

# The Effect of Sleep Deprivation on the Responses to Stress

Jiraporn Yuenyongrattanakorn

Senior Project Submitted in Partial Fulfillment of the Requirements for the Degree of

Bachelor of Science in Psychological Science

Faculty of Psychology

Chulalongkorn University

Academic Year 2013

# # 5337551338: Bachelor of Science

KEYWORDS : SLEEP DEPRIVATION / DEPRESSION ANXIETY STRESS SCALE /  
ELECTROMYOGRAPHY

JIRAPORN YUENYONGRATTANAKORN : THE EFFECT OF SLEEP  
DEPRIVATION ON THE RESPONSES TO STRESS

ADVISOR SUPALAK LUADLAI, 51 pp.

#### Abstract

Sleep was an important factor for human's functioning, especially for the stress management. People tended to cope with stressful situation better when they had sufficient sleep. This experiment was conducted to examine the impact of sleep deprivation on both subjective and physiological response to stress. The study adopted the electroencephalography (EMG) and Depression Anxiety Stress Scale (DASS21) to investigate the level of stress in the individuals. In this experiment, it was hypothesized that the participants with sleep deprivation would response more intensely in terms of muscle tension and self-report of stress, anxiety and depression. The result was found to conform to the hypotheses. The participants scored higher on DASS21 scales when they had inadequate amount of sleep when compared to control participants. Data recorded by EMG also supported, as sleep deprived participants had higher muscle tension than normal individuals with sufficient sleep.

Field of Study: Psychological science      Student's Signature.....

Academic Year: .....2013.....

Advisor's Signature.....

## Acknowledgments

I would like to express my very great appreciation to Dr. Supaluk Luadlai for her valuable and constructive suggestions during the planning and development of this research work. Her willingness to give her time so generously has been very much appreciated. Moreover, I would like to thank Dr. Jason Ludington and Dr. Apitchaya Chaiwutikornwanich for their generous help for my project and interest in my study.

## Table of Contents

Abstract.....	iii
Acknowledgements.....	iv
List of Tables.....	v
List of Figures.....	vi
Chapter 1: Introduction.....	1
Theoretical Framework.....	2
Literature Review.....	2
Objectives.....	13
Hypotheses.....	14
Pilot Study.....	14
Pilot Study: Methodology.....	15
Pilot Study: Results.....	17
Pilot Study: Discussion.....	19
Chapter 2: Methodology.....	19
Participants.....	19
Designs.....	20
Measures.....	20
Procedures.....	22
Chapter 3: Results.....	23
Additional Analysis.....	27
Chapter 4: Discussion.....	34

	vi
Strength and Limitation.....	38
Implication and Future Researches.....	40
Conclusion.....	41
References.....	42
Appendix A: Demographic Information.....	48
Appendix B: Sample of DASS-21.....	49
Appendix C: Sample of the Video as a stressor.....	50
Bibliography.....	51

List of Tables

Table1. Descriptive Statistic for EMG, Stress, Anxiety and Depression.....31

List of Figures

Figure 1. Mean level of muscle tension measured by EMG before and after exposing to stressors.....18

Figure 2. Mean level of stress measured by DASS-21 before and after exposing to stressors.....18

Figure 3. Mean level of muscle tension measured by EMG as a function of sleep pattern between sleep deprived and control groups .....25

Figure 4. Mean level of stress between control and sleep deprivation groups measured by Depression Anxiety Scale (DASS-21).....25

Figure 5. Mean level of depression between control and sleep deprivation groups measured by Depression Anxiety Scale (DASS-21) .....26

Figure 6. Mean level of anxiety between control and sleep deprivation groups measured by Depression Anxiety Stress Scale (DASS-21) .....26

Figure 7. Mean level of muscle tension as a function of sleep pattern and before/after exposure to the stressor.....31

Figure 8. Mean level of overall DASS-21 scores as a function of sleep pattern and before/after exposure to stressor .....32

Figure 9. Mean stress level on DASS-21 as a function of sleep pattern and before/after exposure to the stressor.....32

Figure 10. Mean anxiety level on DASS-21 as a function of sleep pattern and before/after exposure to the stressor.....33

Figure 11. Mean depression level on DASS-21 as a function of sleep pattern and  
before/after exposure to the stressor .....33





## Chapter 1

### **Introduction**

Sleep consumes a big part in human's lifetime. People spend for a quarter to one-third of their lifespan on sleeping (Schardt, 2012). Thus, sleep is important to human body as food and water. However, the study on this topic is not very popular in psychological research, and the logical ground of sleep was not well-documented as expected. Regardless of the lack of research on rationale for sleep, the problem of sleep pattern nowadays has become critical. In 1879, before the invention of light bulb by Thomas Edison, the average amount of sleep reported by National Sleep Foundation was 10 hours per night (Foley, Ancoli-Israel, Britz & Walsh, 2004). The current data suggested that the number of hours people sleep today has reduced dramatically as compared to the past. At the present time, people slept approximately 6.5 to 8.5 hours on each night (Schardt, 2012). People in general population inevitably have to respond to this more demanding lifestyle. And, this dramatic change in sleep pattern is more likely to be increasingly pervasive and ongoing. The problem seems to be mostly associated with social and environmental factors where people have to deal with all variety of everyday challenges including work pressure and psychological stress in around-the-clock society (Winzeler, 2014). Apparently, people tend to be exposed to more pressure and responsibilities than they ever had in the past. Therefore, less amount of sleep hours are available for individuals in each day. Staying up late for leisure and working night shift have generally become common.

## **Theoretical Framework**

Many recent research findings showed that sleep deprivation has become one of the widely concerned issues in the society nowadays. According to the study on the sleep duration using daily self-report from 1975 to 2006, the finding found that there was significantly more people in the average population had begun to sleep less than six hours per night (Schardt, 2012). Apparently, the term “sleep deprivation” or “sleep restriction” refers to an inadequate quantity and quality of sleep. Specifically, sleep deprivation or sleep restriction in human was found to relate to how the individuals function during the day (Winzeler et al., 2014). It could generally result in fatigue and sleepiness in the daytime causing difficulties in performance on daily functioning. Moreover, lacking of sleep tends to lead to many detrimental effects on performance, health, functioning and quality of life. According to the experiment on human and rat, the research presented that sleep deprivation might be able to alter both cognitive and emotional perception of the stressors (Sgoifo et al., 1999). Further researches discovered that inadequate sleep could result in disturbance in ability to perform cognitive tasks; including alteration in memory, executive functions as well as affective and emotional processing (Roca et al., 2012). In sum, the individual’s cognitive function could basically become influenced by the sufficiency of sleep one had during the night.

## **Literature Review**

It was claimed by Meerlo, Sgoifo and Suchecki (2008) that sleep deprivation not only had a consequence on cognitive functioning, but it also had an impact on

psychological stress. Evidences show a relationship between sleep and stress.

Psychological stress and sleep loss are associated with risks for many clinical diseases, such as heart diseases (Franzen, Gianaros, Marsland, Hall, Siegle, Dahl & Buysse, 2011). Particularly, many researchers usually examined both stress and sleep disturbance as a combined effect that tended to co-occur and interact with each other. For example, Vgontzas and his colleagues (1998) claimed that one factor that might contribute to greater perception of stress during sleep derivation was that the individuals were potentially exposed themselves to stress by worrying about their sleep problems during wakefulness. Moreover, sleep loss was discovered to have an influence on how an individual responded to a stressor (Meerlo, Sgoifo & Suchecki, 2008). Specially, people deal with their reactivity to the new stimuli under stress differently when they are lacked of sleep. It was proposed that sleep deprivation had a dramatic impact on both emotional responsiveness to work-related challenge and stress, as it amplified the adverse effect of emotional perception of unforeseen situations (Zohar, Tzischinsky, Epstein & Lavie, 2005). In sum, the available data showed that individual perceived and responded emotionally more intensely to stress once one was deprived of proper sleep.

As aforementioned studies, it could be seen that the previous assessment of stress was commonly based on subjective experiences on the rating scale, such as Relative Stress Scale (Ulstein et al., 2007), Fear Survey Schedule (Miller et al., 1995), Cook-Medley hostility Scale (Weidner et al., 1989), Depression Anxiety Stress Scale (Brown et al., 1997) and Brief Symptom Inventory (Lemyre & Tessier, 2003). These data largely relied on emotional response and individual report. Nevertheless, it is important to

explore how sleep deprivation or sleep loss play a role in the physiological reaction to a subsequent stressor. It is to display how stress influences the way body functions rather than solely relying on the self-report of personal experiences. Thus, stress experiment requires various sensors to obtain objective measures to enhance the understanding the effect of stress. According to Sharma and Gedeon (2012), there were a variety of techniques used to detect the level of stress through physiological responses including electroencephalography (EEG), blood volume pulse (BVP), heart rate variability (HRV), galvanic skin response (GSR), and electromyography (EMG).

Although the initial signal of change in the way individuals handle with sleep deprivation seemed to be on the level of emotional perception, chronic sleep restriction was found to be a crucial factor for the fundamental properties of stress system in a manner that is comparable to what it was shown in stress-related disorders, such as depression (Meerlo, Koehl, Borghot & Turek, 2002). The individuals do not only respond emotionally to stress, but their brain also functions differently when dealing with stress. To our knowledge, available researches examining on both human and rodents demonstrated that the baseline activities of stress system in the brain were influenced by the individuals' sleep duration. Meerlo and his colleague (2012) found the relationship between the sleep restriction and the alternation in neuroendocrine functions. Short duration of sleep or sleep restriction was found to affect the activities of major neuroendocrine stress system. A number of studies stated that sleep deprivation retained the higher level of this stress system during the restlessness beyond the level of relaxed wakefulness (Vgontzas et al., 2001). When the individuals were lacking of sleep, the

activities in both limb of the neuroendocrine systems, which comprised of Hypothalamic Pituitary-Adrenocortical Axes (HPA) and Sympathetic Adrenomedullary (SAM), became relatively elevated (Axelrod & Reisine, 1984).

