THE EFFECTS OF SLEEP ON REPETITION PRIMING

by

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LIST OF ABBREVIATIONS

Abbreviation	Description
EEG	Electroencephalogram
Hz	Hertz, unit of measurement
REM	Rapid Eye Movement
SWS	Slow Wave Sleep

ABSTRACT

Repetition priming is a type of implicit memory in which previous exposure to a stimulus facilitates more efficient processing of that stimulus at later presentations. This facilitation is simultaneously accompanied by a decrement in processing for stimuli that are similar, but not identical to the primed stimulus. This is called the antipriming effect. Sleep-dependent memory consolidation is the process by which recently learned information is strengthened during sleep and can occur at both synaptic and systems levels. Synaptic consolidation during rapid eye movement (REM) sleep in particular has been shown to enhance repetition priming effects. The purpose of the study was to investigate the effects of an afternoon nap on priming and antipriming effects. The current study had four phases that included first presenting 50 audio recordings of the names of common objects one at a time while participants judged how much they liked or disliked that object. In the next phase, participants were presented with 100 common object images one at a time for 15 ms each. Participants were instructed to identify the object as quickly and as accurately as possible. In the third phase, participants were presented with another set of 50 object images one at a time, and similar to the first phase, they were asked to report how much they liked or disliked what the object represented. Following the third phase, there was a break in which participants either remained in the lab and took a 90-minute nap (sleep group) or left the lab and were instructed not to sleep for the same amount of time (wake group). Following the break, participants were presented with 100 object images one at a time for 15 ms each and like

the second phase, participants were instructed to identify the presented object as quickly and as accurately as possible. Half of the object images presented were previously presented in the third phase and therefore acted as the *primed* stimuli. The remaining 50 object images were not previously shown and thus acted as the *antiprimed* stimuli. Priming and antipriming effects in the response time measure were larger in the sleep group than in the wake group. However, this effect was not mediated by REM sleep. Additionally, there were no significant correlations between time spent asleep and the magnitude of priming or antipriming effects. This study replicates and broadens previous findings by demonstrating that sleep during an afternoon nap enhance priming effects and this is the first study to show that sleep also enhances antipriming effects.

I. LITERATURE BACKGROUND

Introduction

Sleep plays a critical role in the process of stabilizing and strengthening long-term memories through a process known as sleep-dependent memory consolidation (Genzel & Wixted, 2017). Research today continues to investigate how sleep-dependent memory consolidation specifically contributes to different types of memory. Memory is organized into implicit or explicit types. Implicit memory refers to knowledge that is accessible without conscious effort and explicit memory involves conscious awareness when accessed (Schacter, 1987). Implicit memories often take the form of automatic processes that are learned and reinforced over time, such as riding a bike. Explicit memory is the information that an individual can consciously recall, such as the type of bike they own. While these types of memory are distinct, they can work seamlessly together or separately to facilitate learning and task performance. The famous case of patient H.M. helped to firmly establish that these types of memory are in fact distinct. After an experimental bilateral medial temporal lobectomy, H.M. lost his ability to form new explicit long-term memories but retained the ability to form new *implicit* memories. This case, among other amnesiacs with similar damage, provide support for the notion that implicit and explicit memory are distinct types of memory (Milner, Corkin, & Teuber, 1986).

This study explores types of implicit memory referred to as repetition priming and antipriming. Repetition priming occurs when previous exposure to a stimulus facilitates increased efficiency in later processing of that same stimulus (Schacter & Buckner, 1998). Antipriming is an effect related to repetition priming in which previous exposure

to a stimulus impairs the processing of new stimuli. This impairment is believed to be positively correlated with the degree of similarity between the *primed* (i.e., previously exposed) stimulus and the new stimulus. For example, presenting an individual with an image of a desk will facilitate (i.e., prime) the identification of a desk at a later presentation. This priming will also simultaneously impair (i.e., antiprime) the identification of a similar object, like a piano (Marsolek, 2008). The purpose of the current study is to investigate how sleep-dependent memory consolidation may influence repetition priming and antipriming.

