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THE ROLE OF SLEEP IN THE ENHANCEMENT AND IMPEDANCE OF EPISODIC EMOTIONAL MEMORIES

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THE ROLE OF SLEEP IN THE ENHANCEMENT AND IMPEDANCE OF EPISODIC

EMOTIONAL MEMORIES

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Thesis Abstract

The current study examined whether sleep enhances the retention of emotionally arousing memories, and whether an emotional memory can be blocked with a single night of sleep deprivation. Using a modified version of a slide show varying in emotionality with respect to the narrative (participants' acting as either a mother or father experiencing the tragic death of their imagined child), 100 (healthy young adult student volunteer) subjects following the experience were randomly assigned to either a normal night of sleep or were variously sleep deprived (in laboratory). After having experienced the emotional event followed immediately by a night of sleep deprivation, and a subsequent full recovery night of sleep, we found that sleep deprivation blocked the augmented retention of the emotional memory (p = 0.01) seen in a group having had a normal night of sleep. Further, we found that the late half-night sleep deprivation blocked the retention of the augmented memory, to an even greater extent (p < .005). The results indicate a central role for late-night Stage 2 and REM sleep in the consolidation of emotional memories. The results further indicate that in the immediate aftermath of an emotionally arousing event, sleep deprivation may serve as a prophylactic intervention preventing long-term consolidation of an emotional (i.e., traumatic) memory.

CHAPTER I: INTRODUCTION

Introduction

Of the many things we encounter it is the emotional aspects that stand out and are the

most personally meaningful in our lives. The impact of emotion, "may be so exciting," as noted

American Psychologist William James (1890/1981), "as almost to leave a scar upon the cerebral

tissue" (p. 670). With respect to emotional memories there are two categories: 1) unconscious,

implicit memories as in general classical (Clark & Squire, 1998) and fear conditioning (LeDoux,

2000) that relies on areas of the limbic system including the amygdala, and 2) conscious,

declarative memories as in recall (Cahill et al., 1996) and recognition (Wagner, Kashyap,

Diekelmann, & Born, 2007) tasks, which relies mostly on the integrity of the medial temporal

lobe (MTL) (Eichenbaum, 2000). The main distinction between non-declarative (implicit) and

declarative (explicit) memories lies in its accessibility to conscious awareness; the former being

unconscious ("knowing how"), and the latter being readily available to conscious awareness

("knowing what") (Ryle, 1949/2000). In relation to the latter, emotional memories[1] are thought

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[1] Eichenbaum (2000) defines emotional memory as "The representation of a positive or negative affect associated with specific stimuli. Typically not subject to conscious recollection but reflected in attraction, avoidance or autonomic nervous system activation" (p. 42).

of as declarative memories, more specifically, episodic memories (memory for previously experienced events in the context in which it originally occurred; Tulving, 1983), that have been emotionally tagged. In other words, a type of memory in which upon encoding elicits an arousal response—i.e., changes in heart rate, blood pressure, temperature, pupil dilation, and skin conductance, among other bodily responses (Bradley & Lang, 2000). Considering its implicated instinctual or visceral-like provoking response (Lang, Greenwald, Bradley, & Hamm, 1993), it is no surprise to find that memory for emotionally arousing information is better remembered as compared to neutral (or non-emotional) information (Christianson, 1992a); the memory enhancing and autonomic arousal effects of emotion is seen across a number of experimentally tested stimuli including, but not limited to, pictures (Bradley, Greenwald, Petry, & Lang, 1992), film clips (Cahill et al., 1996), faces (Gupta & Srinivasan, 2009), words (Kensinger & Corkin, 2003), sounds and tones (Bradley & Lang, 2000), and narrated slide shows (Heuer & Reisberg, 1990; Cahill, Prins, Weber, & McGaugh, 1994). Interestingly, the emotional (versus neutral) memory enhancing effect has been found to benefit most over periods of undisturbed sleep (specifically, the latter portion) as compared to equal periods of wakefulness (see Walker, 2009). In fact, impeding sleep either through enforced sleep deprivation or pharmacological manipulation (or by any other means) following learning (e.g., an emotionally arousing story) can lead to memory impairment.

Episodic emotional memories are enhanced by two important dimensions of emotion: 1) valence (ranging from unpleasant [negative] to pleasant [positive], with neutral often considered an intermediate value; Lang, Bradley, & Cuthbert, 1992) and, 2) arousal (ranging from low [calm] to high [excitement]; Greenwald, Cook, & Lang, 1989). In fact, both valence and arousal

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are known to correlate remarkably well with each other and with memory enhancement (Kensinger, 2004). Using functional magnetic resonance imaging (fMRI), Kensinger and Corkin (2004) identified two separate routes for the distinct processing of valence and arousal: the former dependent on a prefrontal cortex-hippocampal network, while the latter, is dependent on an amygdalar-hippocampal network. Arousal is perhaps the most crucial of the aforementioned dimensions, as states LeDoux (1996), "Without the emotional arousal elicited through the implicit system, the conscious memory would be emotionally flat," (p. 201), and thus poorly encoded, which in turn, leads to equally poor retrieval. The arousal response associated with emotional memories contributes to the vivid and detailed quality in which the actual memory is subsequently recalled; this vividness at retrieval is seen in a type of autobiographical memory (memory specific to personally meaningful information including time(s), place(s), and other contextual details) known as flashbulb memories (Brown & Kulik, 1977). From an evolutionary perspective, remembrance of emotionally arousing stimuli (e.g., a predator) and/or situations (e.g., escaping or avoiding a predator) actively maintained through a mechanism such as reactivation occurring in sleep (during offline periods) may have helped the organism survive by keeping such memories consciously accessible for future use (Fishbein, Lau, De Jesús, & Alger, 2010).

1.1   *Sleep's physiology and its role in offline memory processing*

Sleep is generally divided into two categories: non-rapid eye movement (NREM) (Stages 1-4) and rapid eye movement (REM) sleep (Rechtschaffen & Kales, 1968). In humans, early sleep (first half) consists predominantly of slow wave sleep (SWS; specifically Stages 3 & 4),

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while late sleep (second half) consists predominantly of Stage 2 and REM sleep—the latter of which increases with time (usually reaching a maximum in the morning hours of a normal sleeper); both NREM sleep and REM sleep alternate throughout the course of a night at every 90-120 minutes—thus, a typical 8-hr night will contain about 4-5 complete cycles (Peigneux, Laureys, Delbeuck, & Maquet, 2001). There are a number of characteristic features unique to each of the stages of sleep—electroencephalographic (EEG) patterns in NREM sleep is for the most part synchronous, with such waveforms as sleep spindles, K complexes, and high-voltage slow waves, whereas REM sleep, is defined by low-amplitude, mixed-frequency EEG activation, muscle atonia, and episodic bursts of rapid eye movements (Carskadon & Dement, 1994). Evidence in support for differential processing of memories by distinct sleep stages has accumulated in significant number over the years from a wide variety of sources (Walker & Stickgold, 2006) and has, as a result, obtained considerable experimental and scientific validity in spite of divergent theoretical views (Vertes & Eastman, 2000) and spurious findings (Ellenbogen, Payne, & Stickgold, 2006, pp. 716-717). That post-learning sleep enhances memory and impairs it subsequent to sleep deprivation, a finding credited to the seminal work of Jenkins and Dalenbach (1924), is part of a more elaborate process known as consolidation (McGaugh, 2000). Sleep-dependent memory consolidation, to be more precise, is the stabilization and strengthening of newly acquired memory traces leading to acquired resistance to interference from competing information (Stickgold, 2005). Expanding on the animal work (Fishbein, 1970; Pearlman, 1979; Bloch, Hennevin, & Leconte, 1979; Smith, 1981) a similar link between learning and memory whereby hippocampus-dependent explicit (declarative) versus hippocampus-independent implicit (non-declarative) memories have been shown to benefit from non-REM sleep (specifically Stages 3 & 4; Plihal & Born, 1997, 1999; Tucker et

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al., 2006; Lau, Tucker, & Fishbein, 2010) and REM sleep stages (see Maquet, 2001; Walker & Stickgold, 2006), respectively, in human subjects.

Adding to the literature on memory consolidation there is now evidence showing that declarative memory for emotional events is dependent on REM sleep-related processes (Wagner, Gais, & Born, 2001; Hu, Stylos-Allan, & Walker, 2006; Nishida, Pearsall, Buckner, & Walker, 2009). REM sleep is a dynamic stage of sleep with EEG activation and deactivation (Carkskadon & Dement, 1994). This chaotic neural activity is known to originate at the level of the brainstem wherein sensorimotor signals are generated and later interpreted by the forebrain; these disparate and disconnected signals are bound together to create the often-described "bizarre" nature of the dream phenomena (Hobson & McCarley, 1977). Neurophysiological evidence for REM sleep correlates with data showing a disproportionately higher (≈80%) amount of dream reports from this sleep stage as compared to nearly 50% of dream-like mentation in NREM sleep (Nielsen, 2000). Worth noting is that REM dreams, as Stickgold and colleagues (2001) states, "appear to be not only unpredictable and bizarre but highly emotional as well" (p. 1056). The emotional tinge ascribed to dreams "may reflect the mental representations of high limbic activations in conjunctions with deactivation of high-order cortical regions" (Germain, Buysee, & Nofzinger, 2008, p. 187). Neuroimaging data has confirmed increased activation and regional cerebral blood flow of limbic forebrain structures including the pontine tegmentum, thalamus, amygdaloid complexes, and neighboring regions during REM sleep (Maquet et al., 1996). This finding would suggest, as Germain and colleagues (2008) point out, "that REM sleep is an endogenous state of heightened activity in emotional arousal brain centers" (p. 187).

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The amygdala, an area highly implicated in the modulation of emotional arousal (McGaugh, 2004), is believed to influence the formation of episodic emotional memory via its interaction with the hippocampus and prefrontal cortices and surrounding cortical regions (Cahill & McGaugh, 1998). These processes contribute to the memory enhancing effects reported in studies where emotional arousal is elicited (Bradley et al., 1992; Dolcos & Cabeza, 2002), but more importantly, support the necessary brain-state conditions whereby emotional saliency is retained following sleep, specifically REM sleep, as compared to wakefulness (Hu et al., 2006) and NREM sleep (early sleep vs. late sleep comparisons; Wagner et al., 2001; Wagner, Fischer, & Born, 2002). The preferential selectivity of emotional versus neutral information in sleep apparently is also selective to central/gist over local/peripheral information (Payne, Stickgold, Swangberg, & Kensinger, 2008), a consistent finding in the emotional memory literature (independent of sleep) known as the "weapon focus" effect (Loftus, 1979; Heuer & Reisberg, 1990), which is originally credited to Easterbrook (Easterbook, 1959). Sleep deprivation, on the other hand, counteracts these processes (Maquet, 2001); REM sleep deprivation (REMD), in particular, appears to be sensitive to the impairing effects of emotional versus neutral memory (Wagner et al., 2001). Greenberg, Pearlman, Schwartz, and Grossman (1983) report a selective impairment of emotionally meaningful past memory as compared to other types of memories subsequent to REMD, suggestive of "a role for REM sleep in the storage, integration, and retrieval of emotionally meaningful memories" (p. 378). More recently, and along the same lines, Lara-Carrasco, Nielsen, Solomonova, Levrier, and Popova (2009) reports that REMD blunts the intensity of negative emotions and leads to a decreased intensity of dream emotions. In spite of adverse effects associated with sleep deprivation (selective or otherwise) including impairments in mood, attention, concentration, reaction time, vigilance and social and emotional

regulation (Drummer & Dinges, 2005), favorable effects on mood and behavior, on the other hand, have been reported in clinical populations as in depressed (Vogel, Vogel, McAbee, & Thurmond, 1980) and anxiety patients (Benca, 2008)—a finding that elucidates the intimate relationship that exists between "sleep, emotional brain function, and clinical mood disorders" (Walker & van der Helm, 2009, p. 371).