The first major neuroendocrine system that associated with the response to stressors was Hypothalamic Pituitary-Adrenocortical Axes (HPA). During the sleep deprivation, the hypothalamus induced the activation of corticotrophin-releasing hormone (CRH), which activated the release of adrenocorticotrophic hormone (ACTH) from the pituitary. Subsequently, ACTH started liberating glucocorticoids or cortisol, which served several functions for coping with stress, from the adrenal cortex (Arborelius et al., 1999). These changes in hormone responses during sleep restriction were found to be comparable to what it was shown in many clinical disorders. The study by Meerlo, Sgoifo and Suchecki (2008) claimed that sleep restriction amplified the sensitivity to mood disorders through the regulation in HPA activity and cortisol release. It particularly found that the patients with chronic insomnia often had an increase in cortisol level, which was associated with a decrease in neuronal plasticity and neurogenesis that were pathophysiological mechanisms for mood disorders (Dranovsky & Hen, 2006). Moreover, the further finding documented the reactivity in HPA in normal individuals with chronic lack of sleep that was similar to the regulation shown in clinically depressed patients. Arborelius, Owens, Plotsky and Nemeroff (1999) revealed that patients with depression had attenuated ACTH response in cardiac autonomic activities as it reduced the sensitivity of CRH that simulated the release of ACTH (Lopez, Akil and Watson, 1999). Meanwhile, there was an elevated sensitivity in ACTH in adrenal gland with

exaggerated cortisol response, which was very crucial for regulating the response of stress (Symons, VanHelder & Myles, 1988). These researches showed a potential relationship between sleep deprivation and activities in HPA axes appeared in mental disorders.

The second major neuroendocrine system that associated with response to stressful stimuli was Sympathetic Adrenomedullary (SAM). The activation in sympathetic nervous system induced the secretion of two catecholamines including the noradrenaline from the sympathetic nerve terminals and adrenaline from the adrenal medulla. (Johnson, Kamilaris, Chrousos & Gold, 1992). However, these activities could be influenced by the quality of sleep, as it was argued that the level of catecholamines became higher when the person stayed awake (Axelrod & Reisine, 1984). A number of experiments supported the claim that the individuals were rapidly declined in their activities of adrenaline and noradrenaline during the sleep onset when compared to wakefulness. Therefore, sleep deprivation was likely to be involved in an increase in the sympathetic system and catecholamine levels during the wakefulness. This was in accordance with the experiments on rats and human, which discovered that the subjects with prolonged sleep restriction gradually and progressively increased in their level of catecholamine (Irwin, Thompson, Miller, Gillin & Ziegler, 1999). This could be attributed to greater sympathetic activation supporting the notion that retaining awakening in a state of sleep debt tended to require higher sympathetic activation.

The consequence of sleep restriction could have both direct and indirect impacts

on neuroendocrine system. As claimed by Meerlo, Sgoifo and Suchecki (2008), all types of stressors were known to directly activate the major neuroendocrine stress system. Nonetheless, the magnitude of responses to different types of stressors could be varied. There was actually a specific activation in the brain that was contingent on the nature of stimuli as different stressors were generated by different neural circuits (Herman et al., 2003) For example, previous data reported that stressors involving physiological activities, like intensive exercise, were transmitted to the neuroendocrine control center (Meerlo et al., 2002). On the other hand, cognitive stressors that were associated with interpretation and evaluation relayed through multisynaptic and limbic-forebrain circuit (Lopez, Akil & Watson, 1999). In the case of physical stressor, exercise was known to require glucose and oxygen, which commonly trigger the activation of metabolic stress system from the muscles. This muscle demand for glucose and oxygen eventually signaled the activities in the neuroendocrine center. For the case of emotional challenge, the response to these control centers was modulated in the limbic systems. The relevant evidence suggested that sleep deprivation affected both cognitive and emotional perception of stressors, as sleep loss altered the perception of particular stimuli on a cognitive level and influenced the afferent responses to the neuroendocrine control regions (Sgoifo et al., 2006). These data could be clearly manifested that when people were lacking of sleep, it did not only have a direct impact on the responses of the major neuroendocrine system. At the same time, it also indirectly conditioned the neuroendocrine regions, which were governed by different types of stressors.



Insufficient sleep not only impacted the neural activities in neuroendocrine system, but it also altered the physical responses to the stressors causing the individual to be susceptible to irregular heartbeats (Sgoifo et al., 1997). In the experiment by Sgoifo, Buwalda, Roos, Costoli, Merati and Meerlo (2006), sleep-deprived animals that were subjected to acute restraint stress were found to have an elevated vulnerability to cardiac arrhythmias, namely ventricular and supraventricular premature beats (Sgoifo, Buwalda, Roos Costoli Merati & Meerlo, 2006). The study withdrew vagal antagonism after the sympathetic stimulation reported that there was a decrease in Heart Rate Variability (HRV) in the subject that were under stress, leading to a number of problems in health condition (Franzen, Gianaros, Marsland, Hall, Siegle, Dahl & Buysse, 2011). It might elevate the risk of provoking ventricular tachycardia, ventricular fibrillation and sudden death following myocardial infarction (Magid, Martin & Kehoe, 1985). As it was previously claimed that the lower value of HRV parameter stimulated a high risk of ventricular premature beats when the subjects were exposed to acute stressors, the risk of the premature beats was reduced in subject characterized by higher value of HRV during stress episode (Sgoifo et al., 1999). Heart rate variability could be measured through the specific device called Electrocardiogram (ECG). It was a special technique used to assess and record the electrical activities generated by the impulse of ions through the cardiac muscles. To conclude, several evidences showed that the individuals that were deprived of sleep had a higher tendency to function poorly in their health condition, especially heart rate variability.

To our knowledge, sleep deprivation could also have an effect on heart functioning. Heart was one of the organs that were responsible for the sympathetic nervous system and the sympatho-adrenal system. The studies of Heart Rate Variability (HRV) corresponded to the finding that sympathetic activity was affected by sleep (Sgoifo, 1997) Therefore, both heart rate and blood pressure were expected to increase in response to the rise in sympathetic activity when exposing to stressful stimuli (Lusardi et al, 1996). As a result of an increase in autonomic sympathetic activity during sleep deprivation, heart rate and blood pressure were expected to rise in accordance with the higher level of sympathetic activities (Vgontzas et al., 2001). There was a study was found a significant increase in heart rate and blood pressure when an individual was lacking of sleep, elevating the risk of developing many heart diseases (Tochikubo, Ikeda, Miyajima & Ishii, 1996). The study by Lusardi and his colleagues (1996) revealed that sleep-deprived subject had higher blood pressure after waking up in the morning. In the long run, this higher sympathetic activity and blood pressure might contribute to a permanent elevation in blood pressure, and eventually causing hypertension and cardiovascular diseases (Tochikubo, Miyajima & Ishii, 1996)

In addition, further investigation by Franzen and colleague (2011) demonstrated that sleep deprivation resulted in augment of activities in systolic blood pressure to psychological stress. This effect also became strengthen during the light phase of sleep, which was usually the resting phrase in the experiment with rodents. Consequently, the stress system in the body became more easily influenced by sleep deprivation. This clearly indicated that this problem of sleep heightened susceptibility to cardiovascular

diseases by dysregulating the physiological system in the body. The available data could exhibit some critical understanding of the damaging outcome of sleep deprivation and sleep disturbance on cardiovascular reactivity under stressful situation. To sum up, an inadequate sleep seemed to show negative effects on both heart functioning and blood pressure in the body.

As it was claimed that the body tended to respond to the stressors intensely once an individual did not get enough sleep hours, the moist and temperature in the body were also affected. Galvanic Skin Response (GSR) was one of the most reliable measures of stress where it measured the flow of electrical activities through the surface of the skin. Stress could be captured effectively through skin response. When the person was under stress, the moisture on the surface of the skin would be increased causing the skin conductance to soar (Sharma and Gedeon, 2012). This would raise the flow of electrical activities through the skin of the individual. On the other hand, the skin conductance would be decreased once the individual was less stressed or became relaxed. Skin temperature and moisture were claimed to be higher when people encountered the stressful situation. It could be concluded that the moisture and temperature under the skin also played a role under stressful stage.

Another crucial evidence of physiological parameter of stress that might be affected by sleep deprivation was muscle activities. It was one of the most critical methods to assess the level of stress, which was to investigate the level of muscle tension in the body. There was many studies demonstrated the impact of mental stress on muscles

(Krantz, Forsman & Lundberg, 2004). Electromyography (EMG) is a non-invasive technique that could be applied to measure stress level by detecting the muscular activities in the body. This technique can be used to measure stress by placing the electrodes either on upper trapezius muscle that was located in the shoulder area or frontalis muscles on the forehead (Wijisman, Grundlehner, Penders & Hermens, 2010). The change in muscle activities in EMG would record a rise of amplitude during stressful period and reduction in the amount of gaps during the relaxation. Both suggested that the increased muscle activity was caused by stress tasks. Generally, when the muscle was tensed, it could be indicated that the individual was more likely to become stressed.

Stress was documented to be the problem in which it could lead to stress-related health problem like musculoskeletal disorders (Franzen et al., 2011). Wijisman (2013) revealed an increase in EMG activities in muscular tension that stemmed from work-related issues. This rise in EMG activities was associated with musculoskeletal systems in which sometimes resulted in shivering (Martini, 1998). This muscle tone elevation might therefore be able to forecast the stress level. In addition, there was another way stress could be measured through muscle tension by operating Micro muscle tremors (MMT). The instrument that was generally used was called Voice Stress Analyzer (VSA). By using this technique, stress was measured when an individual was talking or making a speech. MMT detected the muscle tension in the vocal tracts to analyze the level of stress of the individual. These data have proven that the measurement of stress using the muscular tension was widely used with many different tools in the studies. This meant that the muscle tension was one of the reliable and valid measurements of stress.

Recent research found that the habits of insufficient sleep and stress were one of the most important leading causes that might be involved with many physical disorders including office syndrome. The integration of these two factors of problem in sleep pattern and stress was deteriorating. Office syndrome was usually found in office workers in a working age. The syndrome occurred as a consequence of unhealthy habits like sitting in a long period of time without moving sufficiently. However, the main of roots of office syndrome could be stemmed from stress and sleep deprivation due to the workload or work shift (Laohasinurak, 2013). These behaviors might result in inflammation of the muscles and tendons resulting in the pain in the muscles. It could be seen that the effect of insufficient sleep and stress was harmful and detrimental to health condition. Both of these factors were not only affect each other, but they also co-existed causing a number of health damages.

The findings about the relationship between sleep and stress are not compromising. There were contradictory results that still could not find the effect of sleep on the response to stress. A number of studies failed to recognize the impact of sleep deprivation on how the individual responded to stress (Matzner, Hazut, Naim, Bar-Haim & Ben-Eliyahu, S, 2011). The study by Maggio and colleagues (2013) disclosed that the study was not strong enough to conclude that sleep deprivation had an influence the stress, especially the physiological stress, in the individuals. They extracted data from 68 studies regarding the effect of sleep deprivation on the changes in stress hormones in both human and animal. The results showed that the sleep deprivation had no effect on the stress hormones. This data made the relationship between sleep deprivation and stress

remained controversial. Nonetheless, this study aims to investigate this effect of sleep deprivation on stress using self-report Depression Anxiety Stress Scale-21 or DASS-21 (Lovibond & Lovibond, 1995) and muscle tension assessed by Electromyography (EMG). These two tools have proven to be effective indicators of stress (Sharma & Gedeon, 2012).