Implicit Memory and Repetition Priming

Implicit memory allows procedures and patterns to be automatically and unconsciously encoded and later accessed. Over time, as information is repeatedly exposed, subsequent access of the information will be faster and more efficient. Prominent examples of implicit memory are classical conditioning, procedural memory, and priming (Schacter, 1987). Researchers have identified many factors that contribute to the occurrence of priming effects. Both perceptual and conceptual aspects of stimuli can induce priming. Perceptual priming is *modality* specific, meaning that the similarities between physical forms of stimuli creates a sensitivity to priming effects. An example of perceptual priming is presenting an individual with an incomplete outline of a bike and later observing a faster and more accurate identification for a full outline of the bike. The perceptual or physical features of the stimuli in both presentations are related and that relationship facilitates priming. Conceptual relationships between stimuli are not modality specific. Rather, conceptual priming is dependent on the categorial relationships between stimuli. An example of conceptual priming would be priming an individual with

a category like transportation and later witnessing a faster identification for the word or image of a bicycle. The categorical relationships between the primed category and later stimulus that fits into that category induce a priming effect (Farah, 1989; Schacter & Buckner, 1998). Repetition priming, which is the facilitation of processing due to previous exposure, is sensitive to stimuli that are identical in their conceptual or perceptual relationships (Gotts, Chow, & Martin, 2012).

Measuring Repetition Priming Effects

There are many different ways that repetition priming can be measured. For this literature review, the focus will be on the perceptual priming of objects. Repetition priming tasks are designed to observe differences in accuracy in identifying stimuli or in response times for responding to stimuli that are either previously presented or that are novel. Perceptual priming can be tested by presenting stimuli to an individual during what is called an 'encoding period' without providing any explicit instructions to remember the stimuli that are presented. To ensure that participants are sufficiently attending to the presented stimuli, researchers will typically ask them to identify the presented stimuli as quickly and as accurately as possible. Additionally, researchers may ask participants to make subjective judgments on what each of the presented stimuli represent. These procedures are designed to avoid eliciting any type of intentional encoding or rehearsal. In some tasks, the accuracy and response times from this encoding phase are used as a baseline measure to compare later trials against, as all stimuli in this phase are assumed to be equally novel. Subsequent to the encoding phase, researchers can choose to implement a break/distraction task or otherwise move straight into the testing phase. In the testing phase, participants are presented with a mix of stimuli that

were either presented earlier in the encoding phase or that are novel. In these designs, participants will be asked to name/identify presented stimuli as quickly and as accurately as possible. Repetition priming effects are observed when there in a significant increase in the speed and/or accuracy for naming/identifying stimuli that were presented a second time (i.e., these stimuli were presented during encoding phase and again during the testing phase) compared with the performance for stimuli encoding/baseline phase (i.e., when stimuli were presented for the first time) (Schacter & Buckner, 1998).

When a participant consciously remembers seeing a stimulus previously, it may affect their subsequent performance. Therefore, tasks that measure implicit memory must be careful in their design to try and exclude interference from explicit memory. Researchers try to minimize contamination from explicit memory by using short presentation times for stimuli and excluding instructions that would imply that stimuli need to be remembered. Researchers may also use a technique called masking in their design that presents a 'masked' image immediately after a primed image in order to try and eliminate any rehearsal or explicit processing. This theory behind this technique is that it works by first presenting an initial 'prime' image that triggers activation of representations in the visual system that are related to the image, however, a 'masked' image is presented immediately after in order to interrupt processing and thus limit explicit identification and rehearsal (Eddy, Schnyer, Schmid, & Holcomb, 2007). To truly eliminate the possibility that explicit memory is contributing to performance, these tests need to be administered to amnesic patients that have impaired explicit memory, but not impaired implicit memory, like patient H.M. In most cases, the results from healthy controls in these priming tasks are replicated in samples of amnesiacs. These ancillary

findings help to support the idea that these tasks are an adequate measure of implicit memory effects (Roediger & McDermott, 1993).

Theoretical Explanations of Repetition Priming and Antipriming

The study of repetition priming provides insights into how implicit knowledge is represented and organized in the brain. It is known that visual information is processed through a network of visual pathways which receive and process this information at different levels and within different structures in the brain. After visual information is received through the eyes, it is relayed on to the lateral geniculate nucleus of the thalamus, then further on to the primary visual cortex (V1) and finally temporal cortex. While visual information travels through this cortical visual stream, responses to visual information become associated with increasingly more complex features of the stimuli (Prasad & Galetta, 2011). Evidence of priming and antipriming support the idea of interactive and overlapping visual representations in the brain.

A visual stimulus, like an image of a desk, is comprised of a combination of discrete features, i.e., table legs are combined with a flat top and drawers to make a whole representation of a desk. Each of these features (the legs, flat top, and drawers) has their own representational processing unit in the brain. These representational processing units are small groups of neurons that respond to a specific feature of a stimulus. Exposure, and especially repeated exposure, to a particular combination of features (e.g., the features that make up a desk) creates and strengthens connections between these representational units, creating a *distributed representation* of the visual stimulus (Hinton, McClelland, & Rumelhart, 1986; McClelland & Rumelhart, 1985; Tanaka, 1993). Distributed representations allow for the efficient processing of familiar visual

information because the brain is able to represent more stimuli within the same representational space with distributed representations, as opposed to using local representations, where one unit represents one stimulus.