Neurochemical and neurohormonal changes across the sleep stages also interact with memory to promote consolidation (Hasselmo, 1999; Born & Wagner, 2004). NREM sleep is characterized by low levels of acetycholine (ACh) that supports declarative memory consolidation (Gais & Born, 2004), while REM sleep on the other hand, is characterized by markedly low aminergic and comparatively high cholinergic cell activities (Datta, 2007) supporting non-declarative (procedural and motor-skills memories) as well as emotional memory consolidation (Wagner & Born, 2008). Stress hormones (epinephrine and cortisol) released by the adrenal glands also plays a significant role in the interactive effects of memory consolidation, in particular, for emotional memory, which is known to be mediated by the amygdala in the presence of arousal (McGaugh, 2004). Neuroimaging and evoked potential studies have demonstrated a direct linear relationship between amygdala activation during encoding and subsequent memory (Cahill et al., 1996; Hamann, Ely, Grafton, & Kilts, 1999; Canli, Zhao, Brewer, Gabrieli, & Cahill, 2000). In the context of enhanced memory by arousal or "stress," the idea is in line with the assumption originally proposed by Yerkes and Dodson (1908), of an inverted U-shaped function between arousal and performance; a substantial amount of arousal facilitates performance whereas low and excessive amounts impedes it. Payne and colleagues (2006, 2007) found that memories (emotional and neutral) were differentially affected by

pretraining stress—emotional memories are retained whereas neutral memories are impaired. Payne et al. (2007) reports, "hormone release might encourage encoding and consolidation of only those memory elements that were themselves emotionally arousing and salient" (p. 864). These findings are in line with other studies showing that physiological arousal activates beta-adrenergic receptors in the amygdala that contributes to the so-called "memory enhancing effect" (Phelps, 2006). Consistent with animal models (McGaugh, 2003) blockage of these receptors in human subjects through beta-adrenergic antagonists (e.g., propranolol) eliminates the arousal response that is known to contribute to enhanced episodic emotional memory (Cahill et al., 1994).

1.2   Experiment: Nocturnal sleep enhances retention of episodic emotional memory: Total sleep deprivation, and to a greater extent REM and Stage 2 NREM sleep deprivation blocks the enhancement.

Time and again, animal (Fishbein & Gutwein, 1977; Smith, 1985; Wilson & McNaughton, 1994) and human (Karni, Tanne, Rubenstein, Askenasy, & Sagi, 1994; Plihal & Born, 1997; Stickgold, 2005) research studies have shown that post-learning sleep contributes to memory consolidation processes. Recently, there has been a marked interest in sleep in the processing of emotional memories (see Walker, 2009). Theories linking sleep and emotion, however, go as far back as Freud (1900/1996), and were subsequently investigated scientifically with a varied focus ranging from dream mentation (Antrobus, 1990) to effects on mood and behavior (Durmer & Dinges, 2005). Emergent studies focusing on the role of emotion in sleep-

dependent memory processing have centered on identifying the underlying mechanisms by which consolidation (during offline periods) of this type of memory occurs (Hu et al., 2006; Payne et al., 2008; Nishida et al., 2009). In addition, attempting to use sleep as a way of recalibrating social and emotional regulation (Gujar, McDonald, Nishida, & Walker, 2011) as well as exploring its potential use in preventing the consolidation of traumatic negative thoughts or imagery (Wagner, Hallschmid, Rasch, & Born, 2006) in clinical populations (e.g., anxiety, post-traumatic stress disorder [PTSD], and other related affective disorders populations).

Current studies examining differential processing of emotional versus neutral information have confirmed an emotional memory benefit following retention intervals filled with sleep relative to equal durations of wakefulness across daytime (90-min. nap: Nishida et al., 2009; 12-hrs: Hu et al., 2006; Payne et al., 2008; Payne & Kensinger, 2011) and nighttime (3-hr. late-night sleep deprivation: Wagner et al., 2001, 2002; total sleep deprivation: Atienza & Cantero, 2008; Sterpenich et al., 2009) intervals.

1.3   Purpose of current study

In light of these findings we set out to examine, on the one hand, the beneficial impact of sleep, in particular REM sleep, on the processing of emotionally charged information, while on the other hand, examining the impairing effects of wakefulness (across daytime hours) or sleep deprivation (total sleep deprivation and late-night sleep deprivation), on recognition memory. Following the landmark study of Cahill et al. (1994) in which propranolol impaired emotional information (i.e., recognition memory) we utilized a sleep (full-night vs. half-night)/wake design

to examine the impact of an emotional and neutral narrated slide show (adopted from Cahill et al., 1994) on memory retention. In keeping with previous findings, we hypothesized that emotional arousal would: 1) elicit an evoked physiological response, i.e., heart rate deceleration (van Stegeren, Everaerd, Cahill, McGaugh, & Gooren, 1998); 2) influence subjective responses (Cahill et al., 1994) across dimensions of emotionality, valence, and arousal; 3) enhance recognition memory for negative emotional versus neutral information across periods of nocturnal sleep as compared to equal periods of overnight wakefulness; and 4) selectively impair the development of negative emotional information across a full-night (Sterpenich et al., 2009) wake interval and a half-night -- rich in REM sleep – wake interval.

CHAPTER II

Materials and Methods

2.1   Participants

A total of 110 students (52 males, 58 females) of diverse ethnic backgrounds from The City College of New York signed written informed consent to participate in the experiment approved by the college's Institutional Review Board. Participants completing the experiment received course credit for their participation. Of the original sample, five participants failed to complete the experiment; three were excluded for noncompliance with the research criteria regarding maintained wakefulness after daytime training (1 participant) and exclusion of drugs (2 participants). Two additional participants were excluded for near chance performance on the recognition test (1 participant) and technical problems related with the EEG recording (1 participant). Thus, data analyses were performed on 100 participants (46 males, 54 females) with an average age of 20.04 ± 2.21 (mean $\pm$ SD) (range 18-27). All participants were screened prior to the experiment and reported: 1) no prior or current sleep disorders; 2) no current use of medicinal and/or illicit drugs (with the exception of birth control in 2 female participants); and 3) being in good health. Participants were acclimated to the laboratory setting ($\approx$1 week before the actual experiment) including sound-attenuated sleep chambers by spending an adaptation day in the college's Laboratory of Cognitive Neuroscience and Sleep (sleep lab), which included the placement of electrodes. They were required to follow a constant sleep schedule (according to their own rhythm $\pm$ 1 h) for $\approx$7 days before the actual experiment.

2.2   *Stimuli*

The present experiment adopted a narrated slide show from the research study of Cahill and colleagues (1994). The slide show procedure originated from earlier studies (Christianson & Loftus, 1987; Heuer & Reisberg, 1990) to dissociate between memory for emotional and neutral

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information. The slide show (Cahill et al., 1994) is a stimulus set of two (emotional and neutral) closely matched audiovisual presentations that consists of 11 color slides partitioned into three phases (first phase, Slides 1-4; second phase, Slides 5-8; and, third phase, Slides 9-11) accompanied by a taped narration. The first and last phases of both slide show sets are identical in content (pictures and narration); both differ only in narration in the second phase (Slides 5-8). The narration (see Appendix A), however, was slightly modified from the original (Cahill et al., 1994) to elicit an arousal response that was not found in an earlier pilot study with a significantly smaller sample (unpublished data), specifically, a cardiac deceleration (van Stegeren et al., 1998) and enhanced recognition memory (Cahill et al., 1994; 1996) in response to the emotional events introduced in the second phase of the emotional slide show. In the original version (Cahill et al., 1994), the boy is critically injured by a runaway car and rushed to the hospital where surgeons manage to "reattach the boy's severed feet" (phase 2 of the emotional slide show), whereas in the present study, the "boy dies" as a result of "massive blood loss" induced from his injuries (our emotional version of phase 2). The neutral version, on the other hand, remained consistent with Cahill and colleagues' (1994) narration. Each slide (1-11) was displayed for approximately 5 seconds and were of equal brightness and contrast.

2.3   Physiological recordings

Our design was expected to induce the same kind of arousal response that was previously found to produce heart rate deceleration (van Stegeren et al., 1998) using the stimuli from Cahill and colleagues (1994). The electrocardiogram (ECG) was measured via two electrodes attached below the right and left clavicle bone. Electrode sites were cleaned with alcohol to improve

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contact. Mean heart rate (HR) was scored offline by counting the interval between two R-waves (R-R wave peaks) in the ECG; these interbeat intervals were converted to HR in beats-per-minute (bpm).

2.4 *Polysomnographic recordings*

Participants in the sleep group were monitored with digital EEG acquisition software (Gamma System-Grass/Telefactor[tm]) using an eight-channel montage (two EEG [C3-A2, C4-A1], two electro-oculography [EOG], two electrocardiography [ECG], and two chin electromyography [EMG]) during their adaptation and overnight sleep sessions. Sleep stages were scored off-line using standard criteria (Rechtschaffen & Kales, 1968).

2.5 *Design and Procedure*

Figure 1 illustrates the experimental design of the study. The experiment utilized a 2 (*condition: neutral vs. emotional*) × 5 (*group: 8-hr sleep, 8-hr total sleep deprivation (TSD), 4-hr late-night sleep deprivation, 8-hr daytime, and 48-hr daytime groups*) factorial design. Thus, participants were randomly assigned to one of 10 groups (see Fig. 1).

The experiment consisted of three sessions: an adaptation session, a training session, and an incidental testing session (except for the daytime groups that did not adapt) carried out 2 days following a recovery night (at home). An interval of ≈1 week separated the adaptation and

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training sessions. During the adaptation session, participants signed a written consent form, completed a demographic questionnaire, were fitted with electrodes, and took an acclimatization nap in the sleep chambers (lasting 90 minutes). Before leaving the lab, participants were instructed to keep a sleep log (for the five nights preceding the evening of the training session) to record sleep and wake times prior to training. They were also instructed to avoid alcohol and/or caffeinated beverages during the day of the training session as well as given the option of bringing a light snack (or meal) with them upon their return to the sleep lab. The day of the training session, participants arrived at the sleep lab at 9 PM. Upon arrival, participants were acclimated to the lab environment and taken to the sleep chambers to set their belongings and change clothing (if desired) for the overnight stay. After electrode placement (EEG, EOG, EMG, and ECG), participants completed the following: 1) a self-assessment version of the Horne-Ostberg Questionnaire to determine morning and evening types (Terman, Rifkin, Jacobs, & White, 2001), 2) the digit span task (forward learning) to assess general level of concentration (Tucker et al., 2006; Tucker & Fishbein, 2009), and 3) the Stanford Sleepiness Scale (SSS) to assess level of sleepiness at that moment (Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973), before beginning the training session in which participants would view either the neutral or emotional narrated slide show.