### **Objectives**

In this paper, it is attempted to emphasize on the importance of sleep on well-being. Being deprived of sleep could potentially be conditions that result in negative effect both emotional and physiological responses to stress. The presented data illustrated that the stress system and other physiological activities increased the sensitivity in response to the stimuli under stressful situation when individuals did not have enough amount of sleep. This present study is aimed to confirm and further investigate the effect of the amount of sleep hours on the individuals' response to stressors. I would like to explore the negative effect of sleep deprivation on how human deal with stimulus under stressful situation. Specifically, this research comprised of two studies, which were pilot study and Experimental study. The pilot study aimed to check the effect of the stressor, which was the video clip about ferry sinking in Korea in 2014, whether it would be able to induce stress in the subjects in the study. The experimental study was done to investigate the effect of sleep deprivation on the response to stress including muscle tension and self-report.

## **Hypotheses**

The current study aims to examine whether stressor induction could affect on muscles tension and participants' reports of stress, anxiety, and depression levels. In addition, this study mainly compared level of muscles tension and stress, and anxiety, and depression levels between sleep-deprived participants and sleep-sufficient participants after they were induced stressors. Hence, two groups of participants were recruited; 1) participants with sleep deprivation, and 2) sleep-sufficient or control participants. There were mainly two hypotheses in this study. Firstly, it was hypothesized that the individual who sleep less than 4 hours was more likely to have higher tension in their muscles, indicating the sensitivity of physiological response towards the stressors. Secondly, the hypothesis stated that the individuals who slept less than 4 hours was hypothesized to report higher in their stress, anxiety and depression levels measured by Depression Anxiety Stress Scale (DASS21) (Lovibond & Lovibond, 1995) than the individuals who slept 6 hours or more. Post-hoc test using the Bonferroni correction would be employed to confirm aforementioned hypotheses.

## **Pilot Study**

This pilot study aimed to check the effect of the stressor, which was the video clip about ferry sinking in Korea in 2014, whether it would be able to induce stress in the subjects in the study. Additionally, this study aimed to test reliability of Depression, Anxiety, Stress Scale (DASS21) (Lovibond & Lovibond, 1995) whether this questionnaire would have appropriate reliability to employ in Thai students.

### **Pilot Study: Methodology**

In this pilot study, the participants included of 20 students from Chulalongkorn University in which 50% of the subjects were male and another 50% of the subjects were female (mean age = 20.45 years,  $SD = 1.24$ ). The participation was voluntary. The participants provided written informed consent before the experiment proceeded. The design was One-way within-participant designs. The independent variable was the level of stress measured by self-report and muscle tension before the exposure to stressor. The dependent variable was the level of stress measured by self-report and muscle tension after the exposure to stressor.

There were a number of measures used in this study including demographic information sheet, self-report of stress and muscle activities. For the demographic sheets, participants filled in demographic information, which generally inquired some personal details, such as name, gender, occupation and age. For the level of stress, the participants' levels of stress were measured using Depression Anxiety Stress Scale (DASS-21) (Lovibond & Lovibond, 1995). It was a shorter version of the original DASS (Lovibond & Lovibond, 1995). This scale was a 21-items self-report questionnaire used to examine the scale of three negative emotional states including depression, anxiety and stress. The respondent was 4-point Likert scale that ranged from one to four. This self-report tool made up of 21 items of statement about the level of experienced stress. For the first subscale, depression scale assessed the level of depression occurred in the individual. The items included the statements about depression-related signs. For example, "I feel I am



not worth much as a person.” Anxiety scale was the second scale. It examined the level of anxiety of the participant. The item included “I am aware of dryness of my mouth.” Or the last subscale, stress scale measured the level of stress in which the item included, “I tend to over-react to situations.” The response of the scale was based on feeling of depression, anxiety and stress. It began with one to four in which 1 meant the absence of experience in a certain situation; on the other hand, 4 demonstrated an extreme frequency of experience in a particular situation. Each participant completed this scale two times. The first one was the pre-test that was given to the participant before the induction of stress. And, the second one was done as post-test which after the participant exposed to the stressor. High scores indicated high severity of depression, anxiety and stress. The surveys took around five to ten minutes. Overall, this inventory provided a good internal consistency and reliability (Cronbach’s  $\alpha = .96$ ). For last measure, the muscle tension was measured by using Electromyography (EMG). It was a non-invasive tool that was designed to examine the level of muscle tension in the individual. The measurement could be done by placing the electrode on to the participant’s shoulders to measure the trapezius muscles.

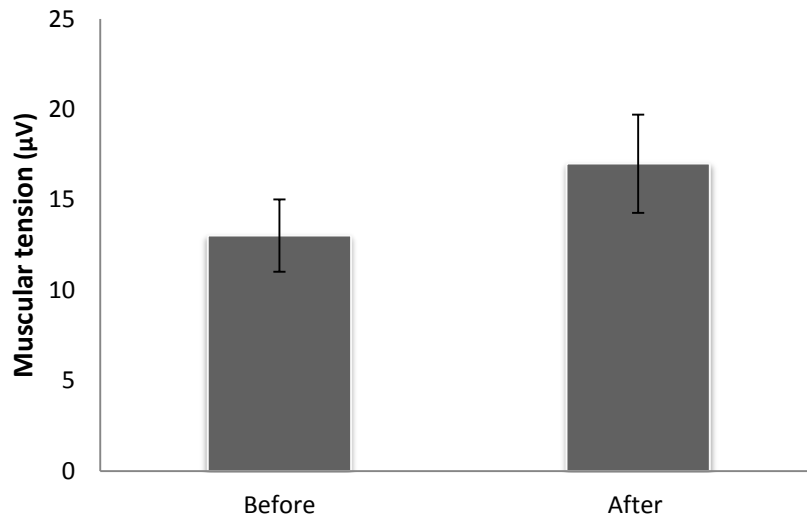
The experiment was begun by having the participant completed the demographic information and provided one’s current level of stress through 21-items self-report scale, Depression Anxiety Stress Scale (DASS) (Lovibond & Lovibond, 1995), as a pre-test. The level of muscle tension of the participants before exposing to the stress was also measured by Electromyography (EMG). The experimenter turned of the light to create a relaxing ambiance for the participant. Then, the experimenter attached electrodes on to

both sides of the shoulders. The participant was not allowed to move their shoulders during the five minutes of the measurement of muscle tension. Subsequently, the stress was induced by having the participant watched five-minutes video clip about the tragedy of ferry sinking in Korea. The muscle tension was also assessed by Electromyography (EMG) while the participant was watching the video clip. After the exposure to stressors, participant filled in report of stress using the post-test DASS-21. At the end, the experimenter debriefed about the information of the experiment and informed the participant that their personal informed collected in this experiment would be kept confidentially.

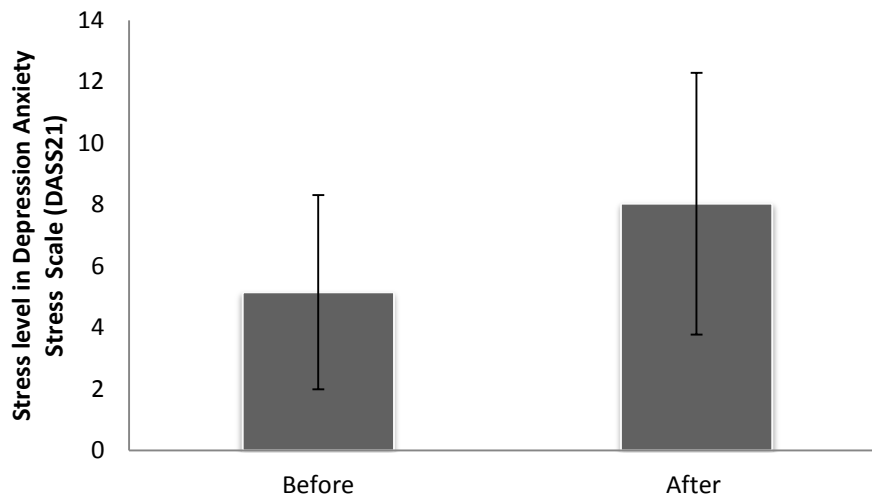
### **Pilot Study: Results**

The study compared the mean of muscle tension in the individuals before and after the exposure to the stressful video clip in despite of the sleep condition. It was done to examine the effectiveness of the stressor whether it would be able to induce stress among the subjects. The results found that there was a difference in the score for before- ( $M = 13.03$  mA,  $SD = 2.00$ ) and after- ( $M = 17.00$  mA,  $SD = 2.71$ ) conditions. As shown in figure 1, the level of muscle tension was higher in the condition that was exposed to stressor than in the condition before being induced by the stressor. Moreover, the result also compared scores of pre-test and post-test of DASS21, which measured the level of stress in the individuals. This was done as the manipulation check to test the effectiveness of the stressor using the scores on DASS21. Figure 2 displayed that the scores of stress scale for post-test ( $M = 8.03$ ,  $SD = 4.26$ ) in DASS21 were significantly higher than scores

on stress scale for the pre-test ( $M= 5.15$ ,  $SD = 3.16$ ) in DASS21



*Figure 1.* Mean level of muscle tension measured by EMG before and after exposing to the stressors



*Figure 2.* Mean level of stress measured by DASS-21 before and after exposing to the stressors

**Pilot Study: Discussion**

This pilot study was the manipulation check, which was done to assess the effectiveness of the stressor. It could be seen that the video clip of Korean ferry sinking in the sea was able to induce stress in the individual. The result found that the participants increased in their level of stress after they were exposed to the video clip. The experiment was successful in terms of inducing the stress among the participants. The video clip clearly increased both the subjective reported stress by DASS21 and the level of muscle tension measured by EMG. The data found in this pilot study could be concluded that the video clip was an effective stressor that was able to induce the level of stress in the individuals.

**Chapter 2****Methodology****Participants**

Two groups of participants were recruited, comprising one group of 21 Chulalongkorn University students who had sufficient sleep (sleep > 6 hours) and a second group of 20 Chulalongkorn University students who had sleep deprivation (sleep < 4 hours). The participants consisted of 50% female and 50 % male; mean age = 21.75 years,  $SD = 1.47$ . 1 participant in control group was excluded due to her symptoms of thyroids that might have an impact on the overall results of the study. The participation was voluntary. All participants provided written informed consent before the experiment proceeded.