In repetition priming, repeated exposure to a stimulus creates small adjustments that strengthen the synaptic connections between the units that make up the distributed representation of the stimulus. This results in more efficient processing of that stimulus at later presentations (Marsolek, 2002). This process is referred to as 'sharpening'. The *sharpening* of connections between representational units means that poorly responding neurons (those that are less centrally involved in representing the features of the stimulus) tend to weaken and drop-off and/or cease firing such that only neurons that are more centrally involved in representing the features of the stimulus remain firing and thus strengthen their connections (Gotts et al., 2012). These are the neural underpinnings of the repetition priming effect, but this process can come with a cognitive cost that results in antipriming.

Antipriming occurs because objects are represented with distributed representations. Therefore, objects that share features will also share representational units. When a stimulus is primed, the connections between units involved in representing the stimulus are strengthened, but connections with units not involved in representing the primed stimulus but are involved with representing other stimuli are weakened. This creates a deficit in processing a novel non-primed stimulus that shares representational units with the previously primed stimulus. An example of antipriming would be that after being primed to a stimulus like a desk, an individual would have a deficit in processing a stimulus that is not a desk but shares similar features and representational units, like a

piano, that also has legs and a flat top like a desk does. This processing deficit is behaviorally observable in slower response times and less accurate identification of similar but novel stimuli compared with primed stimuli (Marsolek, 2008).

Measuring Antipriming Effects

Antipriming effects can be observed through priming tasks in which baseline response times and accuracy values can be compared to those collected in a testing phase that consists of repeated stimuli (primed) and novel but similar stimuli (antiprimed). When response times are significantly slower and accuracy values are significantly lower for antiprimed stimuli compared to baseline, antipriming effects have been observed (Marsolek, 2008).

It has only been in the past few decades that antipriming was identified as a distinct effect in implicit memory. Through a series of experiments, Marsolek and colleagues (2006, 2010) modified previous repetition priming designs with a new consideration for antipriming. In their 2006 task design, there were four phases. In the first phase participants heard auditory recordings of 50 common object names and they reported judgements for how much they liked/disliked what the object represented. This portion of the task was included to have a period where participants were processing stimuli, but not processing visual stimuli, which would occur in subsequent stages. In the second phase, baseline measures of response times and accuracy in identification were collected in response to 15 ms presentations of 100 drawings of common objects (i.e. visual processing). In the third phase, participants were presented with 50 new object drawings for 3 s each that they judged based on how much they liked or disliked what the object represented. In the final phase, participants were presented with 100

drawings of common objects for 15 ms, just like in the baseline/second phase. However, half of the drawings of objects presented were repeated from the third phase and acted as primed stimuli and the other half were novel and acted as antiprimed stimuli. This design was applied to samples of healthy young adults, a sample of amnesic patients with impaired explicit memory abilities, and a sample of healthy controls matched in age, IQ, and education level to the amnesic patients. The inclusion of amnesic patients is critical in defining antipriming as an implicit memory effect, because it allows for the exclusion of explicit memory effects that may still be at work in healthy controls. In the group of healthy controls matched to the amnesiacs and in the healthy young adults, results showed faster response times and increased accuracy for images of primed objects compared with baseline objects, and decreased accuracy and slower response times for antiprimed objects compared with baseline objects, replicating previous findings. In amnesic patients, accuracy values were lower and response times were slower compared with the control groups, but priming and antipriming effects were still present. It should be noted that throughout the experiments with young healthy adults, amnesic patients, and their matched controls, the magnitude of the antipriming effect, i.e. the degree to how much slower and less accurate responses were to antiprimed objects compared with baseline objects, was smaller than the magnitude of the priming effect. While the literature on visual antipriming is small, these observations have been replicated. Using Chinese characters as visual stimuli, but using the same task design, another lab was able to replicate the previous findings by Marsolek and colleagues (2006, 2010) (Zhang, Fairchild, & Li, 2017).

Repetition Priming and Antipriming: Neural Evidence

Using functional magnetic resonance imaging, researchers have observed the neural response patterns associated with repetition priming and antipriming. Neuroimaging methods show that repeated exposure to stimuli over time is correlated with a decrease in activation of the sensory cortices. Specifically, there are reduced hemodynamic responses in posterior processing areas in the occipital lobe for visual stimuli that have been repeatedly exposed (Schacter & Bucker, 1998). This decrease in neural activation for primed stimuli is referred to as the 'repetition suppression' effect. The sharpening effect mentioned earlier is believed to be responsible for this drop off in neural activity for primed stimuli (Gotts, et al., 2012). Antipriming has only been observed using neurological methods in a small set of experiments but has been found to be associated with an increase in neural activity in posterior cortical regions that is significantly higher than that seen in priming and in baseline measures. This increase in activation for antiprimed stimuli is believed to reflect the physical changes that occur through the increased synaptic activity needed to re-establish and strengthen connections between representational units that were weakened due to recent priming of other similar objects (Marsolek et al., 2010).