Following the procedure by Cahill et al. (1994) participants were told the study focused on "physiological reactions to visual stimuli," and that their ECG would serve as a measure of this. Therefore, participants were unaware that their memory would be assessed later on. The instructions given to the participants were: 1) that the slide show consisted of 11 color slides accompanied by a narration, 2) that each slide was followed by an instructional screen that asked

for their subjective ratings (on a 10-point scale) across dimensions of emotionality, valence, and arousal, 3) that they pay "close attention to each slide," and 4) to "imagine that you (the participants) are the mother/father (according to their sex) of the child in the story" as a way of strengthening encoding through personal association with the event (Sharot, Martorella, Delgado, & Phelps, 2007). The instructions and set-up for the slide show spanned 10 minutes, after which a pre-baseline ECG measure was taken. At about 11:00 pm after baseline recording, the slide show began. The slides were presented via Microsoft Powerpoint on a 20'' computer screen for 15 seconds (each slide), after which an instructional screen containing three questions regarding emotionality, valence, and arousal appeared. Participants were required to rate on a 10-point scale various dimensions of emotion. After the slide show ended, a post-baseline ECG was recorded. Soon thereafter, participants were asked to complete a visual analogue scale (VAS) modified version of the positive affectivity-negative affectivity scale (PANAS) to assess positive and negative affective experience related to the slide show. Following this, participants completed the Student Opinion Scale (SOS; Wolf & Smith, 1995) to assess their "performance and general motivation regarding the study" (Tucker & Fishbein, 2009).

At 11:30 pm participants were allocated to either the 8-hr sleep group ($n = 10$), the 8-hr TSD group ($n = 10$), or the 4-hr late-night sleep deprivation group ($n = 10$). Lights were turned off at 11:45 pm to enable sleep. As these participants winded down for an undisturbed period of nocturnal sleep ($\approx$4-hours vs. $\approx$8-hours) the other participants (8-hr TSD group) were instructed to sit in a chair for a period of 15 minutes to provide a comparable period for rehearsal. After this period, TSD participants remained awake in the laboratory and engaged in a number of activities including viewing pre-selected videos, playing card/board-games, or doing some light

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reading. Those in the 4-hr late-night sleep deprivation group were awakened approximately four hours after sleep onset and taken outside the sleep chambers to have their electrodes removed. Along with those in the TSD group, these participants remained awake in the laboratory until 8 am. In the morning, participants in the 8-hr sleep group were awakened, had electrodes removed, and allowed to leave the laboratory along with the other participants, and were instructed to return the following evening (after a recovery night at home) to "view a different slide show."

Upon their return, 2 days after the training session, participants were taken to a sleep chamber where they were seated in a chair and completed a digit span task (with different number series), a SSS, and informed that there would be no viewing of a different slide show. Instead, participants were told that their memory for the slide show they had viewed 2 days prior would be assessed. Thereafter, participants' recognition for the slide show was assessed using an 88 four-alternative forced choice (4 AFC) item questionnaire (8 questions per slide; adapted and expanded from Cahill et al., 1994). Participants answered the questions in the same sequence as the original presentation of the narrated slide show.

For participants in the daytime (wake control) groups, the procedure was exactly the same as in the overnight groups save that the corresponding 8-hr retention intervals consisted of daytime wakefulness instead of sleep. Following training of the narrated slide show, the 8-hr daytime group remained awake in the laboratory until about 7:00 pm (8 hours after training) when the testing session took place. The 48-hr daytime group, on the other hand, was allowed to leave the laboratory after the training session, but were asked to return 2 days later in the evening

(corresponding with the return of the 8-hr sleep group, the 8-hr TSD group, and the 4-hr late-night sleep deprivation group), at which time their incidental testing session took place.

2.6   Digit span attention task

The digit span task is one of the subtest components measured on the WAIS-III, using only the forward learning part of the test. This task was used in the present study as a measure of attention prior to both training and testing. Numbers in this task are presented serially on a 20" computer screen for 1 s each followed by a 1-s interstimulus interval. After each series, participants write down on a response sheet the numbers in the order they were presented. The first test series consists of three numbers with each series thereafter increasing by one number until reaching the last series, which contains 10 numbers. The digit span always preceded the training and testing sessions. The same task using different numbers, however, was administered at testing. Performance is measured as the average number of correctly recalled number series during training and testing.

2.7   Affective measure

A visual analogue scale (VAS) modified version of the positive affectivity-negative affectivity scale (PANAS) developed by Watson, Clark, and Tellegen (1988) was used to assess positive and negative affective experience related to the slide show. The PANAS consists of two 10-item mood scales: a positive-affective (PA) subscale and a negative-affective (NA) subscale. The PA subscale measures mean rating for the following positive affect descriptors: interested,

alert, excited, inspired, strong, determined, attentive, enthusiastic, active, and proud. The NA subscale, on the other hand, measures mean rating for the following negative affect descriptors: irritable, distressed, ashamed, upset, nervous, guilty, scared, hostile, jittery, and afraid. The participants were told that the scale consists of a number of words that describe different feelings and emotions and were asked to rate each item by placing a mark on the horizontal line to indicate "to what extent you [participants] felt this way about the slide show you viewed." The scale (from "very slightly or not at all" to "extremely") measures 11 centimeters (cm) in length.

CHAPTER III

Results

3.1   *Arousal data*

As described in the Materials and Methods section, each participant had continuous ECG recordings throughout the presentation of the slide show. As such, an average heart rate was

recorded for each of the 11 slides. These were then averaged to yield values for each of the three phases: Slides 1-4 (phase 1 which were similar for both conditions), Slides 5-8 (phase 2 which differed in narration between the conditions), and Slides 9-11 (phase 3 which were similar for both conditions). Table 1 shows mean heart rate in beats per minute (bpm) for the neutral and emotional treatment conditions across the three phases. There were no baseline (pre to post) differences in the neutral condition ($t(49) = -1.621$, $p = .111$), but there was a reliable difference in the emotional condition ($t(49) = -2.075$, $p = .043$). A 2×3 (condition $\times$ phase) mixed ANOVA revealed a highly significant main effect of phase ($F(2,196) = 15.865$, $p < .0001$) and a highly significant interaction of condition by phase ($F(2,196) = 17.361$, $p < .0001$). Follow up t-tests revealed no significant differences between the neutral and emotional conditions in phase 1 ($t(98) = .588$, $p = .558$) and phase 3 ($t(98) = .536$, $p = .593$) of the slide show. As expected (van Stegeren et al., 1998), there was a reliable difference between the neutral and emotional conditions in phase 2 ($t(98) = 2.016$, $p = .047$). Comparisons within groups (conditions) revealed no reliable differences in heart rate across the phases in the neutral condition (p's > .05), however, a reduction in heart rate in phase 2 (69.49 ± 1.53 bpm) of the emotional condition revealed a highly significant difference when compared to phase 1 ($t(49) = 6.063$, $p < .0001$) and phase 3 ($t(49) = -7.276$, $p < .0001$). No reliable difference was found between phase 1 and 3 ($t(49) = -1.719$, $p = .092$). The effect of arousal is exhibited by the significant drop in heart rate in phase 2 (in which the emotional events are introduced) in the emotional condition (see Fig. 2). This pattern is not seen in the neutral condition in which the arousal response is noticeably absent (as is the first and last phase of the emotional slide show).

3.2   Subjective emotional ratings

The results for subjective emotional reactions are presented in Table 2. Independent ratings were assessed for each of the 11 slides using a 10-point likert-scale across dimensions of emotionality (from "not at all emotional" to "very emotional"), valence (from "negative/unpleasant" to "positive/pleasant" and "neutral" as an intermediate value), and arousal (from "very calm" to "very aroused" and "average" as an intermediate value). Individual slide ratings (ranging 1-10) were summed to yield values for each of the three phases, Slides 1-4 (phase 1 yielding a maximum rating of 40), Slides 5-8 (phase 2 yielding a maximum rating of 40), and Slides 9-11 (phase 3 yielding a maximum rating of 30).

Differences in emotionality, valence, and arousal ratings between neutral and emotional treatment conditions were highly significant, as confirmed by t-tests, in phase 2 (p's < .0001) and phase 3 (p's < .0001) (see Table 2). Separate one-way mixed ANOVAs confirmed highly significant main effects of emotionality, valence, and arousal (p's < .0001). Additional ANOVAs revealed highly significant interaction effects between condition and all three dimensions: emotionality (p < .0001), valence (p < .0001), and arousal (p < .0001). These subjective measures of emotional reaction indicate that participants in the emotional condition perceived the slide show, in particular phase 2, as more emotional, more negative, and more arousing as compared to the neutral condition.

Additional subjective measures based on a visual analogue scale (VAS) modified version of the positive affectivity-negative affectivity scale (PANAS) was used to assess positive and negative affective experience related to the slide show. The scale (from "very slightly or not at

all" to "extremely") measures 11 centimeters (cm) in length. Results for these subjective ratings are partitioned into positive-affectivity (PA) and negative-affectivity (NA) subscales. On the PANAS PA subscale rating the differences between neutral and emotional conditions was short of significance ($t(98) = -1.935$, $p = .056$). PANAS NA subscale rating, however, revealed a highly significant difference between neutral and emotional conditions ($t(98) = -6.017$, $p < .0001$).

3.3   Presleep variables

Analysis of the average total sleep time (TST) using an ANOVA revealed no differences between the groups across the 5 nights prior to the training session ($F(4,95) = 1.323$, $p = .267$). Sleep log data (see Table 3) for the recovery night among the overnight (8-hr sleep, 8-hr TSD, 4-hr late-night sleep deprivation) and 48-hr daytime groups revealed no significant differences ($F(3,76) = .901$, $p = .445$).

The Stanford Sleepiness Scale (SSS) developed by Hoddes et al. (1973) was used to assess the level of alertness/sleepiness for all participants prior to training and testing (see Table 4). The SSS consists of a numerical scale ranging from 1-7 (1 being least sleepy, 7 most). An ANOVA revealed no significant differences between the groups prior to training ($F(4,95) = 1.392$, $p = .243$) and testing ($F(4,95) = 2.118$, $p = .085$).

The Student Opinion Scale (SOS) developed by Wolf and Smith (1995) was used to assess general motivation level following training and testing sessions (see Table 4). An

ANOVA revealed no significant differences between the groups following training (F(4,95) = .694, p = .598) and testing (F(4,95) = 1.212, p = .311).

3.4   Digit span

Digit span data (see Table 4) revealed no significant differences between the groups prior to training (One-way ANOVA, F(4,95) = .587, p = .673), and testing (One-way ANOVA, F(4,95) = 1.35, p = .258). However, using repeated-measures ANOVA, with session (training, testing) as the within-subject factor, we found a significant main effect of session (F(1,95) = 7.56, p = .007), suggesting that performance, across all groups, improved from training to testing (as a function of time). There was no significant session × group interaction (F(4,95) = .801, p = .528).

3.5   Sleep data

A summary of sleep parameters for participants in the overnight groups are found in Table 5. The mean TST was approximately 4 hours (average: 243.93 min.) for the late-night sleep deprivation group, and approximately 8 hours (average: 454.20 min.) for the full-night sleep group. Based on these data, we estimated what the total amount of sleep the second half-night would have had if the participants slept the full-night through (454.20 min. less 243.93 min. = 210.27 min.). On this basis we then estimated the sleep stage percent the participants would have had, had they slept the remainder of the night: S1: (3.60 min.) 1.71%; S2: (113.45 min.) 53.95%; SWS: (14.43 min.) 6.86%; REM sleep: (78.80 min.) 37.48% (Table 5).