## **Designs**

One-way between participant designs was adopted in this experiment. The independent variables of this experiment were the conditions of sleep pattern, whether the individuals had sufficient sleep or deficient sleep. The dependent variable of the study was the level of stress of the individuals.

## **Measures**

**Demographic information.** Participants filled in demographic information, which generally inquired some personal details, such as name, gender, occupation and age. Sleep pattern and amount of sleep were also investigated. The questionnaire also required the subject to inform how many hours did the one slept per night (See Appendix A).

**Stress level.** Participants' levels of stress were measured using Depression Anxiety Stress Scale (DASS-21) (Lovibond & Lovibond, 1995). It was a psychological instrument that was adopted in this experiment. DASS-21 (see Appendix B) was a shorter version of the original DASS. This scale was a 21-items self-report questionnaire designed to assess the magnitude of three negative emotional states including depression, anxiety and stress. These negative emotions were used to categorize the questionnaire into three main subscales of Depression, Anxiety and Stress scales. DASS-21 emphasized on the reports of low mood, motivation, and self-esteem, physiological arousal, perceived panic, fear, tension and irritability. The respondent indicated on 4-point Likert scale

ranging from one to four. This self-administered survey composed of 21 items of direct inquiries about current level of experienced stress. For the first subscale, depression scale was responsible for examine the level of depression occurred in the individual. The items included the statements about depression-related signs. For instance, "I cannot seem to experience any positive feeling at all." The second subscale, anxiety scale, investigated the level of anxiety of the participant. The examples of each item included, "I experience trembling". Lastly, the last subscale, stress scale, assessed the level of stress in the individuals in which the items included, "I find it difficult to relax." The response scale was evaluated based on individual experience relating to the feeling of depression, anxiety and stress, starting from one to four where 1 indicated no experience in a particular situation, while 4 represented a very frequent experience of the stated situation. This questionnaire was used to identify how depressed, anxious and stressed an individual felt before and after the exposure to stressors. Each participant completed this scale twice as the pre-test and post-test in which examined the current level of stress of the participant. The pre-test was given to the participant before the exposure to stressor. This was when the individual was in their normal stage of relaxation. On the other hand, post-test was done after the subject underwent the stressful experience. Higher scores demonstrated an increase in severity of depression, anxiety and stress. Completion took approximately five to ten minutes for each participant. Overall, this inventory had a good internal consistency and reliability (Cronbach's  $\alpha = .96$ )

**Muscle tension.** Electromyography (EMG) was utilized to assess the level of muscle tension in the body. This tool was done by placing the electrodes on to both sides of the participant's shoulders.

## **Procedures**

The experiment was carried out by initially having the participant filled in the demographic information, which determined one's age, gender and sleep hours and pattern. Participant was required to provide his/or current level of stress by completing the 21-items self-report scale, Depression Anxiety Stress Scale (DASS) (Lovibond & Lovibond, 1995). Participant returned the questionnaire to the experimenter once one has completed all the questions. He/she was asked to turn off the light in experimental room. Then, his/her level of muscle tension of the participants during this relaxing period was also assessed by using EMG equipment. The participant was attached with electrodes on both sides of the shoulders in which he or she was not allowed to move their shoulders during the tension evaluation. This manipulation during this relaxing period took approximately 5 minutes. Afterwards, the experimenter continued the study by inducing the stressor using stressful video clip. It was a video clip about the tragedy of ferry sinking in Korea (see Appendix C). The video took approximately 5 minutes. The participant was instructed to watch this video without moving their shoulders. At the same time, the experimenter measured the muscle tension of the participant during the stressful situation provoked by the stressful video clip using Electromyography (EMG) equipment. Physiological responses like muscle tension were measured by placing the

electrodes on the trapezius muscles on both sides of the participant's shoulder. The experimenter removed the electrodes from the participant's shoulders. Subsequent to the exposure to stressors, participant reported their stress level as the post-test of DASS21. The experimenter ended the experiment by debriefing about the objectives of the study and informed that the data in this experiment would be kept as confidential.

### Chapter 3

#### Results

The baseline activity of muscle tension measured by EMG in both sleep deprived and control group was the same for both group. The level of muscle tension during the relaxation did not differ among the individuals who were lacking of sleep and individuals who had sufficient sleep. The independent-sample t-test was conducted. The result showed that there was no significant difference between the muscle tensions measured by EMG of sleep deprived ( $M = 13.58 \mu V$ ,  $SD = 2.05$ ) and control ( $M = 12.50 \mu V$ ,  $SD = 1.84$ ) condition before the exposure to stressor;  $t(38) = 1.76$ ,  $p = .086$ .

An independent-samples t-test was conducted to compare muscle tension measured by EMG after the exposure to stressor in sleep deprived and control conditions. The result showed that the level of muscle tension significantly differed among control ( $M = 15.87 \mu V$ ,  $SD = 2.78$ ) and sleep deprived ( $M = 18.10 \mu V$ ,  $SD = 2.18$ ) groups;  $t(38) = 2.83$ ,  $p < .05$ . As illustrated in figure 3, it suggested that the sleep had an impact on muscle tension. The group of participants that did not have sufficient sleep had higher muscle tension than the group of participants that had sufficient sleep.

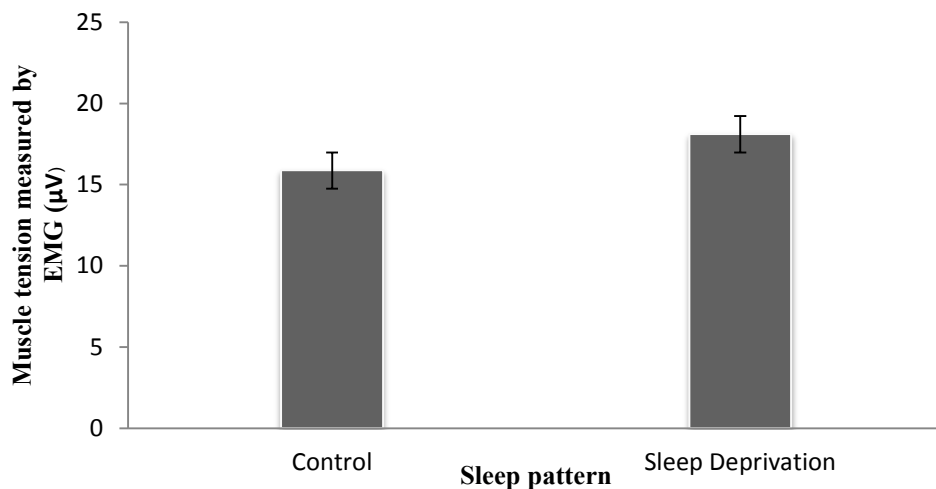


The level of self-report stress measured by DASS21 was also examined. The result of an independent-samples t-test of the level of stress in sleep deprived and control groups found a significant difference in the scores for control ( $M = 4.90$ ,  $SD = 1.86$ ) and sleep deprived ( $M = 11.15$ ,  $SD = 3.63$ ) conditions;  $t(38) = 6.85$ ,  $p < .05$ . Figure 4 clearly demonstrated that the sleep pattern significantly had an effect on the level of stress. The group of participants that did not have sufficient sleep was shown to have higher scores on stress level than the group of participants that had sufficient sleep.

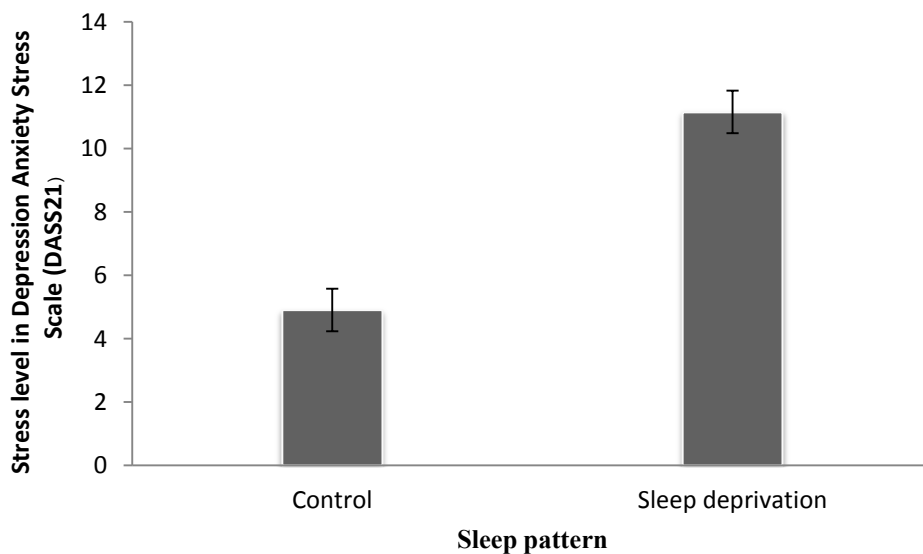
The level of depression determined by DASS21 was evaluated. The result of an independent-samples t-test of the level of depression in sleep deprived and control group showed a significant difference in the scores for control ( $M = 5.05$ ,  $SD = 1.93$ ) and sleep deprived ( $M = 10.95$ ,  $SD = 4.26$ ) conditions;  $t(38) = 5.64$ ,  $p < .05$ . These results demonstrated that the sleep pattern corresponded to the level of depression. As illustrated in figure 5, the group of participants slept less than 4 hours held higher scores on level of depression than the group of participants that had sufficient sleep of more than 6 hours.

The level of anxiety by DASS21 was also measured. An independent-samples t-test was applied to study the level of anxiety in the participants with sleep deprivation and control participants. The result indicated that there was a significant difference in the scores for control ( $M = 5.45$ ,  $SD = 2.19$ ) and sleep deprived ( $M = 11.45$ ,  $SD = 3.97$ ) conditions;  $t(38) = 5.92$ ,  $p < .05$ . Figure 6 displayed that the sleep pattern could influence the level of anxiety. The group of participants that did not have sufficient sleep had

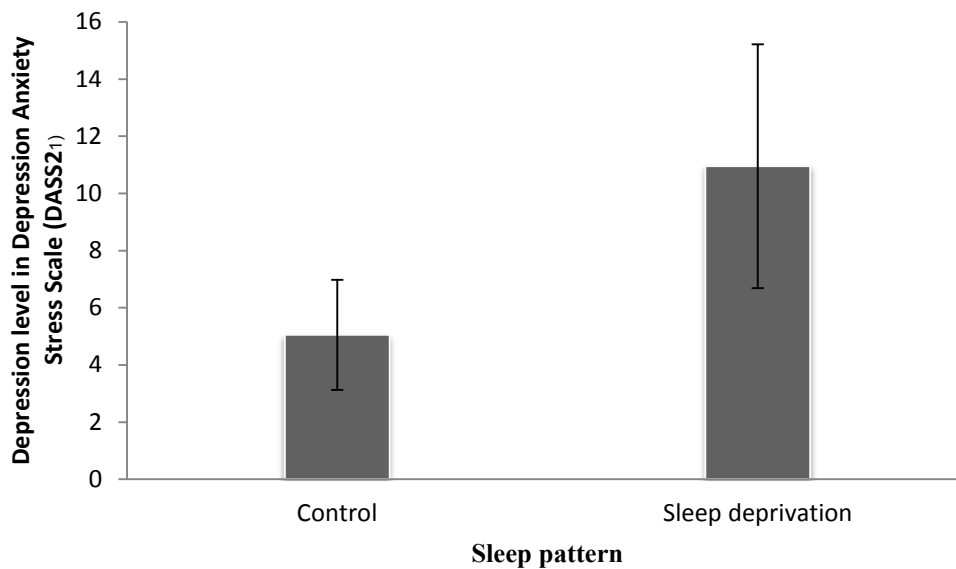
higher scores on anxiety level measured by DASS21 than the group of participants that had healthy sleep pattern.



