hypercortisolemia as a result of their Cushing's syndrome—demonstrated cognitive dysfunction when tested in a comprehensive neuropsychological examination (Starkman et al, 1992).

The deleterious effect of this hormone in the hippocampus is by way of atrophy of the dentritic processes, disruption of the neural pathways, neural degeneration and death (Sapolsky 1999). Brain imaging such as positron emission tomography (PET) revealed that cortisol directly influenced the uptake of glucose in the hippocampus (De Leon et al, 1997). Another brain

imaging technique that was utilized with a far more superior resolution quality was—functional magnetic resonance imaging (fMRI). In collaboration with researchers from the Netherlands, researchers from Germany were interested in testing the effects of cortisol on memory retrieval while utilizing the fMRI (Oei et al, 2007). Twenty one male subjects participated in the study consisting of a double-blind placebo design. The subjects were given 20mg of either hydrocortisone or a placebo, in which they were instructed to ingest one hour before the scanning in order to elevate cortisol level. The subjects than had to learn a list of words outside the scanner and recognition was performed inside. This study found that during the memory retrieval, there was a significant reduction in hippocampal activation in the brain of the subjects in the cortisol treatment condition (**see figure 3**). The result of the study suggests that cortisol affects the hippocampus by disrupting memory retrieval (Wolf 2009).

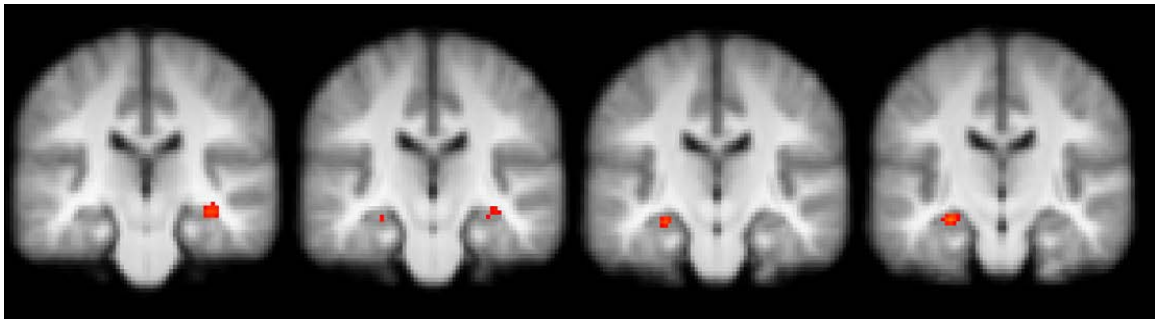

Figure 3: This image depicts the influence of cortisol on hippocampal activity during memory retrieval. (Source: Oei et al, 2007)

In another study conducted by de Quervain and colleagues showed how stress impaired memory retrieval in rats when tested in a water maze. The rats were initially trained inside the water maze to locate a platform that was submerged at a particular area; the rats were tested on whether they could remember the location of the platform following foot-shocks. The study found that non-stressed rats were able to locate the platform in the water maze through memory better than their stressed counterparts. Stress induced high level of glucocorticoids circulation in

the stressed-rats thereby, disrupting memory retrieval and their ability to locate the platform (de Quervain et al, 1998). Collectively, these results indicate that glucocoriticoids can impair memory retrieval relative to the basal levels which did not exert any adverse effects, as well as the HPA axis mediating the impairment in memory retrieval (Sapolsky 1999).

Extensive evidence can be found from the scientific literature on research conducted with animals and humans that indicate stress can have a profound adverse effect on cognitive process particularly that of memory retrieval, as a result of the over-secretion of glucocorticoids. Gerra (2001) and colleagues report elevated plasma concentrations of norepinephrine, epinephrine, adrencorticoorticotropic hormone, and cortisol when healthy subjects performed public speaking in addition to other cognitive task in front of a small group of audience.