3.6   *Recognition memory*

Results for the 88-item multiple choice recognition test (percent of correct answers) are shown in Fig. 3 as are raw data values presented in Table 6. Overall recognition memory using a 2×5 (condition $\times$ group) between subjects ANOVA revealed significant main effects of condition ($F(1,90) = 7.188$, $p = .009$) and group ($F(4,90) = 3.581$, $p = .009$), but no reliable interaction between condition and group ($F(4,90) = .603$, $p = .662$). Performance across the phases (1-3) using a $2 \times 5 \times 3$ (condition $\times$ group $\times$ phase) mixed ANOVA revealed a highly significant effect of phase ($F(2,180) = 52.145$, $p < .0001$), and a trend toward an interaction of phase $\times$ condition ($F(2,180) = 1.812$, $p = .077$). There was no three-way interaction of condition by group by phase as confirmed by the ANOVA ($F(8,180) = 1.160$, $p = .326$).

For each of the groups, retention data across the three phases was analyzed with a paired samples t-test. For the emotional condition, a within-groups comparison revealed that scores pertaining to phase 2 were significantly higher than for either phase 1 (8-hr sleep: $t(9) = -8.062$, $p < .0001$; 8-hr TSD: $t(9) = -2.529$, $p = .032$; 8-hr daytime: $t(9) = -3.973$, $p = .003$; 48-hr daytime: $t(9) = -4.714$, $p = .001$) or phase 3 (8-hr sleep: $t(9) = 3.466$, $p = .007$; 4-hr late-night sleep deprivation: $t(9) = 4.198$, $p = .002$; 8-hr daytime: $t(9) = 3.072$, $p = .013$; 48-hr daytime: $t(9) = 2.491$, $p = .034$). Further analysis of phase 2 showed that the scores of the 8-hr sleep group were significantly higher (80.63 ± 1.60) than all the other treatment groups (t-tests): 8-hr daytime group ($p = .04$), 48-hr daytime group ($p = .002$), 8-hr TSD group ($p = .01$), and 4-hr late-night sleep deprivation group ($p = .005$). Although there was no significant difference between the 8-

hr TSD group and the 4-hr late-night sleep deprivation group (p = .599); numerically, however, the 4-hr late-night sleep deprivation group accounts for the greater part of the all night sleep deprivation impairment (67%), suggesting an important role for Stage 2 NREM sleep and REM sleep in the consolidation of emotional memories. Phase 3 findings parallel those of phase 2, being also significant, although less so (t-tests; 8-hr sleep group vs. 8-hr TSD group, p = .03; 8-hr sleep group vs. 4-hr late-night sleep deprivation group, p = .01) (Fig. 3A).

For the neutral condition, there were no significant differences in scores on the recognition test between any of the groups across the phases (p's > .05), with the exception of a reliable difference in phase 2 between the 8-hr daytime group and the 4-hr late-night sleep deprivation group (p = .01) (Fig. 3B). A within groups comparison (t-tests) revealed that similar to the emotional condition, scores for phase 2 were significantly higher for either phase 1 (8-hr sleep: $t(9) = -3.823$, p = .004; 8-hr TSD: $t(9) = -3.888$, p = .004; 8-hr daytime: $t(9) = -4.670$, p = .001) or 3 (8-hr sleep: $t(9) = 4.335$, p = .002; 8-h TSD: $t(9) = 3.413$, p = .008; 8-hr daytime: $t(9) = 3.793$, p = .004; 48-hr daytime: $t(9) = 2.961$, p = .016), suggesting that the middle phase was not purely "neutral." In spite of this significant phase-by-phase within groups difference, there was no reliable sleep benefit, suggesting that neutral memories (unlike emotional memories) are not facilitated (or enhanced) by sleep.

A between-groups comparison (t-tests) revealed no significant differences across the phases between the neutral and emotional treatment conditions in the 8-hr TSD groups (p's > .05). Comparisons between conditions also revealed no significant differences across the phases in the 8-hr daytime groups (p's > .05). No significant differences were found in the 48-hr

daytime groups between the neutral and emotional treatment conditions across the phases (p's > .05). Additional comparisons between the neutral and emotional treatment conditions in the 8-hr sleep groups revealed no significant differences in phase 1 (t(18) = −1.282, p = .216) and phase 2 (t(18) = −1.495, p = .152), but a reliable difference in phase 3 (t(18) = −3.142, p = .006). Further analyses between the neutral and emotional treatment conditions in the 4-hr late half-night sleep deprivation groups showed no significant differences in phase 2 (t(18) = −1.132, p = .273) and phase 3 (t(18) = .236, p = .816), but a reliable difference in phase 1 (t(18) = −2.100, p = .050).

3.7   Relevant correlations

Data from the 4-hr late-night sleep deprivation group did not yield any relevant significant relationships in both the neutral (*n* = 10) and emotional (*n* = 10) conditions. The 8-hr sleep group data, however, for the emotional treatment condition (*n* = 10), yielded the following significant relationships: NA (negative) subscale correlated with overall recognition memory (r = .809, p = .005) as well as phase 1 (r = .841, p = .002) and 2 (r = .658, p = .039) performance; Stage 2 NREM sleep (in min.) correlated with emotionality for phase 2 (r = .636, p = .048); and, Stage 2 NREM sleep (%) correlated with emotionality for phase 2 (r = .724, p = .018). The 8-hr sleep group data for the neutral treatment condition (*n* = 10), on the other hand, yielded no significant relationships.

CHAPTER IV

4.1   General discussion

The present study investigated the role of sleep (and lack thereof) in the processing of emotional as compared to neutral information using a narrated slide show (Cahill et al., 1994). Our main hypothesis was guided by the finding that sleep, in particular REM sleep, facilitates emotional memories (Wagner et al., 2001). Additionally, that emotional arousal leads to a distinctive autonomic response (i.e., heart rate deceleration) (Lacey & Lacey, 1974). Consistent with these findings, our study demonstrates that participants exposed to a traumatizing emotional slide show were exclusively affected by emotional story elements, which produced the notable pattern of bradycardia associated with aversive imagery (van Stegeren et al., 1998). Coincidently, this distinct pattern characterizing arousal described as an "orienting response" (Lacey & Lacey, 1974) is also known to engage processes involved in attention, which aid memory encoding (Kahneman, 1973). Abercombrie, Chambers, Greischar, and Monticelli (2008) report that the amygdala plays a role in the regulation of heart rate, and that noradrenergic activation including the release of stress hormones (i.e., epinephrine and norepinephrine) contributes to "heightened memorability of emotional information" (p. 648). In fact, induced stress prior to encoding has been reported to enhance emotional memories (using Cahill and colleagues' slide show) yet impairs neutral memories (Payne et al., 2007). Aside from an evoked

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physiological response, emotional arousal elicited by the slide show, specifically during phase 2, selectively enhanced emotional memory in participants that slept a full-night (≈8 hrs.). The effect of emotional arousal also appeared to influence phase 3 performance, suggestive of either a possible carry-over effect of emotion or reflective of poststimulus elaboration processes (Christianson, 1992b). Interestingly, and along the same lines, as can be viewed in Figure 3B, the neutral treatment condition (like the emotional) also shows the stereotyped upward tendency (increment in performance) at phase 2, suggesting that the so-called "neutral" slide show may not reflect a pure (non-emotionally arousing) stimuli but serve rather as a "neutral" sham control (a finding consistent with Cahill et al., 1994).

The finding that emotional arousal enhances memory (independent of sleep) is a well documented phenomenon (Christianson, 1992a). However, when we speak of "memory enhancement" or "facilitation" in the context of sleep by emotional arousal, the evidence appears to show a disproportionate benefit on the side of sleep relative to wake. The present study underscores this by showing that even a few hours after viewing (encoding) an emotional slide show over daytime hours, although remembering better than their counterparts (numerically), participants obtaining nocturnal sleep (of equal retention duration) performed above and beyond wake controls. This effect, however, was only seen for the emotional condition. The results as confirmed in the deprivation groups (8-hr TSD and 4-hr late-night sleep deprivation) can easily be argued in favor of interference, whereby performance is presumably inflated in the sleep group. However, to allay this concern (and to control for unspecific effects of sleep deprivation) the design of our study incorporated a recovery night (at home) following the training session for all participants in the overnight (8-hr sleep, 8-hr TSD, and 4-hr late-night sleep deprivation) and

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48-hr daytime groups. Moreover, testing was not commenced until 2 days later (during which time opportunities for interference were equally as likely to occur for participants in these groups). Additionally, sleep deprived (8-hr TSD and 4-hr late-night sleep deprivation) and daytime (wake) participants (8-hr and 48-hr) did not differ from sleep participants (8-hr sleep) in their level of sleepiness prior to training and testing as confirmed by subjective sleepiness scores. As such, the recovery night served to restore sleep deprived participants' cognitive functioning. Motivational measures associated with task performance following training and testing also did not differ between the groups, suggesting equal levels of task engagement among participants.

The possibility of a circadian influence on performance was controlled in our study by conducting training and testing sessions at the same time of day (with the exception of the daytime groups). The finding that recognition memory (and fear-conditioned learning) is enhanced (or preserved) by arousal is consistent with animal and human studies demonstrating that neutral information is impaired and emotional information is facilitated (LaBar & Cabeza, 2006). We also report a differential response across dimensions of emotionality, valence, and arousal between the emotional and neutral treatment conditions, suggesting a possible interaction of these dimensions with sleep to enhance memory offline. In the same vein, subjective measures across positive (PA) and negative (NA) affect have also been shown to differentially influence emotional information processing (Anderson & Phelps, 2002; Liberzon et al., 2002), which may account for the biased emotional over neutral memory enhancing effects in the literature. Post hoc analyses between emotional dimensions and recognition memory revealed significant correlations in our study, suggesting that emotional valence (negative and positive) relative to neutral valence, as confirmed by previous studies (Cahill & McGaugh, 1998; Dolan,

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2002), enhances recognition memory. In the context of sleep-dependent memory consolidation, Gujar et al. (2011) found that participants who slept over the course of a 90-minute nap as compared to wake controls showed facilitated recognition and enhanced ratings of reward-related positive facial expressions, and that performance was dependent on REM sleep. Independent of emotional valence, Wagner et al. (2007) found that an 8-hr retention interval consisting of nocturnal sleep relative to an 8-hr wake control enhanced memory accuracy for faces (which arguably is one of the most emotionally salient stimuli). Contrastingly, neutral stimuli appear to have no beneficial effects on memory consolidation processes as compared to emotional stimuli. Research from our own laboratory (Alger, Lau, & Fishbein, 2010) have confirmed that neutral pictures of non-renowned people, objects, and landscapes in a visual recognition task failed to show a memory enhancing effect following a period of sleep as compared to wakefulness over a 90 minute daytime interval, yet showed a performance gain as a function of time (protracted testing), suggestive of a time-dependent effect associated with the strengthening of neutral memories. Interestingly, using a sleep/wake paradigm, Wamsley, Kensinger, Payne, and Stickgold (2010) reported a non-significant finding in memory performance across the phases using the unaltered version of the emotional slide show from Cahill et al. (1994); yet, found a sleep relative to wake performance benefit when the phases were collapsed (i.e., overall recognition memory). Such findings speak to the importance of arousal (and other dimensions of emotion) in facilitating memory processing (LaBar & Phelps, 1998), which as it may seem, acts as a mediator (perhaps interactively with sleep) in consolidating and enhancing emotional memories; this is especially evident in light of functional magnetic resonance imaging (fMRI) data showing a linear relationship between amygdala

activation during encoding and subsequent memory (Cahill et al., 1996; Hamann et al., 1999; Canli et al., 2000).