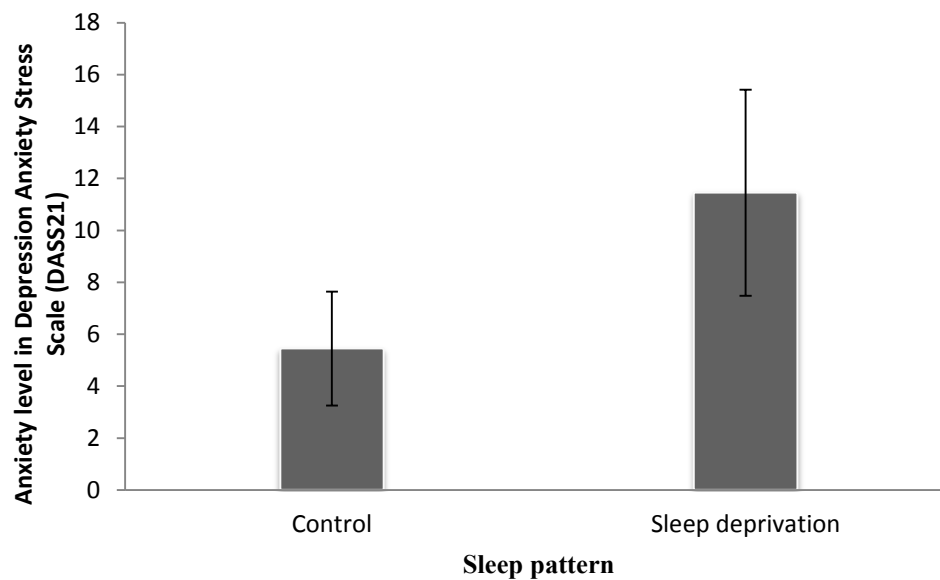
*Figure 3.* Mean level of muscle tension measured by EMG as a function of sleep pattern between sleep deprived and control groups



*Figure 4.* Mean level of stress between control and sleep deprivation groups measured by Depression Anxiety Stress Scale (DASS2)



*Figure 5.* Mean level of depression between control and sleep deprivation groups measured by Depression Anxiety Stress Scale (DASS21)



*Figure 6.* Mean level of anxiety between control and sleep deprivation groups measured by Depression Anxiety Stress Scale (DASS21)

### **Additional Analysis**

The repeated-measures analysis of variance was used to investigate the effect of sleep pattern and before/after exposure to stressor on EMG level, and to examine the differences levels of muscles tension in both sleep-deprived and control participants. As illustrated in figure 7, it found a significant main effect of before/after exposing to stressor,  $F(1,38) = 82.55, p < .05$ . That is, stressor induction influenced on muscles tension in both groups of participants. Post hoc test using the Bonferroni revealed that participants particularly scored lower in their muscle tension in before ( $M = 13.03\mu N, SD = 2.00$ ) condition than after ( $M = 16.98\mu N, SD = .2.71$ ) condition. The effect of sleep pattern was also shown to be significant,  $F(1,38) = 8.85, p < .05$ . Post hoc test using Bonferroni showed that sleep deprived participants ( $M = 15.84\mu N, SD = .51$ ) had higher muscle tension than control participants ( $M = 14.18\mu N, SD = .51$ ). However, it was found no significant interaction between before/after exposing to stress and sleep pattern,  $F(1,38) = 1.74, p = .20$ .

The results of repeated-measures ANOVA assessed the effect of before/after exposure to stressor and sleep pattern on overall DASS-21 scores. It could be seen in Figure 8 that the scores on DASS-21 before exposing to stressor ( $M = 15.95, SD = 8.79$ ) significantly lower than DASS-21 scores after exposure to stressor ( $M = 24.47, SD = 12.44$ ),  $F(1,38) = 121.08, p < .017$ . In addition, there was also a significant main effect of sleep pattern on overall DASS-21 scores,  $F(1,38) = 50.36, p < .017$ . Subjects in sleep deprivation group ( $M = 27.98, SD = 1.55$ ) showed to score higher in overall DASS-21 than control participants ( $M = 12.45, SD = 1.55$ ). The interaction between scores

before/after exposure to stressor and sleep pattern was also significant.  $F(1,38) = 11.48, p < .05$ . To better understand the meaning of this interaction, difference scores of DASS-21 were calculated by subtracting the scores before and after exposure to the stressor. The results found that sleep deprived group ( $M = 11.15, SD = 5.96$ ) had significantly higher difference scores than the control groups ( $M = 5.90, SD = 5.38$ ),  $t(38) = -3.39, p < .05$ .

Post Hoc was done to further examine each subscale of the DASS-21, including stress, depression and anxiety. The analysis in each subscale would be measured against alpha of  $p < .017$  to counteract the tripled chance of type I error.

As shown in figure 9, the results of repeated measure ANOVA of the effect of before/after exposure to stressor and sleep pattern on stress level in DASS-21. It showed that there was a significant main effect of before/after exposing to stress,  $F(1,38) = 108.54, p < .017$ . The level of stress significantly increased after the participants were exposing to the stressor ( $M = 8.03, SD = 4.326$ ); meanwhile, stress level was lower before the exposure to stressor ( $M = 5.15, SD = 3.13$ ). Moreover, there was also a significant main effect of sleep pattern on the stress level,  $F(1,38) = 50.24, p < .017$ . This indicated that the level of stress differed significantly between sleep deprivation and control groups. People that were sleep deprived ( $M = 9.28, SD = .54$ ) found to score higher in the level of stress in DASS-21 than the people with adequate sleep ( $M = 3.90, SD = .54$ ),  $p < .017$ . There was also found a significant interaction between the before/after exposing to stressor and sleep pattern,  $F(1,38) = 10.05, p < .017$ . Data demonstrated that sleep deprived people ( $M = 7.40, SD = 2.54$ ) seemed slightly more stressed than control group ( $M = 2.90, SD = 1.71$ ) before they watched stressful video, but this difference seemed

even larger after a stressor. In after condition, individuals in sleep deprivation group ( $M = 11.15$ ,  $SD = 3.63$ ) seemed to have higher stress level than non-sleep deprived individuals ( $M = 4.90$ ,  $SD = 1.86$ ). To investigate this, difference scores of stress were calculated by subtracting stress scores measured before and after the stressor. It was found that the difference scores of sleep deprived participants ( $M = 3.85$ ,  $SD = 2.11$ ) were significantly greater than the control participants ( $M = 2.00$ ,  $SD = 1.38$ ),  $t(38) = -3.29$ ,  $p < .05$ .

Another repeated measure ANOVA was adopted to investigate the effect of before/after exposure to stressor and sleep pattern on anxiety level. It could be seen in figure 10 that there was a significant main effect of before/after exposure to the stressor,  $F(1,38) = 60.99$ ,  $p < .017$ . The participants had higher level of anxiety in after condition ( $M = 8.45$ ,  $SD = 4.39$ ) than before condition ( $M = 5.55$ ,  $SD = 3.05$ ). Their anxiety level in DASS-21 rose after they were induced with the stressor. There was also discovered a main effect of sleep pattern,  $F(1,38) = 38.85$ ,  $p < .017$ . This meant that control group found to differed in their level of anxiety from the sleep deprivation group. People who did not have enough sleep ( $M = 9.50$ ,  $SD = .57$ ) showed to have higher anxiety than people with enough sleep ( $M = 4.50$ ,  $SD = .57$ ). The results also displayed a significant interaction between the sleep pattern and before/after exposure to the stressor,  $F(1,38) = 7.25$ ,  $p < .017$ . The results found that people who did not have enough sleep ( $M = 7.55$ ,  $SD = 2.58$ ) appeared to be more anxious than normal people ( $M = 3.55$ ,  $SD = 2.01$ ) before a stressor. However, this effect seemed greater after a stressor. Sleep deprived participants ( $M = 11.45$ ,  $SD = 3.97$ ) greatly showed higher anxiety than control participants ( $M = 5.45$ ,  $SD = 2.19$ ) after a stressor. In order to test this, difference scores

of anxiety were computed by subtracting anxiety scores measured before and after the stressor. The results indicated that difference scores of sleep deprived people ( $M = 3.90$ ,  $SD = 2.70$ ) were significantly higher than normal people ( $M = 1.90$ ,  $SD = 1.94$ ),  $t(38) = -2.69$ ,  $p < .05$ .

In figure 11, it displayed the results of repeated measure analysis of variance of depression level as a function of sleep pattern and before/after exposure to the stressor. It found that people differed significantly before and after they were exposed to the stressor,  $F(1,38) = 87.75$ ,  $p < .017$ . The participant increased in their level of depression in after condition ( $M = 8.00$ ,  $SD = 4.43$ ); meanwhile, they lowered in their depression level before condition ( $M = 5.25$ ,  $SD = 3.21$ ). In addition, there was a significant main effect of sleep pattern,  $F(1,38) = 36.10$ ,  $p < .017$ . Participants in sleep deprivation group ( $M = 9.20$ ,  $SD = .61$ ) showed to have higher level of depression than control participants ( $M = 4.05$ ,  $SD = .61$ ). There was also an significant interaction between sleep pattern and before/after exposing to the stressor,  $F(1,38) = 6.53$ ,  $p < .017$ . Data revealed that individuals in sleep deprivation ( $M = 7.50$ ,  $SD = 2.93$ ) seemed to be more depressed than individuals with adequate sleep ( $M = 3.10$ ,  $SD = 1.53$ ) before they were exposed to the stressor. This difference appeared to be bigger after they were exposed to the stressor. Sleep deprived people ( $M = 10.10$ ,  $SD = 4.26$ ) showed higher depression than non-sleep deprived people ( $M = 3.10$ ,  $SD = 1.53$ ), especially in after exposure to the stressor. To examine this, difference scores of depression were calculated by subtracting depression scores measured before and after stressor. Results showed that difference scores of sleep

deprivation group ( $M = 3.50$ ,  $SD = 2.26$ ) were significantly greater than normal people ( $M = 2.00$ ,  $SD = 1.34$ ),  $t(38) = -2.55$ ,  $p < .05$ .