Methodology:

Participants:

Students at Portland State University were recruited to participate in the research study. There were a total of 42 students ranging in grade level from sophomore-seniors. Out of the 42 students, 15 were male and 27 were female students. They were drawn predominantly from psychology courses, and they were offered course credit in exchange for participation. Participation in the study was voluntary for the students, if they decided not to participate in the study; they were given the opportunity to receive course credit by completing an alternative assignment that required the same time commitment as the research study.

Procedure:

The students were randomly divided into two groups, the control group and the experimental group. In the experimental condition, students were led to believe they would be giving a two-minute speech to a small audience who were going to evaluate the quality of their

presentation (stress-inducing task). They were led to a room with a one-way mirror and led to believe that the small audience who were going to evaluate their speech were stationed at the other end of the one-way mirror. The experimenter after explaining the purpose of the study and signing of the consent form from the student would immediately instruct the student to give a two minute presentation about a topic of their choice unrehearsed. The experimenter was present in the room with the student throughout the duration of the experiment. The experimenter timed the presentation and following its completion would instruct the student to stop and begin the next task. The control group did not perform the public speaking task, however, they were instructed to write a two-minute paragraph about a topic of their choice and that whatever they had written would not be read by anyone. Following the completion of the respective tasks, the students were instructed to complete a short questionnaire and the PSS (perceived stress scale). Following the completion of the questionnaire, the list of unrelated words from Mangels (1997) Strategic Processing and Memory for Temporal Order in patients with Frontal Lobe Lesions was utilized. The students were given 90 seconds to memorize the unrelated words. When 90 seconds elapsed the students were given a piece of paper with numbered lines in which the students had to write down as many of the words as they could recall within a three minute time limit. Finally, a debriefing session followed the completion of the memory task. The goal and purpose of the study was completely disclosed to the students and any questions they had about the study was answered.

Results:

Preliminary Analysis:

An analysis was conducted to ensure that the experimental manipulation did indeed result in participants believing they were being watched behind the one-way mirror. Results indicated

that significantly more students in the experimental condition relative to the control group (17 vs. 4) believed someone was watching them, χ^2 (2, N=42) =15.65.

Next, we tested to see whether there were significant differences in memory for words as a function of gender. Test of between-subject effects with regards to gender and word memory indicated that there were no significant differences in memory recall F (1, 42) =.702, p=.41, by gender. However, additional analyses **(see table 1)** indicated that there was a trend for female students to indicate higher levels of perceived stress than their male counterparts F (1, 42) = 6.43, p=.015.

Table 1:

Perceived Stress in Relation to Gender

Gender	Mean	Standard deviation	Sample Size
Male	14.87	5.81	15
Female	20.63	7.64	27
Total	18.57	7.51	42

Class standing in relation to word memory indicated that there was not a difference in how many words were remembered according to class standing. Therefore, class level was excluded from all additional analyses. These preliminary analyses collectively indicate that the stress induction (in the form of public speaking task) worked, and that gender and class standing did not play significant role in word recall.

Data Analysis:

First, we checked to see if participants remembered fewer words in the experimental condition than the control condition. However in contrast to predictions, we found no significant

differences in memory for words as a function of condition. *M*=12.55, *SD*=3.57 and *M*=12.45, *SD*=3.80 **(see figure 4)**.

Then, we examined the levels of perceived stress and by group condition **(see table 2).** Results showed that the experimental induction did not have a significant effect on levels of stress. Therefore, while the manipulation check suggested that students in the experimental condition did indeed believe there to be an audience behind the one-way mirror, this did not seem to affect their perceived levels of stress.