The contribution of sleep stage to memory processing was a definite variable of interest in our study as stated in our a priori hypothesis. We started out with the assumption that sleep relative to wakefulness would demonstrate a memory benefit (Stickgold, 2005), and that REM sleep, in particular, would prove beneficial for emotional as compared to neutral memories (see Walker, 2009). The results support our hypothesis (as discussed in the preceding paragraphs). Although there were no significant sleep stage and recognition memory correlations, the experimental manipulation produced an enhancement of emotional memories by sleep as compared to daytime wakefulness (80% vs. 74%), total sleep deprivation (70%), or half-night sleep deprivation (67%). Wagner et al. (2001) found that 3-hr retention intervals comprised of post-learning late-night sleep (rich in REM sleep and Stage 2 NREM sleep) relative to early-night sleep (predominantly SWS) benefited emotional (as compared to neutral) memories for texts, a finding, which is reported to last for years (Wagner et al., 2006). In a separate study, Wagner et al. (2007) found that overnight retention intervals comprised of sleep as compared to wakefulness (8 hours) enhanced recognition memory for faces, and that the enhancement was correlated with the amount of non-REM sleep.

For the most part, however, a stronger role for REM sleep (almost exclusively) has been made for sleep-dependent emotional memory processing. Rauchs et al. (2004) reports that episodic memories (such as that investigated in our study) defined by "autonoetic consciousness" (i.e., recall of factual, spatial, and temporal events in the context in which it originally occurred;

Tulving, 1983) are consolidated by REM sleep. In the truest sense, our study also committed to this definition of episodic memory given that our slide show simulated "aspects of real-life experiences by fusing multimodal perception with emotional and cognitive overtones" (Furman, Dorfman, Hasson, Davachi, & Dudai, 2007). Using an early/late sleep design, Rauchs et al. (2004) showed that performance for a task (closely associated with the Remember/Know paradigm) using factual (i.e., memorizing a word), spatial (i.e., its location), and temporal (i.e., what list it belongs to) components was affected in participants deprived of REM sleep relative to SWS. Although their finding (as well as ours) seems to counter the SWS/declarative memory and REM sleep/non-declarative memory dichotomy, it may in fact clarify such a distinction with respect to emotional and neutral stimulus specificity and its influence on consolidation processes. The mechanisms behind the influence of REM sleep on emotional memories are poorly understood at this point. However, research points to REM sleep as a possible brain-state implicated in emotional memory consolidation given its functional role in dreaming and unique biology (Stickgold, 2005). The neurophysiology work is corroborated by indirect evidence showing support for REM sleep as an active process in the consolidation emotional memories (see Walker & van der Helm, 2009 for a review). Nishida et al. (2009) found a significant relationship between the amount of REM sleep and a selective enhancement of emotional as compared to neutral memory in participants obtaining a 90-minute nap relative to no-nap (wake) controls. They also report that this offline emotional memory benefit is correlated with spectral activity in the theta-band range, which has been shown to support encoding processes during wakefulness (Buzsáki, 1998). Buzsáki (1998) posits a "hippocampal-neocortical dialogue" whereby memories initially dependent on the hippocampus become progressively less dependent on this structure as memories are reorganized in the neocortex during a so-called "transfer" of

stored representations via sharp wave bursts occurring in sleep. Using fMRI, Payne and Kensinger (2011) have identified differential post-learning consolidation processes and networks specific to emotional memories following sleep and wake retention intervals (12 hours respectively). They found that sleep relative to wakefulness engages a diffused "memory retrieval network" that includes prefrontal and parietal cortices as well as the amygdala and ventromedial prefrontal cortex (vMPFC)—areas of which show not only greater activation but also stronger connectivity. A similar finding using fMRI was reported across 3-days (Sterpenich et al., 2007) and 6-months (Sterpenich et al., 2009) retention intervals.

The present study yielded several main findings related to emotional arousal. The first, indicating that emotionally arousing stimuli elicits a distinctive autonomic response (i.e., heart rate deceleration) that is different from that of neutral stimuli (Bradley & Lang, 2000). The second, demonstrating that memory for emotional as compared to neutral information is enhanced across retention intervals filled with nocturnal sleep (Wagner et al., 2001) as compared to daytime wakefulness (Hu et al., 2006) or across a total night of sleep deprivation (Atienza & Cantero, 2008). These findings, independent of each other support and extend a substantial literature on the effects of emotional arousal on psychophysiology and sleep-dependent memory processing. Taken together, these findings offer a new perspective on the interaction between sleep, memory, and emotion.

4.2 Clinical implications and future direction

At present, there is no cure for PTSD, only symptom management through some form of pharmacotherapy and/or cognitive-behavior therapy (Otto, Smits, & Reese, 2004). The third finding herein demonstrated that the development of an episodic emotional memory can be blocked after a single night of sleep deprivation. This finding offers a behavioral intervention that has the potential to be used prophylactically as a way to impede the consolidation of negative thoughts or imagery. In fact, Wagner et al. (2006) have proposed that sleep deprivation "in the immediate aftermath of traumatic events" may serve as "a possible therapeutic measure to prevent a long-term engravement of these events in memory, thereby at least partly counteracting the development of PTSD" (p. 789). Research using fMRI data has confirmed that post-learning sleep deprivation stifles effective memory processing including for emotional information by diminishing activation and disengaging neural connectivity (Payne & Kensinger, 2011; Sterpenich et al., 2009). A similar finding was reported by Yoo et al. (2007) demonstrating that sleep deprivation (prior to learning) impairs the ability to form new memories as evidenced by a significant deficit in hippocampal activity and alertness networks of the brainstem and thalamus relative to sleep controls. Alternately, instead of exploiting the possibilities afforded to this particular brain-state, the latest advances on PTSD has focused almost exclusively on pharmacological interventions including experimental use of propranolol (Pitman et al., 2002) and morphine (Holbrook, Galarneau, Dye, Quinn, & Dougherty, 2010) to control symptoms, most notably, those of "increased arousal" i.e., falling or staying asleep, irritability, difficulty concentrating, hypervigilance, and exaggerated startle response (American Psychiatric Association [APA], 2000). Although these and other psychotropic drugs have shown effectiveness (Yehuda, 2002), they are not without concern over issues regarding margin of

safety, side effects, and medication adherence. For this reason, behavioral techniques have been advocated as an alternative and viewed in a much more favorable light.

The present study offers one such technique with a unique focus on REM sleep. Interestingly, we found that late-night, predominantly REM sleep and Stage 2 NREM sleep deprivation had an even greater impact on memory impairment than TSD. At least for emotional memories, REM sleep deprivation has previously been shown to selectively impair this specific type of memory (Greenberg et al., 1983; Cartwright et al., 1975). Aside from memory processes, disrupted REM sleep has been implicated in the development of PTSD as well as fear and anxiety states (Mellman, Bustamante, Fins, Pigeon, & Nolan, 2002). Traumatic intrusive imagery, a distressing symptom of PTSD (APA, 2000), has also been linked to REM sleep (Witvliet, 1997). On the whole, these findings (at least on the surface) seem to point to an intimate relationship between REM sleep and emotion. Exploring this relationship more closely through experimental paradigms such as our own may prove to be useful in impeding the consolidation of negative thoughts or imagery as experienced in certain clinical populations.

<center>**References**</center>

Abercrombie, H., Chambers, A., Greischar, L., & Monticelli, R. (2008). Orienting, emotion, and memory: Phasic and tonic variation in heart rate predicts memory for emotional pictures in men. *Neurobiology of Learning and Memory, 90,* 644-650.

Alger, S. E., Lau, H., & Fishbein, W. (2010). Delayed onset of a daytime nap facilitates retention of declarative memory. *PLoS ONE, 5(8),* e12131.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Rev.). Washington, DC: Author.

Anderson, A. K., & Phelps, E. A. (2002). Is the human amygdala critical for the subjective experience of emotion? Evidence of intact dispositional affect in patients with lesions of the amygdala. *Journal of Cognitive Neuroscience*, *14,* 709–720.

Antrobus, J. (1990). The neurocognition of sleep mentation: Rapid eye movements, visual imagery, and dreaming. In R. R. Bootzin & J. F. Kihlstrom (Eds.), *Sleep and cognition* (pp. 3-24). Washington, DC: APA.

Atienza, M., & Cantero, J. L. (2008). Modulatory effects of emotion and sleep on recollection and familiarity. *Journal of Sleep Research, 17,* 285–294.

Benca, R. (2008). Sleep in psychiatric disorders. American Academy of Chest Physicians (Eds.), *ACCP 2008 Sleep medicine board review* (pp. 71-87). Chicago: ACCP Sleep Institute.

Bloch, V., Hennevin, E., & Leconte, P. (1979). Relationship between paradoxical sleep and memory processes. In M. A. Brazier (Ed.), *Brain mechanisms in memory and learning: From the single neuron to man* (pp. 329–343). New York: Raven Press.

Born, J., & Wagner, U. (2004). Memory consolidation during sleep: Role of cortisol feedback. *Annals of the New York Academy of Sciences, 1032,* 198–201.

Bradley, M. M., Greenwald, M. K., Petry, M. C., & Lang, P. J. (1992). Remembering pictures: Pleasure and arousal in memory. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 18,* 379–390.

Bradley, M. M., & Lang, P. J. (2000). Affective reactions to acoustic stimuli. *Psychophysiology*, *37*, 204–215.

Brown, R., & Kulik, J. (1977). Flashbulb memories. *Cognition, 5,* 73-99.

Buzsáki, G. (1998). Memory consolidation during sleep: a neurophysiological perspective. *Journal of Sleep Research, 7,* 17–23.

Cahill, L., Prins, B., Weber, M., & McGaugh, J. L. (1994). Beta-adrenergic activation and memory for emotional events. *Nature, 371,* 702-704.

Cahill, L., Haier, R. J., Fallon, J., Alkire, M., Tang, C., Keator, D., et al. (1996). Amygdala activity at encoding correlated with long-term, free recall of emotional information. *Proceedings of the National Academy of Sciences, USA, 93,* 8016-8021.

Cahill, L., & McGaugh, J. L. (1998). Mechanisms of emotional arousal and lasting declarative memory. *Trends in Neuroscience, 21,* 294–299.

Canli, T., Zhao, Z., Brewer, J., Gabrieli, J. D. E., & Cahill, L. (2000). Event-related activation in the human amygdala associated with later memory for individual emotional experience. *Journal of Neuroscience, 20,* 1-5.

Carskadon, M. A., & Dement, W. C. (1994). Normal human sleep: An overview. In M. H. Kryger, T. Roth, & W. C. Dement (Eds.), *Principles and practice of sleep medicine* (2nd ed., pp. 16-25). Philadelphia: W. B. Saunders.

Cartwright, R. D., Lloyd, S., Butters, E., Weiner, L., McCarthy, L., & Hancock, J. (1975). Effects of REM time on what is recalled. *Psychophysiology, 12,* 561-568.

Christianson, S.-Å., & Loftus, E. F. (1987). Memory for traumatic events. *Applied Cognitive Psychology, 1,* 225–239.

37

Christianson, S.-Å. (Ed.) (1992a). *The handbook of emotion and memory: Research and theory*. Hillsdale, NJ: Erlbaum.

Christianson, S.-Å. (1992b). Emotional stress and eyewitness memory: A critical review. *Psychological Bulletin, 112,* 284-309.

Clark, R. E., & Squire, L. R. (1998). Classical conditioning and brain systems: A key role for awareness. *Science, 280,* 77–81.