Table 1. *Descriptive Statistic for EMG, Stress, Anxiety and Depression*

Independent variable	Level	<u>EMG</u>		<u>Stress</u>		<u>Anxiety</u>		<u>Depression</u>	
		<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Control	Before	12.49	1.84	2.90	1.71	3.55	2.01	3.10	1.53
Control	After	15.87	2.78	4.90	1.86	5.45	2.19	5.10	1.93
Sleep deprivation	Before	13.58	2.05	7.40	2.54	7.55	2.58	7.50	2.93
Sleep deprivation	After	18.10	2.17	11.20	3.63	11.45	3.97	10.10	4.26

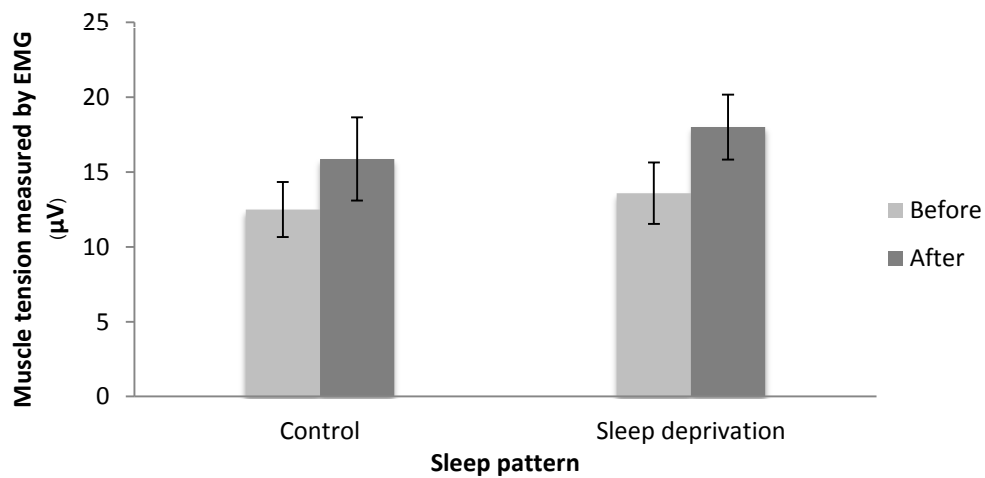
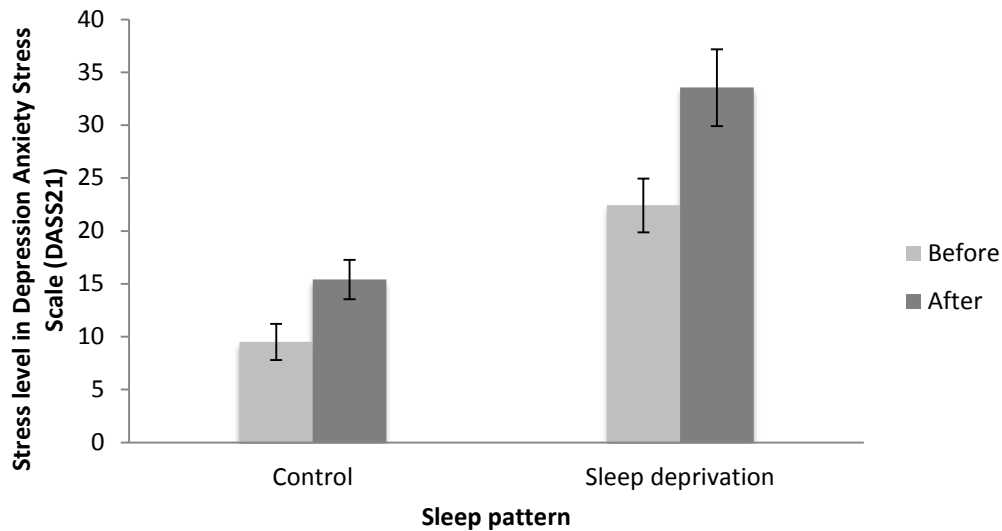
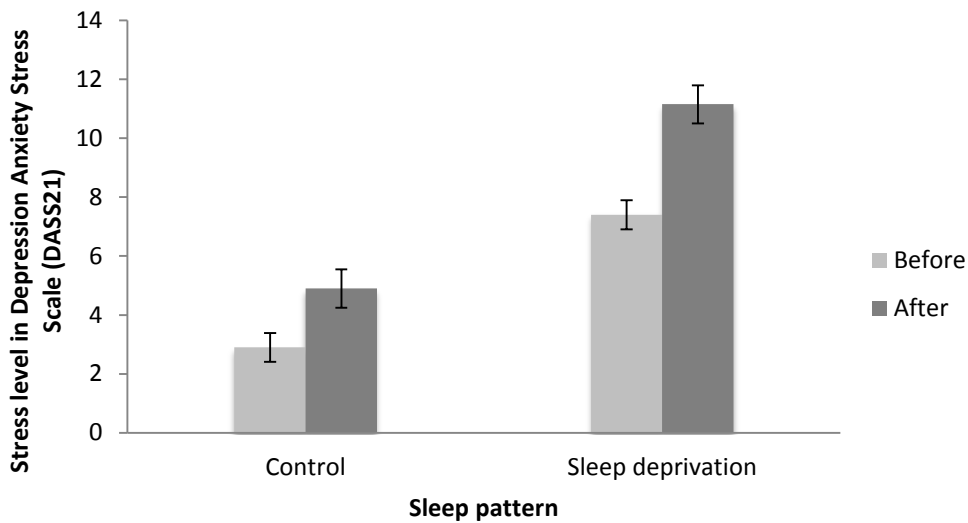


Figure 7. Mean level of muscle tension as a function of sleep pattern and before/after exposure to stressor

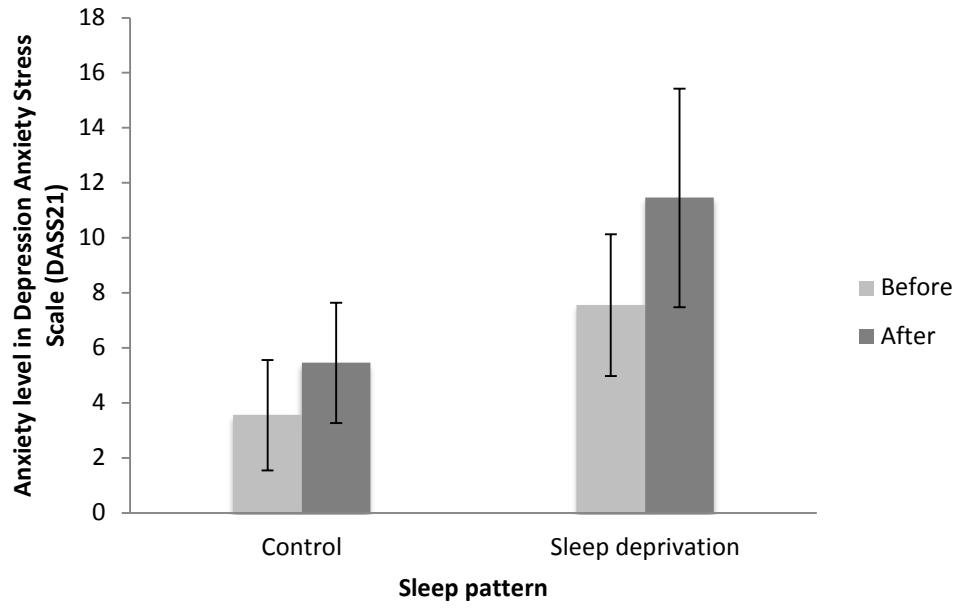




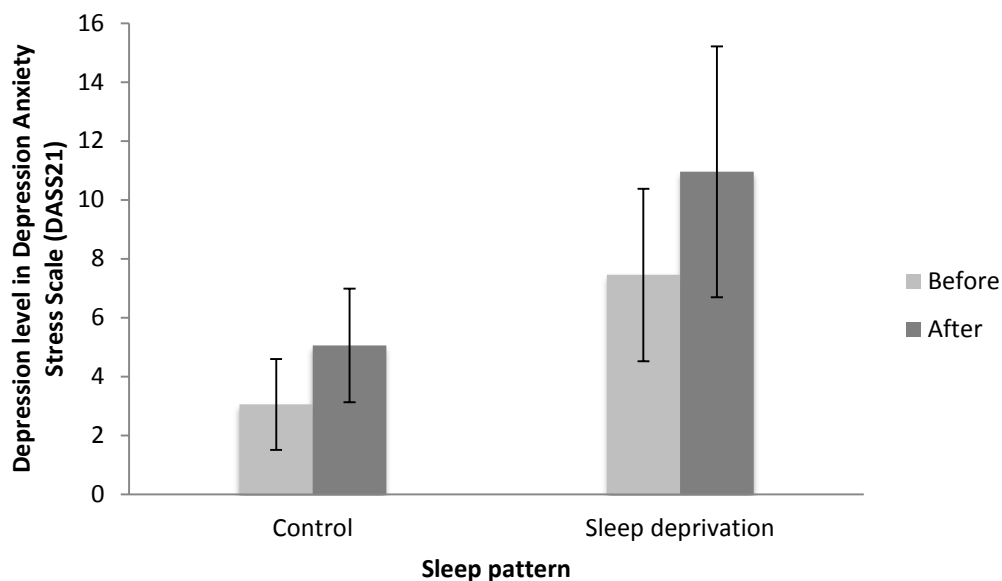
*Figure 8.* Mean level of overall DASS-21 scores as a function of sleep pattern and before/after exposure to stressor



*Figure 9.* Mean stress level on DASS-21 as a function of sleep pattern and before/after exposure to stressor



*Figure*  
10. Mean anxiety level on DASS-21 as a function of sleep pattern and before/after exposure to stressor



*Figure 11.* Mean depression level on DASS-21 as a function of sleep pattern and before/after exposure to stressor

## Chapter 4

### Discussion

This experimental study was conducted to examine the impact of sleep deprivation on the response to stress. Particularly, this study discovered new findings to the literature in which it used the muscle tension measured by EMG and self-report DASS-21 as the indicators of stress in order to investigate the effect of sleep deprivation on the response to stress. Specifically, this study comprised of two groups of participants, which were the sleep deprived and control groups. The results were conformed to the first hypothesis. That is, participants in sleep deprivation condition scored higher in the Depression Anxiety Stress Scale (DASS21), especially in stress scale, than the control group. Furthermore, the second hypothesis was also supported. It was claimed that the

individuals who were sleep deprived were more likely to have higher muscle tension than the individuals who had adequate sleep.