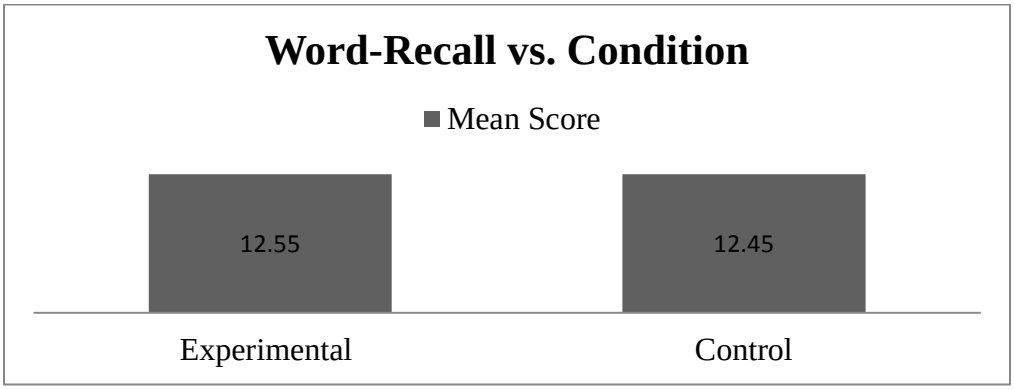

Figure 4: The graph represents the mean scores for word recall in both conditions.

Table 2:

Perceived Stress in Relation to Condition

Condition	Mean	Standard deviation	Sample Size
Experimental	19.1	6.85	22
Control	18.0	8.32	20
Total	18.57	7.51	42

Discussion:

We predicted that asking participants to engage in a public speaking task would result in an elevation in perceived stress, and that the rise in stress hormones would then lead to deficits in information processing, specifically a decreased number of words remembered in the experimental condition. However, our results indicate that our induction did not have any significant effects on number of words remembered. In other words, students in both condition (experimental and control) were able to recall about the same amount of words two minutes after being instructed to complete a task (either public speaking task or writing a paragraph). We suspect that the public speaking task was too mild to alter the short-term memory retrieval of students who were placed in the experimental condition, and that the timing of the memory task may have been too short to be affected by the induction.

The result of this study supports the findings from de Quervain et al (1998), and Gerra et al (2001), that the interplay between stress and memory is a time-dependent phenomenon. de Quervain and colleagues report that when the rats were given foot-shocks 2-minutes and 4-hours before navigating the platform--they did not show impairments in memory, however, when the foot-shocks were given 30-minutes before navigating the platform there was a significant impairment in memory. Gerra and colleagues also found elevated plasma concentrations 30-minutes after stress induction. They measured plasma concentrations of different hormones before and after the subjects performed the designated tasks (which included stress inducing tasks) and reported that there was a significant increase in the different hormones including cortisol 30-minutes after.

The duration of the stress induction we utilized was two-minutes and henceforth, did not impair the memory retrieval of the college students. Our preliminary analysis confirms that the actual induction worked; students did indeed believe our statements about an observer behind

the one-way mirror. However, the induction was not strong enough and lasting longer duration (2-minutes vs. 30-minutes) to adversely affect memory recall. The findings in this study relative to the aforementioned studies would therefore suggests that as the plasma concentration of the hormones for the students in the experimental condition was rising—it did not reach its peak soon enough (about 30-minutes) to negatively impact their short-term memory retrieval.

Another reason why our stress induction did not negatively impact memory could be due to the stress-induction we utilized. Our stress-induction (public speaking task) was possibly too mild to elevate the plasma concentrations of the hormones to affect memory. For instance, in the study conducted by Gerra and colleagues, the public speaking task they utilized that resulted in elevation of cortisol level in the subjects, was based on having the subject stand at a microphone and speak for 10 minutes in the presence of three people who had a video-camera and a tape recorder. In addition to the duration of the task, the presence of the three people with the objects at hand further exacerbated the subjects stress level.

Future research should include a larger sample size, as well as taking into consideration the time-dependence of this phenomenon and students should be drawn from other disciplines as well. Students in this study were drawn predominately from psychology courses that often times require their students to participate in class discussion; therefore, a task involving public speaking might not be as stressful to them as it might for other students in other disciplines. Another benefit of having students from multiple disciplines is that it would eliminate the possibility of the students sharing the study with their peers. For example, if a student participates in the study and debriefing session follows in which the full extent of the research study is disclosed to the student, they might possibly share that information with their peers who also decide to participate in the study. It is of paramount importance to understand the interplay

between stress and memory, and in particular how stress induction (in form of public speaking) can affect the memory of college students so that methods can be devised to mitigate this condition.