Datta, S. (2007). Activation of pedunculopontine tegmental PKA prevents GABAB receptor activation-mediated rapid eye movement sleep suppression in the freely moving rat. *Journal of Neurophysiology, 97,* 3841–3850.

Dolan, R. J. (2002). Emotion, cognition, and behavior. *Science, 298,* 1191–1194.

Dolcos, F., & Cabeza, R. (2002). Event-related potentials of emotional memory: Encoding pleasant, unpleasant, and neutral pictures. *Cognitive, Affective, & Behavioral Neuroscience, 2,* 252-263.

Durmer, J. S., & Dinges, D. F. (2005). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology, 25,* 117-129.

Easterbrook, J. A. (1959). The effect of emotion on cue utilization and the organization of behavior. *Psychological Review, 66,* 183–201.

Eichenbaum, H. (2000). A cortical-hippocampal system for declarative memory. *Nature Reviews Neuroscience, 1,* 41–50.

Ellenbogen, J.M., Payne, J.D., & Stickgold, R. (2006). The role of sleep in declarative memory consolidation: Passive, permissive, active, or none? *Current Opinion in Neurobiology, 16,* 716–722.

Fishbein, W. (1970). Interference with conversion of memory from short-term to long-term storage by partial sleep deprivation. *Communications in Behavioral Biology, 5,* 171-175.

Fishbein, W., McGaugh, J. L., & Swarz, J. R. (1971). Retrograde amnesia: Electroconvulsive shock effects after termination of rapid eye movement sleep deprivation. *Science, 172,* 80-82.

Fishbein, W., & Gutwein, B. M. (1977). Paradoxical sleep and memory storage processes. *Behavioral Biology, 19,* 425-464.

Fishbein, W., Lau, H., De Jesús, R., & Alger, S. E. (2010). Sleep, neural reuse, and memory consolidation processes. [Peer commentary on the journal article "Neural reuse: A

fundamental organizational principle of the brain"]. *Behavioral and Brain Sciences, 33,* 273-273.

Freud, S. (1900/1996). *The interpretation of dreams.* New York: Gramercy Books.

Furman, O., Dorfman, N., Hasson, U., Davachi, L., & Dudai, Y. (2007). They saw a movie: Long-term memory for an extended audiovisual narrative. *Learning and Memory, 14,* 457-467.

Gais, S., & Born, J. (2004). Declarative memory consolidation: Mechanisms acting during human sleep. *Learning & Memory, 11,* 679–685.

Germain, A., Buysse, D. J., & Nofzinger, E. (2008). Sleep-specific mechanisms underlying PTSD. *Sleep Medicine Reviews, 12,* 185–195.

Greenberg, R., Pearlman, C, Schwartz, W. R., & Grossman, H. Y. (1983). Memory, emotion, and REM sleep. *Journal of Abnormal Psychology, 93,* 378-381.

Greenwald, M. K., Cook, E. W., & Lang, P. J. (1989). Affective judgment and psychophysiological response: Dimensional covariation in the evaluation of pictorial stimuli. *Journal of Psychophysiology, 3,* 51-64.

Gujar, N, McDonald, S. A., Nishida, M, & Walker, M. P. (2011). A role for REM sleep in recalibrating the sensitivity of the human brain to specific emotions. *Cerebral Cortex, 21,* 115-123.

Gupta, R., & Srinivasan, N. (2009). Emotions help memory for faces: Role of whole and parts. *Cognition and Emotion, 23,* 807-816

Hamann, S. B., Ely, T. D., Grafton, S. T., & Kilts, C. D. (1999). Amygdala activity related to enhanced memory for pleasant and aversive stimuli. *Nature Neuroscience, 2,* 289–293.

Hasselmo, M. (1999). Neuromodulation: acetylcholine and memory consolidation. *Trends in Cognitive Sciences, 3,* 351–359.

Heuer, F., & Reisberg, D. (1990). Vivid memories of emotional events: The accuracy of remembered minutiae. *Memory & Cognition, 18,* 496–506.

Hobson, J. A., & McCarley, R. (1977). The brain as a dream-state generator: An activation-synthesis hypothesis of the dream process. *American Journal of Psychiatry, 134*, 1335–1368.

Hoddes, E., Zarcone, V., Smythe, H., Phillips, R., & Dement, W. C. (1973). Quantification of sleepiness: A new approach. *Psychophysiology, 10,* 431-436.

Holbrook, T. L., Galarneau, M. R., Dye, J. L., Quinn, K., & Dougherty, A. L.   (2010).   Morphine use after combat injury in Iraq and post-traumatic stress disorder.   *New England Journal of Medicine, 362,* 110-117.

Hu, P., Stylos-Allan, M., & Walker, M. P.   (2006).   Sleep facilitates consolidation of emotional declarative memory.   *Psychological Science, 17,* 891-898.

James, W.   (1890/1981).   *The principles of psychology*.   Cambridge, MA: Harvard University Press.

Jenkins, J. B., & Dallenbach, K. M.   (1924).   Oblivescence during sleep and waking.   *American Journal of Psychology, 35,* 605–612.

Kahneman, D.   (1973).   *Attention and Effort.*   Englewood Cliffs, NJ: Prentice-Hall.

Karni, A., Tanne, D., Rubenstein, B. S., Askenasy, J. J., & Sagi, D.   (1994).   Dependence on REM sleep of overnight improvement of a perceptual skill.   *Science, 265,* 679-682.

Kensinger, E. A., & Corkin, S.   (2003).   Memory enhancement for emotional words: Are emotional words more vividly remembered than neutral words?   *Memory and Cognition, 31,* 1169-1180.

Kensinger, E. A. (2004). Remembering emotional experiences: The contribution of valence and arousal. *Reviews in the Neurosciences, 15,* 241-251.

Kensinger, E. A., & Corkin, S. (2004). Two routes to emotional memory: Distinct neural processes for valence and arousal. *Proceedings of the National Academy of Sciences, USA, 101,* 3310-3315.

LaBar, K. S., & Phelps, E. A. (1998). Arousal-mediated memory consolidation: Role of the medial temporal lobe in humans. *Psychological Science, 9,* 490–493.

LaBar, K. S., & Cabeza, R. (2006). Cognitive neuroscience of emotional memory. *Nature Neuroscience Reviews, 7,* 54-64.

Lacey, J. I., & Lacey, B. C. (1974). On heart rate responses and behavior: A reply to Elliott. *Journal of Personality and Social Psychology, 30,* 1-18.

Lang, P. J., Bradley, M. M., & Cuthbert, B. N. (1992). A motivational analysis of Emotion: Reflex-cortex connections. *Psychological Science, 3,* 44-49.

Lang, P. J., Greenwald, M. K., Bradley, M. M., & Hamm, A. O. (1993). Looking at pictures: Affective, facial, visceral, and behavioral reactions. *Psychophysiology*, *30*, 261-273.

Lara-Carrasco, J., Nielsen, T. A., Solomonova, E., Levrier, K., & Popova, A. (2009). Overnight emotional adaptation to negative stimuli is altered by REM sleep deprivation and is correlated with intervening dream emotions. *Journal of Sleep Research, 18,* 178–187.

Lau, H., Tucker, M. A., & Fishbein, W. (2010). Daytime napping: Effects on human direct associative and relational memory. *Neurobiology of Learning and Memory, 93,* 554-560.

LeDoux, J. E. (1996). *The emotional brain: The mysterious underpinnings of emotional life.* New York: Simon & Shuster.

LeDoux, J. E. (2000). Emotion circuits in the brain. *Annual Review of Neuroscience, 23,* 155–184.

Liberzon, I., Zubieta, J. K., Fig, L. M., Phan, K. L., Koeppe, R. A., & Taylor, S. F. (2002). μ-Opioid receptors and limbic responses to aversive emotional stimuli. *Proceedings of the National Academy of Sciences, USA, 99,* 7084-7089.

Loftus, E. F. (1979). *Eyewitness testimony.* Cambridge, MA: Harvard University Press.

Maquet, P., Peters, J., Aerts, J., Delfiore, G., Degueldre, C., Luxen, A., et al. (1996). Functional neuroanatomy of human rapid-eye-movement sleep and dreaming. *Nature, 383,* 163-166.

Maquet, P. (2001). The role of sleep in learning and memory. *Science, 294,* 1048–1052.

McGaugh, J. L. (2000). Memory—a century of consolidation. *Science, 287,* 248-251.

McGaugh, J. L. (2003). *Memory and emotion: The making of lasting memories.* New York: Columbia University Press.

McGaugh, J. L. (2004). The amygdala modulates the consolidation of memories of emotionally arousing experiences. *Annual Review of Neuroscience, 27,* 1–28.

Mellman, T. A., Bustamante, V., Fins, A. I., Pigeon, W. R., & Nolan, B. (2002). REM sleep and the early development of posttraumatic stress disorder. *American Journal of Psychiatry, 159,* 1696–1701.

Nielsen, T. A. (2000). A review of mentation in REM and NREM sleep: "Covert" REM sleep as a possible reconciliation of two opposing models. *Behavior and Brain Sciences, 23*, 851–866.

Nishida, M., Pearsall, J., Buckner, R. L., & Walker, M. P. (2009). Prefrontal theta during REM sleep enhances emotional memory. *Cerebral Cortex, 19,* 1158–1166.

Otto, M. W., Smits, J. A. J., & Reese, H. E. (2004). Cognitive-behavioral therapy for the treatment of anxiety disorders. *Journal of Clinical Psychiatry, 65,* 34-41.

Payne, J. D., Jackson, E. D., Ryan, L. Hoscheidt, S., Jacobs, W. J., & Nadel, L. (2006). The impact of stress on memory for neutral vs. emotional aspects of episodic memory. *Memory*, *14(1),* 1-16.

Payne, J. D., Jackson, E. D., Hoscheidt, S., Ryan, L., Jacobs, W. J., & Nadel, L. (2007). Stress administered prior to encoding impairs neutral but enhances emotional long-term episodic memories. *Learning & Memory, 14,* 861-868.

Payne, J. D., Stickgold, R., Swanberg, K., & Kensinger, E. A. (2008). Sleep and memory consolidation for complex emotional scenes. *Psychological Science, 19,* 781–788.

Payne, J. D., & Kensinger, E. A. (2011). Sleep leads to changes in the emotional memory trace: Evidence from fMRI. *Journal of Cognitive Neuroscience, 23,* 1285-1297.

Pearlman, C. (1979). REM sleep and information processing: evidence from animal studies. *Neuroscience and Biobehavioral Reviews, 3,* 57–68.

Peigneux, P., Laureys, S., Delbeuck, X., & Maquet, P. (2001). Sleeping brain, learning brain. The role of sleep for memory systems. *Neuroreport, 12*(18), A111-124.

Phelps, E.A. (2006). Emotion and cognition: Insights from studies of the human amygdala. *Annual Review of Psychology, 24 (57),* 27-53.

Pitman, R. K., Sanders, K. M., Zusman, R. M., Healy, A. R., Cheema, F., Lasko, N. B., et al. (2002). Pilot study of secondary prevention of posttraumatic stress disorder with propranolol. *Biological Psychiatry, 51,* 189–192.

Plihal, W., & Born, J. (1997). Effects of early and late nocturnal sleep on declarative and procedural memory. *Journal of Cognitive Neuroscience, 9,* 534–547.

Plihal, W., & Born, J. (1999). Effects of early and late nocturnal sleep on priming and spatial memory. *Psychophysiology, 36,* 571-582.