There was the effect of sleep deprivation on self-report stress. People who were lost of sleep tended to react towards the stressor more emotionally than the healthy individuals that had sufficient sleep. In this experiment, participants in sleep deprived condition scored highly in all of the subscales, including depression, anxiety and stress scales, in DASS21. As shown in Brown and colleagues (1997), the interpretation of each subscale varied. The scores could be explained different within different subscales. To our knowledge, Lovibond and Lovibond (1955) indicated that the individuals that scored higher than 7 in stress scale would be considered as having higher level of stress than normal population. The result clearly manifested that people in sleep deprivation condition scored averagely 11 in which was more than 7 in this subscale. The level of stress could be interpreted by the stress scale as moderately higher than normal individuals. However, for the depression and anxiety scales in DASS21, the sleep-deprived subjects also had average scores of 11. This might be explained that the subjects could be more depressed and anxious more than normal individual. The severity of the scores was also found to be very high, indicating that the subject with inadequate sleep hours tended to be very depressed and anxious than typical individuals that had sufficient hours of sleep.

Furthermore, data examined by the experiment clarified that the sleep-deprived subjects perceived themselves as more depressed, anxious and stressed than the normal

subjects with healthy sleep pattern. Specially, the sleep deprived individuals appeared to rate themselves moderately high in the stress scale. In addition, they were considered to rate themselves very high for depression and anxiety scales. This suggested that the individuals who slept less than 4 hours showed high level of self-report stress, depression and anxiety than the individuals who slept more than 6 hours. The reaction to stress, depression and anxiety corresponded to the quality of sleep. These factors exerted influence on one another. The result of the study appeared to be in accordance with Zohar, Tzischinsky, Epstein and Lavie, (2005). It revealed that people would be able to manage and cope with their stress more effectively when they had healthy hours of sleep. On the other hand, individuals with sleep deprivation would be less productive in terms of coping with their subjective feeling of stress. Furthermore, these data appeared found in the experiment might be able to explain by the research from Vgontzas and his colleagues (2001). It was claimed that people who did not have an adequate sleep were more likely to spend time worrying about their sleep-deprived condition. This tended to elevate the scores on subjective report of stress, as the individuals became worried about their sleepless conditions. And, they would probably report their worries into the self-report. In conclusion, the level of subjective report by DASS-21 increased when the individual had insufficient sleep when compared to normal individual.

Physiological data based on this experiment were also supported the previous findings by many researchers about the impact of sleep on the bodily response to stress. According to Sgoifo and his colleagues (1997), it was declared that the physiological reaction of the individual towards stressors tended to be more strongly during the sleep

deprivation. These subjects would be tense in their muscles when they encountered with stressful situation. The participants that were insufficient of sleep were presented with approximately 18 mA as compared to control subjects that showed around 15 mA after they had exposed to the stressful video clip. This showed that the stress was more likely to increase muscular tension when the subjects experience stressful situation. The alteration in muscle activities in EMG recorded an increase of amplitude during stressful period and reduction during the relaxation. Basically, when people were under stressful stage, their muscular tension raised. The further research articulated that the psychological stress played an important role in causing musculoskeletal disorder demonstrating that the individual would have higher muscle tension when they become stress (Lundberg, 1994).

The participants showed higher DASS-21 scores after they watched stressful video clip. This means that they responded more intensely to stress when they were exposed to the stressor. More importantly, this effect was even greater when the subjects were sleep deprived. This increase in DASS-21 scores specially rose when people did not have enough sleep. This indicated that people become susceptible to stress, anxiety or depression when they were exposed to stressful situation, particularly when they had inadequate sleep.

Follow up analyses revealed that people significantly increased in their level of stress, anxiety and depression after they were exposed to stressor, especially in sleep deprivation group. This increase did not only happen to the overall DASS-21 scores. But,

this effect also occurred in each subscales. Sleep deprived group was more susceptible to the stress, anxiety and depression in response to stressful stimuli than normal people with adequate sleep. All participants had higher muscle tension after they watched the tragic video of the Korean ferry sinking.

This data manifested that people really increased in their muscle tension when undergoing stressful stimuli. They became more responsive towards stressors, complying with previous researches. As it was shown in EMG report, the trapezius muscles on the shoulders of the subjects became more tensed and strained in the subjects when they watched the stressful video. The body became sensitive to stress, which made it more difficult for the individual's stress management. This might end up leading to the higher risk of developing many physiological and psychological disorders (Wijisman, 2013).

As claimed by Franzen and colleagues (2011), the mental stress could lead to many negative outcomes. It increased the risks of many pathological diseases as well as stress-related health problem like musculoskeletal disorders. In order to decrease this risk of these harmful effects, it was very crucial for the individuals to get appropriate hours of sleep. Sleep was known to be one of the most important factors for individual's daily functioning. It was very vital for health and well-being. It helped the body maintained in healthy stage and functioning (Von Treuer, Norman & Armstrong, 1996). Specially, it was suggested that sleep was very essential for stress regulation. Stress coping would be productive and functioning well with an appropriate amount of sleep. The study by Lemyre and Tessier (2003) showed that people reported less stress when they had a good

night sleep. On the other hand, the individuals tended to become more stressed when they were lacking of sufficient sleep. Individual would perceive one's level of stress as higher when lacking of proper amount of sleep hours.

### **Strength and Limitation**

The strength of this study was the use of scientific tool. This study relied on both objective and subject devices, including electromyography (EMG) and the shorter version of Depression Anxiety Stress Scale (DASS21). The experimental finding provided two different perspectives of support that could be discussed in terms of scientific and emotional approaches. There were both the results of self-report and physiological data from EMG biofeedback. Moreover, this experiment had high internal validity. The study directly investigated the variables, which were sleep and stress in the laboratory. This could reflect the data without many confounds or other uninterested variables that were not examined in the study. It was certain that those confounding factors could be ruled out. However, the experiment was still far from perfection.

The limitation of this study concerned with the ecological validity. It was because the experiment was conducted in the laboratory. Thus, it was a two-sided sword in which it might be hard for the research to generalize to the real-world setting. The participants might be aware that they were being observed; consequently, they responded differently when they knew they were studied by the experimenter. Moreover, the study has limited number of participants. The order effect might play a role in the interpretation of the study in which it was associated with the passage of time including practice effect. This



made the participant became improved in their performance due to repeated practice with a task and got fatigue and bored of the task after multiple trials. In addition, the studied still relied on the self-report, which might be inaccurate. In the experimental study, the participant reported DASS-21 for both before and after the exposure to stressor. The time gap only took less than 15 minutes. The participant might recognize the original responses in the pre-test in which the responses on pre-test tended to have an impact on the response in post-test. Moreover, another limitation of self-report, the way people reported their hours of sleep could be misrecognized or false. The subjects sometimes could be uncertain about their sleep hours. More importantly, the operational definition of sleep deprivation was not clear. Different individuals might report different hours of sleep deprivation. People tended to have different degree of their stage of sufficient sleep. Therefore, 4 hours might be considered as deprivation for some people yet not in some others. The number of hours people slept might not be the indication of insufficient sleep. The quality of sleep could be more important. Last but not least, the study had neglected the average hours of sleep of the participant. It merely depended on the hours of sleep the subject had last night before participating in the experiment.

### **Implication and Future Researches**

One important implication of this study was to show the importance of sleep. As we learned that it was crucial to have sufficient sleep to better cope with stressful situation. However, the number of hours is not the key. It is the quality of sleep an individual had that actually play an important role in stress management. For the future

study, the experiment could be developed in numbers of way. Firstly, it could be improved by examining more variety of physiological responses including the electrical activities in the brain, moisture of the skin, heart rate and blood volume. These factors could be studied by using many different tool including electroencephalography (EEG), blood volume pulse (BVP), heart rate variability (HRV) and galvanic skin response (GSR). They would be very interesting to study to, so that, the experimental study could gain more support from different objective data. Moreover, the factors, like personality and gender differences, were elements that should also be closely investigated. These factors might also link to and have an influence on the relationship between sleep and stress. The future study might be able to find out more about the effect of these factors. Finally, the researches in the future might be able to separate the individuals with high stress and low stress before exposing to the stressors. Their original level of stress could be important to look into. This could be done to see the impact on the response to stress after exposing to stressful stimuli. The future study could explore about the relationship of sleep and stress in a more valid and reliable method.

## **Conclusion**

In conclusion, this study found the impact of sleep deprivation on how individual responded to stress. It discovered the meaningful data and evidence that supported that insufficient sleep really affected the responses to stress, as it made people have larger responses towards stress. This data contributed to the literature showing the negative

effect of being deprived of sleep on stress in which the data might be more or less useful for the research in the future.

#### References

- Arborelius, L., Owens, M. J., Plotsky, P. M., & Nemeroff, C.B. (1999). The role of corticotropin-releasing factor in depression and anxiety disorders. *J Endocrinol*, 1–12.
- Axelrod, J., Reisine, T. D. (1984). Stress hormones: their interaction and regulation. *Science*, 224, 452–9.

Brown, T. A., Chorpita, B. F., Korotitsch, W., & Barlow, D. H. (1997). Psychometric properties of the Depression Anxiety Stress Scales (DASS) in clinical samples.

*Behav Res Ther*, 35(1), 79–89.

Dranovsky, A., & Hen, R. (2006). Hippocampal neurogenesis: Regulation by stress and antidepressants. *Biol Psychiatry*, 59, 113.

Foley, D. Ancoli-Israel, S., Britz, P., & Walsh, J. (2004). Sleep disturbances and chronic disease in older adults: Results of the 2003 National Sleep Foundation Sleep in America Survey. *Journal of Psychosomatic Research*, 56 (5), 497-502.

Franzen, P.L., Gianaros, P.J., Marsland, A.L., Hall, M.H., Siegle, G.J., Dahl, R.E., & Buysse, D.J. (2011). Cardiovascular reactivity to acute psychological stress following sleep deprivation. *Psychosomatic Medicine*, 73(8), 679-82.