References:

1. Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, *24*, 385-396.

2. De Quervain, D., Roozendaal, B., & McGaugh, J. (1998). Stress glucocorticoids impair retrieval of long-term spatial memory. *Nature*, *394*(6695), 787.

3. Gerra, G, Zaimovic, A, Mascetti, G.G, Gardini, S, & Zambelli, U. (2001). Neuroendocrine responses to experimentally-induced psychological stress in healthy humans. *Psychoneuroendocrinology*, *26*, 91-107.

4. Luine, V., Beck, K., Bowman, R., Frankfurt, M., & MacLusky, N. (2007). Chronic Stress and Neural Function: Accounting for Sex and Age. *Journal of Neuroendocrinology*, *19*(10), 743-751

5. Mangels, J.A. (1997). Strategic processing and memory for temporal order in patients with frontal lobe lesions. *Neuropsychology*, *11*(2), 207-221.

6. McEwen, B. (1998). Protective and Damaging Effects of Stress Mediators. *New England Journal of Medicine*, *338*(3), 171-179.

7. Oei, N.Y., Elzinga, B.M., Wolf, O.T., de Ruiter, M.B., Damoiseaux, J.S., Kuijer, J.P., Veltman, D.J., Scheltens, P., Rombouts, S.A., 2007. Glucocorticoids decrease hippocampal and prefrontal activation during declarative memory retrieval in young men. Brain Imaging Behavior. 1, 31–41.

8. Packard, M. (2009). Anxiety, cognition, and habit: A multiple memory systems perspective. *Brain Research*.

9. Sapolsky, R. (1999). Glucocorticoids, stress, and their adverse neurological effects: relevance to aging. *Experimental Gerontology*, *34*, 721-732.

10. Stamatakis, A., Pondiki, S., Kitraki, E., Diamantopoulou, A., Panagiotaropoulos, T., Raftogianni, A., et al. (2008). Effect of neonatal handling on adult rat spatial learning and memory following acute stress. *Stress: The International Journal on the Biology of Stress*, *11*(2), 148-159

11. Smith, A., Whitney, H., Thomas, M., Perry, K., & Brockman, P. (1997). Effects of Caffeine and Noise on Mood, Performance and Cardiovascular Functioning. *Human Psychopharmacology: Clinical & Experimental*, *12*(1), 27-33.

12. Starman, M.N, Gebarski, S.S, Berent, S, & Schteingart, D.E. (1992). Hippocampal formation volume, memory dysfunction, and cortisol levels in patients with Cushing's syndrome. *Biological Psychiatry*, *32*, 756-765.

13. Tollenaar, M., Elzinga, B., Spinhoven, P., & Everaerd, W. (2008). Long-Term Outcomes of Memory Retrieval under Stress. *Behavioral Neuroscience*, *122*(3), 697-703.

14. Weiss, E., Krupka, N., Bähner, F., Both, M., & Draguhn, A. (2008). Fast Effects of Glucocorticoids on Memory-Related Network Oscillations in the Mouse Hippocampus. *Journal of Neuroendocrinology*, *20*(5), 549-557.

15. Wolf, O. (2009). Stress and memory in humans: Twelve years of progress?. *Brain Research*, *1293*142-154.

16. Woolley, C.S, Gould, E, & McEwen, Bruce. (1990). Exposure to excess glucocorticoids alters dendritic morphology of adult hippocampal pyramidal neurons. *Brain Research*, *531*, 225-231.

17. Zoladz, P., & Diamond, D. (2009). Linear and Non-Linear Dose-Response Functions Reveal A Hormetic Relationship Between Stress and Learning. *Dose-Response*, *7*(2), 132-148.