Rauchs, G., Bertran, F., Guillery-Girard, B., Desgranges, B., Kerrouche, N., Denise, P., et al. (2004). Consolidation of strictly episodic memories mainly requires rapid eye movement sleep. *Sleep, 27,* 395–401.

Rechtschaffen, A., & Kales, A. (1968). *A manual standardized terminology, techniques and scoring system for sleep stages of human subjects.* Bethesda, MD: U.S. Department of Health.

Ryle, G. (1949/2000). *The concept of mind.* London: Penguin Books.

Sharot, T., Martorella, E. A., Delgado, M. R., & Phelps, E. A. (2007). How personal
experience modulates the neural circuitry of memories of September 11. *Proceedings of
the National Academy of Sciences, USA, 104,* 389-394.

Smith, C. (1981). Learning and sleep states in the rat. In W. Fishbein (Ed.), *Sleep, dreams,
and memory* (pp. 19-35). New York: Spectrum Book Publications.

Smith, C. (1985). Sleep states and learning: a review of the animal literature. *Neuroscience
& Biobehavioral Reviews, 9,* 157–168.

Sterpenich, V., Albouy, G., Boly, M., Vandewalle, G., Darsaud, A., Balteau, E., et al.
(2007). Sleep related hippocampo-cortical interplay during emotional memory
recollection. *PLoS Biology, 5(11),* e282.

Sterpenich, V., Albouy, G., Darsaud, A., Schmidt, C., Vandewalle, G., Dang Vu, T. T., et al.
(2009). Sleep promotes the neural reorganization of remote emotional memory. *Journal
of Neuroscience, 29,* 5143–5152.

Stickgold, R., Hobson, J. A., Fosse, R., & Fosse, M. (2001). Sleep, learning and dreams:
Off-line memory reprocessing. *Science, 294,* 1052–1057.

Stickgold, R. (2005). Sleep-dependent memory consolidation. *Nature*, *437,* 1272-1278.

Terman, M., Rifkin, J. B., Jacobs, J., & White, T. M. (2001). *Morningness-Eveningness Questionnaire Self-Assessment Version (MEQ-SA)*. New York, NY: State Psychiatric Institute.

Tucker, M. A., Hirota, Y., Wamsley, E. J., Lau, H., Chaklader, A., & Fishbein, W. (2006). A daytime nap containing solely non-REM sleep enhances declarative but not procedural memory. *Neurobiology of Learning and Memory, 86,* 241–247.

Tucker, M. A., & Fishbein, W. (2009). The impact of sleep duration and subject intelligence on declarative and motor memory performance: how much is enough? *Journal of Sleep Research, 18,* 304-312.

Tulving, E. (1983). *Elements of episodic memory.* New York: Oxford University Press.

van der Helm, E., Gujar, N., & Walker, M. P. (2010). Sleep deprivation impairs the accurate recognition of human emotions. *Sleep, 33,* 335-342.

van Stegeren, A. H., Everaerd, W., Cahill, L., McGaugh, J. L., & Gooren, L. J. G. (1998). Memory for emotional events: differential effects of centrally versus peripherally acting β-blocking agents. *Psychopharmacology, 138,* 305-310.

Vertes, R. P., & Eastman, K. E. (2000). The case against memory consolidation in REM sleep. *Behavioral and Brain Sciences, 23,* 867-876.

Vogel, G. W., Vogel, F., McAbee, R. S., & Thurmond, A. J. (1980). Improvement of depression by REM sleep deprivation. New findings and a theory. *Archives of General Psychiatry, 37*, 247-253.

Wagner, U., Gais, S., & Born, J. (2001). Emotional memory formation is enhanced across sleep intervals with high amounts of rapid eye movement sleep. *Learning & Memory, 8,* 112-119.

Wagner, U., Fischer, S., & Born, J. (2002). Changes in emotional responses to aversive pictures across periods rich in slow-wave sleep versus rapid eye movement sleep. *Psychosomatic Medicine, 64,* 627–634.

Wagner, U., Hallschmid, M., Rasch, B., & Born, J. (2006). Brief sleep after learning keeps emotional memories alive for years. *Biological Psychiatry, 60,* 788-790.

Wagner, U., Kashyap, N., Diekelmann, S., & Born, J. (2007). The impact of post-learning sleep vs. wakefulness on recognition memory for faces with different facial expressions. *Neurobiology of Learning and Memory, 87,* 679–687.

Wagner, U. & Born, J. (2008). Memory consolidation during sleep: Interactive effects of sleep stages and HPA regulation. *Stress, 11(1),* 28-41.

Walker, M. P., & Stickgold, R. (2006). Sleep, memory and plasticity. *Annual Review of Psychology, 10,* 139–166.

Walker, M. P. (2009). The role of sleep in cognition and emotion. *Annals of the New York Academy of Sciences, 1156,* 168–197.

Walker, M. P., & van der Helm, E. (2009). Overnight therapy? The role of sleep in emotional brain processing. *Psychological Bulletin*, *135*, 731–748.

Wamsley, E. J., Kensinger, E. A., Payne, J. D., & Stickgold, R. (2010). The impact of sleep on emotional episodic memory performance. *Abstract, Associated Professional Sleep Societies (APSS).*

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54,* 1063-1070.

Wilson, M. A., & McNaughton, B. L. (1994). Reactivation of hippocampal ensemble memories during sleep. *Science, 265,* 676–679.

Witvliet, C. V. (1997). Traumatic intrusive imagery as an emotional memory phenomenon: A review of research and explanatory information processing theories. *Clinical Psychology Review, 17,* 509–536.

Wolf, L. F. & Smith, J. K. (1995). The consequence of consequence: Motivation, anxiety, and test performance. *Applied Measurement in Education, 8,* 227-242.

Yehuda, R. (2002). Post-traumatic stress disorder. *New England Journal of Medicine*, *346*, 108-114.

Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit formation. *Journal of Comparative and Neurological Psychology, 18,* 459-482.

Yoo, S. S., Gujar, N., Hu, P., Jolesz, F. A., & Walker, M. P. (2007). The human emotional brain without sleep—A prefrontal amygdala disconnect. *Current Biology, 17,* R877–R878.

Table 1

ECG (in bpm) for the slide show (mean ± SEM)

	Pre-ECG	Phase 1	Phase 2	Phase 3	Post-ECG
Neutral (*n* = 50)	72.22 ± 1.30	73.14 ± 1.23	73.56 ± 1.31	73.68 ± 1.29	73.32 ± 1.31
Emotional (*n* = 50)	71.44 ± 1.48	72.05 ± 1.38	69.49 ± 1.53	72.63 ± 1.48	72.36 ± 1.43
t-test	t(98) = .397, p = .692	t(98) = .588, p = .558	t(98) = 2.016, p = .047	t(98) = .536, p = .593	t(98) = .494, p = .622

Table 2
Subjective emotional ratings (neutral vs. emotional; mean ± SEM)

	Emotionality	Valence	Arousal
Phase 1	(08.60 ± 0.72 vs. 09.94 ± 0.77)	(22.78 ± 0.57 vs. 22.50 ± 0.79)	(10.74 ± 0.98 vs. 10.34 ± 0.93)
t-test	p = .208	p = .774	p = .768
Phase 2	(16.04 ± 0.96 vs. 30.94 ± 0.91)	(16.04 ± 0.96 vs. 30.94 ± 0.91)	(18.84 ± 1.01 vs. 27.64 ± 1.26)
t-test	p < .0001	p < .0001	p < .0001
Phase 3	(10.20 ± 0.82 vs. 16.60 ± 0.89)	(15.00 ± 0.29 vs. 11.88 ± 0.45)	(10.48 ± 0.78 vs. 15.60 ± 0.95)
t-test	p < .0001	p < .0001	p < .0001

Table 3
Sleep log data

Treatment Group	8-hr Sleep	8-hr TSD	8-hr Daytime	48-hr Daytime	4-hr (half-night)
†5 night average (mean ± SEM)	(7.42 ± 0.21)	(7.26 ± 0.22)	(7.9 ± 0.98)	(7.68 ± 1.19)	(7.36 ± 0.97)
One-way ANOVA	$F_{(4,95)} = 1.323$, $p = .267$				
‡Recovery night (mean ± SEM)	(7.93 ± 0.32)	(8.45 ± 0.47)		(7.95 ± 0.41)	(7.48 ± 0.45)
One-way ANOVA	$F_{(3,76)} = 0.901$, $p = .445$				

Note. †average TST of 5 sleep nights for each group prior to training session; ‡average TST for the recovery night.

Table 4
Presleep variables (mean ± SEM)

	SSS		SOS		DS	
	Pre-training	Pre-testing	Post-training	Post-testing	Pre-training	Pre-testing
8-hr Sleep	3.85 ± 0.34	1.75 ± 0.20	37.45 ± 0.88	38.40 ± 0.89	4.03 ± 0.24	4.13 ± 0.18
8-hr TSD	2.80 ± 0.28	2.15 ± 0.26	35.55 ± 1.30	36.60 ± 0.96	4.48 ± 0.26	4.73 ± 0.24
4-hr (half-night)	3.30 ± 0.29	2.20 ± 0.30	36.95 ± 1.16	38.05 ± 1.35	4.28 ± 0.28	4.68 ± 0.31
8-hr Daytime	3.30 ± 0.33	2.85 ± 0.34	35.90 ± 0.95	36.80 ± 1.03	4.40 ± 0.24	4.43 ± 0.31
48-hr Daytime	3.35 ± 0.36	2.55 ± 0.32	37.55 ± 1.13	39.35 ± 0.92	4.55 ± 0.31	4.95 ± 0.29
ANOVA	$F = 1.39$, $p = .243$	$F = 2.12$, $p = .085$	$F = 0.69$, $p = .598$	$F = 1.21$, $p = .311$	$F = 0.59$, $p = .673$	$F = 1.35$, $p = .258$

Note. SSS, Standford Sleepiness Scale; SOS, Student Opinion Scale; DS, Digit Span

Table 5

Sleep parameters

Sleep parameter	≈4-hr (half-night) (*n* = 20)		≈8-hr (full-night) (*n* = 20)		Estimate "2nd ½ Sleep" half-night
	Mean	SEM	Mean	SEM	
Sleep latency (min.)	4.28	0.62	4.78	0.69	
TST (min.)	243.93	2.11	454.20	4.19	
S1 (min/%)	(11.03/4.68%)	(2.87/1.29)	(14.63/3.28%)	(2.85/0.65)	(3.60/1.71%)
S2 (min/%)	(108.88/44.63%)	(5.49/2.19)	(222.33/48.99%)	(9.06/2.00)	(113.45/53.95%)
SWS (min/%)	(92.55/37.82%)	(6.11/2.38)	(106.98/23.41%)	(9.63/1.98)	(14.43/6.86%)
REM (min/%)	(31.48/12.91%)	(2.87/1.19)	(110.28/24.31%)	(4.51/1.02)	(78.80/37.48%)

Note. TST, total sleep time; S1, Stage 1; S2, Stage 2; SWS, slow wave sleep; REM, rapid eye movement sleep.