Herman, J. P., Figueiredo, H., Mueller, N. K., Ulrich-Lai, Y., Ostrander, M. M., Choi, D. C., & Cullinan, W. E. (2003) Central mechanisms of stress integration: Hierarchical circuitry controlling hypothalamo-pituitary-adrenocortical responsiveness. *Front Neuroendocrinol*, 24, 151–80.

Irwin, M., Thompson, J., Miller, C., Gillin, J. C., & Ziegler, M. (1999). Effects of sleep and sleep deprivation on catecholamine and interleukin-2 levels in humans: Clinical implications. *J Clin Endocrinol Metab*, 84, 1979–85

- Johnson, E. O., Kamilaris, T. C., Chrousos, G. P., & Gold, P. W. (1992). Mechanisms of stress: A dynamic overview of hormonal and behavioral homeostasis. *Neurosci Biobehav Rev*, *16*, 115–30.
- Krantz, G., Forsman, M., & Lundberg, U. 2004. Consistency in physiological stress responses and electromyographic activity during induced stress exposure in women and men. *Integrative Psychological and Behavioral Science*, *39*(2), 105–118.
- Lemyre, L., & Tessier R. (2003) Measuring psychological stress: Concept, model, and measurement instrument in primary care research. *Canadian Family Physician*, *49*, 1159.
- Lopez J. F., Akil, H., & Watson, S. J. (1999). Neural circuits mediating stress. *Biol Psychiat*, *46*, 1461–71.
- Lovibond, S.H. & Lovibond, P.F. (1995). *Manual for the Depression Anxiety Stress Scales*. (2<sup>nd</sup>. Ed.) Sydney: Psychology Foundation.
- Lundberg, U. (1994). Psychophysiological stress and EMG activity of the trapezius muscle. *International journal of behavioral medicine*, *1*(4), 354-370.
- Lusardi, P., Mugellini, A., Preti, P., Zoppi, A., Derosa, G., & Fogari, R. (1996). Effects of a restricted sleep regimen on ambulatory blood pressure monitoring in normotensive subjects. *Am J Hypertension*, *9*, 503–5.

- Magid, N. M., Martin, G.J., & Kehoe, R.F. (1985). Diminished heart rate variability in sudden cardiac death. *Circulation*, *72*, 111– 241.
- Maggio, M. Colizzi, E., Fisichella, A., Valenti, G., Ceresini, G., Dall’Aglia, E., Ruffini, L... & Ceda, G. P. (1998) Stress hormones, sleep deprivation and cognition in older adults. *Fundamentals of Anatomy and Physiology*, *76*(1), 22-44.
- Matzner, P., Hazut, O., Naim, R. Bar-Haim, Y. & Ben-Eliyahu, S. (2011). Immune robustness of healthy young students to thirty hrs sleep deprivation and psychological stress, brain, behavior, and immunity, (25), 206 – 207.
- Meerlo P., Koehl M, Van der Borght K., & Turek F.W. (2002). Sleep restriction alters the hypothalamic-pituitary-adrenal response to stress. *J Neuroendocrinol*, *14*, 1-11.
- Meerlo P., Sgoifo A., & Suchecki D. (2008). Restricted and disrupted sleep: effects on autonomic function, neuroendocrine stress systems and stress responsivity. *Sleep Medicine Reviews* *12*(3), 197-210.
- Miller, J. J. et al., (1995) Three-year follow-up and clinical implications of a mindfulness meditation-based stress reduction intervention in the treatment of anxiety disorders. *General Hospital Psychiatry*, *17*, 192–200.
- Roca, J., Fuentes, L. J., Marotta, A., López-Ramón, M. F., Castro, C., Lupiáñez, J., & Martella, D. (2012). The effects of sleep deprivation on the attentional functions and vigilance. *Acta Psychologica*, *140*, 164-176.

- Schardt, D. (2012). Sleep on it. *Nutrition Action Health Letter*, 39(3), 9-11.
- Sgoifo A., Buwaldab B., Roosb M., Costolia T., Meratic G., & Meerlo P. (2006) Effects of sleep deprivation on cardiac autonomic and pituitary-adrenocortical stress reactivity in rats. *Psychoneuroendocrinology*, 31, 197–208.
- Sgoifo, A., de Boer, S.F., Westenbroek, C., Maes, F.W., Beldhuis, H., Suzuki, T., & Koolhaas, J.M. (1997). Incidence of arrhythmias and heart rate variability in wild-type rats exposed to social stress. *Am. J. Physiol. (Heart Circ. Physiol.)* 273, H1754–H1760.
- Sgoifo, A., Koolhaas, J.M., de Boer, S.F., Musso, E., Stilli, D., Buwalda, B., & Meerlo, P. (1999). Social stress, autonomic neural activation, and cardiac activity in rats. *Neurosci. Biobehav. Rev.* 23, 915–923.
- Symons, J. D., VanHelder, T., & Myles, W. S. (1988) Physical performance and physiological responses following 60h of sleep deprivation. *Med Sci Sports Exerc*, 20, 74–80.
- Sharma, N. & Gedeon, T. (2012). Objective measures, sensors and computational techniques for stress recognition and classification: A survey. *Computer Methods and Programs in Biomedicine*.
- Tochikubo, O., Ikeda, A., Miyajima, E., & Ishii, M. (1996). Effects of insufficient sleep on blood pressure monitored by a new multibiomedical recorder. *Hypertension*, 27, 1318-1324.

- Ulstein, I. et al. (2007). High score on the Relative Stress Scale, a marker of possible psychiatric disorder in family carers of patients with dementia. *International Journal of Geriatric Psychiatry*, 22, 195–202.
- Vgontzas, A. N., Bixler E.O., Lin, H. M., Prolo, P., Mastorakos, G., Vela-Bueno, A., Kales, A., & Chrousos, G. P. (2001). Chronic insomnia is associated with nyctohemeral activation of the hypothalamic–pituitary–adrenal axis: Clinical implications. *Journal of Clinical Endocrinology and Metabolism*, 86, 3787–94.
- Vgontzas, A. N., Tsigos, C., Bixler, E. O., Stratakis, C. A., Zachman, K., Kales, A., Vela-Bueno, A., & Chrousos, G. P. (1998). Chronic insomnia and activity of the stress system: a preliminary study. *J Psychosom Res*, 45, 21–31.
- Von Treuer, K., Norman, T. P., & Armstrong, S.M., (1996). Overnight human plasma melatonin, cortisol, prolactin, TSH, under conditions of normal sleep, sleep deprivation, and sleep recovery. *J. Pineal Res.* 20, 7–14.
- Weidner, G. et al. (1989) Hostility and cardiovascular reactivity to stress in women and men. *Psychosomatic Medicine*, 51, 36.
- Wijsman, J.L.P., Grundlehner, B., Penders, J. & Hermens, H.J. (2010) Trapezius Muscle EMG as Predictor of Mental Stress. *Wireless Health*, 155-163.
- Wijsman, J.L.P., Grundlehner, B., Penders, J. & Hermens, H.J. (2013) Trapezius muscle EMG as predictor of mental stress. *ACM Transactions on Embedded Computing Systems*, 12 (4). 99:1-99:20.



Winzeler K, Voellmin A, Schäfer V, Meyer AH, Cajochen C, Wilhelm FH, & Bader K.

(2014). Daily stress, presleep arousal, and sleep in healthy young women: a daily life computerized sleep diary and actigraphy study. *Sleep Medicine*.

Zohar D., Tzischinsky O., Epstein R., & Lavie, P. (2005). The effects of sleep loss on medical resident' emotional reactions to work events: A cognitive-energy model.

*Sleep*,28, 47–54.

## Appendix A: Demographic Information

**DEMOGRAPHIC INFORMATION**

NAME: \_\_\_\_\_

AGE: \_\_\_\_\_

SEX: \_\_\_\_\_

OCCUPATION: \_\_\_\_\_

HOW MANY HOURS DID YOU SLEEP LAST NIGHT?

\_\_\_\_\_

HOW MANY HOURS DO YOU USUALLY SLEEP?

\_\_\_\_\_

DO YOU USUALLY WAKE UP DURING THE NIGHT?

\_\_\_\_\_

THANK YOU FOR YOUR PARTICIPATION ☺

## Appendix B: Sample of DASS-21

**DASS<sub>21</sub>**

Name:

Date:

Please read each statement and circle a number 0, 1, 2 or 3 which indicates how much the statement applied to you *over the past week*. There are no right or wrong answers. Do not spend too much time on any statement.

*The rating scale is as follows:*

- 0 Did not apply to me at all
- 1 Applied to me to some degree, or some of the time
- 2 Applied to me to a considerable degree, or a good part of time
- 3 Applied to me very much, or most of the time

1	I found it hard to wind down	0	1	2	3
2	I was aware of dryness of my mouth	0	1	2	3
3	I couldn't seem to experience any positive feeling at all	0	1	2	3
4	I experienced breathing difficulty (eg, excessively rapid breathing, breathlessness in the absence of physical exertion)	0	1	2	3
5	I found it difficult to work up the initiative to do things	0	1	2	3
6	I tended to over-react to situations	0	1	2	3
7	I experienced trembling (eg, in the hands)	0	1	2	3
8	I felt that I was using a lot of nervous energy	0	1	2	3
9	I was worried about situations in which I might panic and make a fool of myself	0	1	2	3
10	I felt that I had nothing to look forward to	0	1	2	3
11	I found myself getting agitated	0	1	2	3
12	I found it difficult to relax	0	1	2	3
13	I felt down-hearted and blue	0	1	2	3
14	I was intolerant of anything that kept me from getting on with what I was doing	0	1	2	3
15	I felt I was close to panic	0	1	2	3
16	I was unable to become enthusiastic about anything	0	1	2	3
17	I felt I wasn't worth much as a person	0	1	2	3
18	I felt that I was rather touchy	0	1	2	3
19	I was aware of the action of my heart in the absence of physical exertion (eg, sense of heart rate increase, heart missing a beat)	0	1	2	3
20	I felt scared without any good reason	0	1	2	3
21	I felt that life was meaningless	0	1	2	3

## Appendix C: Sample of the Video as a Stressor



(Link: <http://www.youtube.com/watch?v=rN1-vjqPsmc>)

## Bibliography

### Jiraporn Yuenyongrattanakorn

Jiraporn Yuenyongrattanakorn is an undergraduate student in the Joint International Psychology Program at Chulalongkorn University, Thailand. As a psychology student, she is very opened to variety field of psychological researches. In 2013, Jiraporn also received a Bachelor of Arts in Psychology from University of Queensland, Australia, as a part of the program. She gained many experiences from both in Thailand and Australia. She had opportunity to conduct an experimental research on the topic of the effect of sleep deprivation on the responses to stress in which this research was presented at the 13<sup>th</sup> National Psychology Conference in Thailand.