Table 6

Recognition memory (% correct answers) for the slide show (mean ± SEM)

|	Phase 1	Phase 2	Phase 3	OR
Neutral (*n* = 50)				
8-hr Sleep	55.31 ± 2.68	73.13 ± 4.76	56.67 ± 3.24	62.73 ± 2.88
8-hr TSD	55.94 ± 2.00	69.38 ± 4.11	57.08 ± 2.85	61.02 ± 2.43
4-hr (half-night)	51.88 ± 4.60	60.94 ± 3.99	57.92 ± 3.75	56.82 ± 2.68
8-hr Daytime	61.88 ± 3.22	73.75 ± 2.43	64.17 ± 2.86	66.82 ± 2.30
48-hr Daytime	59.06 ± 2.73	66.25 ± 3.05	53.33 ± 5.30	60.11 ± 2.87
One-way ANOVA	F = 1.449, p = .234	F = 1.980, p = .114	F = 1.129, p = .355	F = 1.928, p = .122
Emotional (*n* = 50)				
8-hr Sleep	60.31 ± 2.84	80.63 ± 1.60	70.42 ± 2.94	70.45 ± 1.76
8-hr TSD	58.75 ± 2.83	70.00 ± 3.61	60.00 ± 3.52	63.07 ± 1.70
4-hr (half-night)	63.75 ± 3.30	67.19 ± 3.82	56.67 ± 3.74	63.07 ± 2.88
8-hr Daytime	63.75 ± 2.04	74.06 ± 2.64	66.67 ± 3.04	68.30 ± 1.96
48-hr Daytime	60.00 ± 3.12	70.31 ± 2.43	59.17 ± 4.92	63.52 ± 2.79
One-way ANOVA	F = .648, p = .631	F = 3.124, p = .024	F = 2.396, p = .064	F = 2.314, p = .072

Note. OR, Overall Recognition
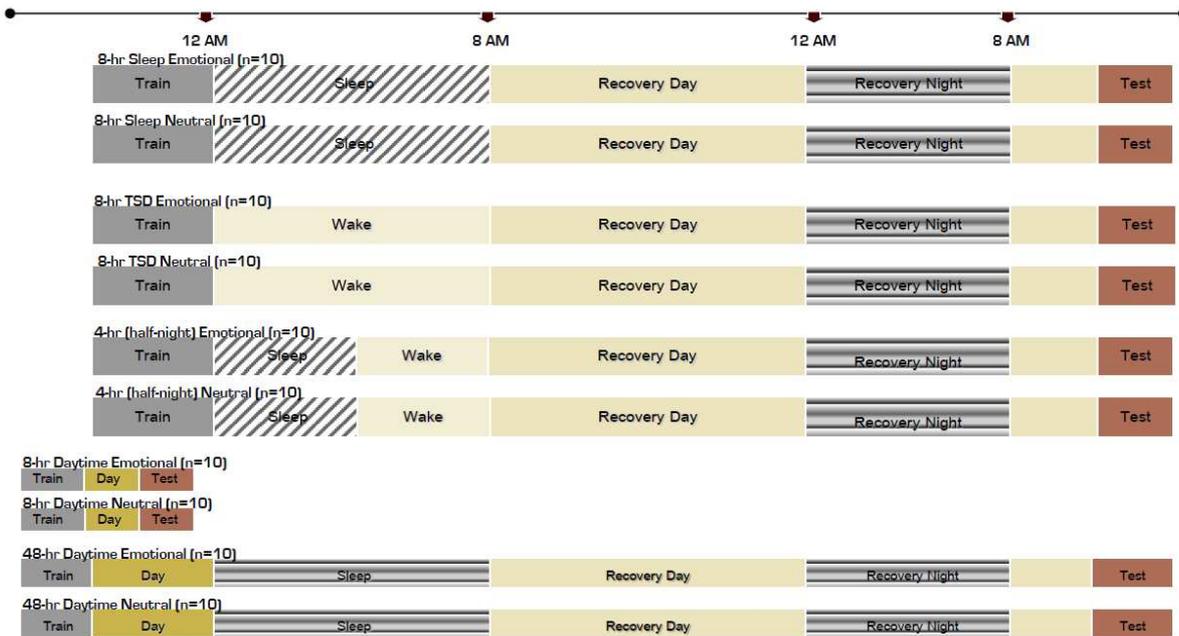
Figure 1

Experimental design. Participants were randomly assigned one of ten groups: an 8-hr sleep group consisting of emotional (n = 10) and neutral (n = 10) conditions; an 8-hr total sleep deprivation (TSD) group consisting of emotional (n = 10) and neutral (n = 10) conditions; a 4-hr late-night sleep deprivation group consisting of emotional (n = 10) and neutral (n = 10) conditions; an 8-hr daytime (wake control) group consisting of emotional (n = 10) and neutral (n = 10) conditions; and a 48-hr daytime (wake control) group consisting of emotional (n = 10) and neutral (n = 10) conditions. See Design and Procedure section for a description of the experimental protocol.
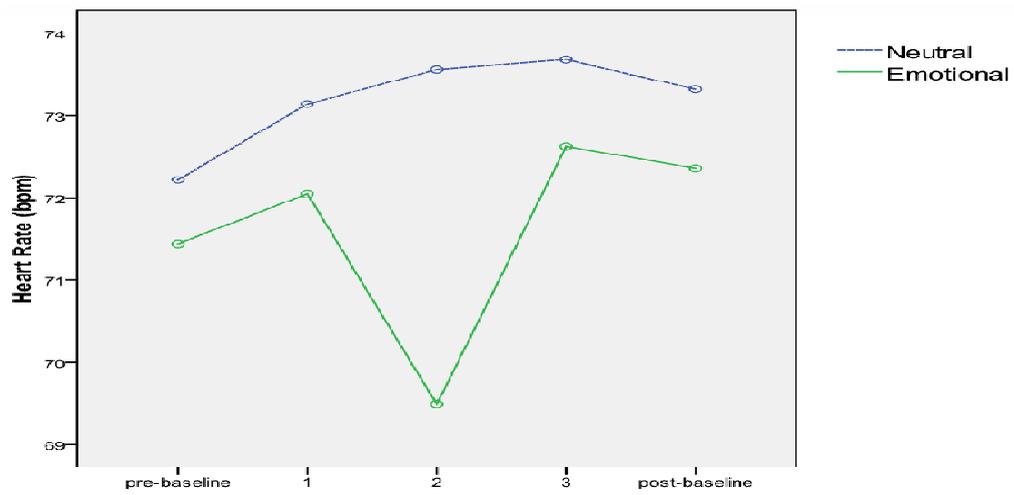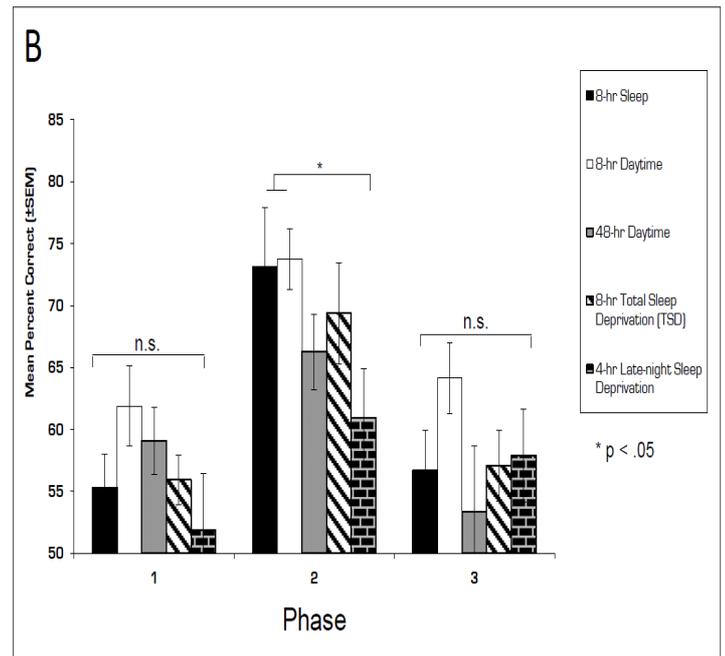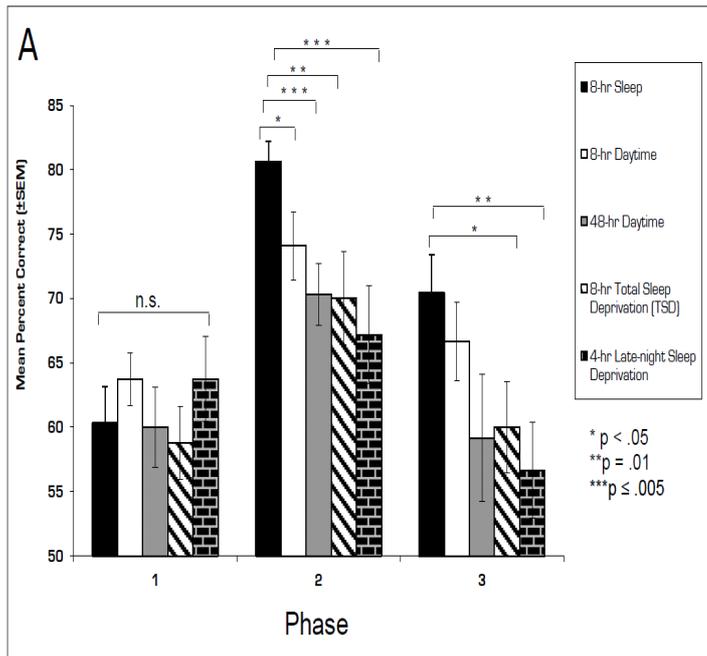
Figure 2
Heart rate data for the neutral (in blue hatch marks) and emotional (in green solid marks) conditions was measured prior to viewing the slide show (pre-baseline), during phase 1, phase 2, phase 3, and following the slide show (post-baseline).

Figure 3

Recognition memory (% of correct response) measured 2 days after training following an at home recovery sleep night (except for the 8-hr daytime groups). (A) Emotional treatment condition: Note the significant enhancement (black vs. white histogram) of memory retention in the 8 hr Emotional sleep group compared to the lack of enhancement in the Neutral treatment group (B). Additionally, note the impairment of the enhancement (hash and brick histograms) in the TSD and late-night sleep deprivation groups. Of further note, the late-night deprivation group produces as much memory impairment as the full-night of deprivation.

Appendix A

Emotional Slide Show	Neutral Slide Show

Emotional Slide Show

1. A mother and her son are leaving home in the morning.
2. She is taking him to visit his father's workplace.
3. The father is the chief laboratory technician at the nearby hospital.
4. They check before crossing a busy road.
5. While walking along, the boy is struck by runaway car, which critically injures him.
6. At the hospital, the staff prepare the emergency room, to which the boy is rushed.
7. All morning long, surgeons struggled to save the boy's life
8. Although successful at reattaching his severed feet, the boy died after the surgery from massive blood loss.
9. After the surgery, while the father stayed with the boy, the mother left to phone her other child's preschool.
10. Feeling distraught, she phones the pre-school to tell them she will soon pick up her child.
11. Heading to pick up her child, she hails a taxi at the number nine bus stop.

Neutral Slide Show

1. A mother and her son are leaving home in the morning.
2. She is taking him to visit his father's workplace.
3. The father is the chief laboratory technician at the nearby hospital.
4. They check before crossing a busy road.
5. While walking along, they pass the scene of a minor accident, which the boy finds interesting.
6. At the hospital, the staff are preparing for a practice emergency drill, which the boy will watch.
7. All morning long, surgeons practice the standard emergency drill procedures.
8. Special make-up artists were able to create realistic looking injuries on actors for the drill.
9. After the drill, while the father stayed with the boy, the mother left to phone her other child's preschool.
10. Running late, she phones the pre-school to tell them she will soon pick up her child.
11. Heading to pick up her child, she hails a taxi at the number nine bus stop.