Sleep

Sleep-dependent memory consolidation plays a significant role in stabilizing and strengthening information in long-term memory. Researchers can investigate these effects experimentally through testing memory performance across periods of sleep compared with across periods of wakefulness. Through these types of experimental designs, researchers have found in several instances that periods of sleep enhance performance on

several types of memory tasks (Diekelmann & Born, 2010; Rauchs, Desgranges, Foret, & Eustache, 2005; Wagner, Hallschmid, Verleger, & Born, 2002). One hypothesis to explain why sleep has this effect claims that during sleep, the brain is able to process previously experienced information without active interference from new incoming information or environmental stimulation (Diekelmann & Born, 2010).

Stages of Sleep

Sleep stages are characterized by their distinct electrophysiological features observed through electroencephalographic (EEG) measures of bioelectric neural activity. Stage 1 sleep is a brief period of rest in which breathing becomes deeper and the muscular system relaxes. This stage of sleep is characterized by bursts of activity in the alpha and theta frequencies (8-13 Hz and 4-8 Hz, respectively). Stage 2 sleep is distinguished by the presence of sleep spindles, which are short bursts of activity (1-3 s) in the sigma range (10-15 Hz), along with K-complexes, which are high-amplitude biphasic waves. Prominent low-frequency activity in the delta band (0-4 Hz) characterizes periods of slow-wave sleep (SWS). Rapid eye movement (REM) sleep is often referred to as a 'paradoxical sleep' as the EEG activity witnessed during this period of sleep is similar to that during wakefulness. REM sleep is typically characterized by increased theta activity in the hippocampus and EEG recorded at the scalp exhibits activity in the beta range (14 Hz +). These stages typically progress in the same order: Stage 1, Stage 2, SWS, REM, throughout one sleep cycle that takes between 90-120 minutes on average. In typical nocturnal sleep, SWS is more prominent in cycles that occur earlier in the night, compared to cycles later in the night when REM sleep is more prominent (Berry & Wagner, 2015; Diekelmann & Born, 2010; Seibt & Frank, 2019).

Relationships between Sleep and Memory

After initial encoding, recently acquired memories undergo a process of consolidation, which can occur at synaptic and systems levels of the brain. During synaptic consolidation, synapses involved in representing a memory are strengthened through long-term potentiation. Synaptic consolidation occurs within minutes of learning but can also occur later during sleep. During systems consolidation, there is a reorganization of neural circuits involved in representing a memory. For example, over time, explicit memory representations will typically become less dependent on the hippocampus and more dependent on relevant neocortical regions. This reorganization occurs through continued activation of the memory over time. It is believed that systems consolidation occurs primarily during sleep.

For a memory to undergo consolidation processing during sleep, it must first be reactivated, and this reactivation process has been linked to hippocampal sharp wave ripples that predominantly occur during non-REM sleep (Stage 2 and SWS). Systems consolidation is thought to progress during SWS, as slow oscillations during this stage provide a candidate mechanism for the exchange of information across widespread regions of the brain. However, synaptic consolidation of memories reactivated during non-REM sleep most likely occurs during REM sleep, as the physiological mechanisms associated with long-term potentiation are more consistent with the molecular events that are known to occur during REM (Born & Wilhelm, 2012; Bramham & Srebro, 1989; Diekelmann & Born, 2010; Genzel & Wixted, 2017; Stickgold & Walker, 2007).

Gains in explicit memory task performance are typically associated with non-REM sleep whereas gains in implicit memory task performance are associated with both

non-REM and REM sleep (Diekelmann & Born, 2010). While implicit memory tasks in general seem to benefit from both non-REM and REM sleep, experiments using implicit repetition priming tasks have shown that REM sleep appears to be a stronger contributor to priming effects than non-REM sleep. A study by Plihal and Born (1999) tested differences in memory after the first half of a night of sleep, which has relatively more SWS relative to REM sleep, compared with memory after the second half of a night of sleep, which contains more REM relative to SWS. The memory tests included an implicit memory word-stem priming task and an explicit memory spatial rotation task. The results showed larger gains in explicit memory when memory was tested after the first half of the night whereas larger gains in implicit memory were observed when testing occurred after the second half of the night. The researchers concluded that the late night, or REM-dominated sleep, provided a critical opportunity for enhanced synaptic consolidation and strengthening of representations that were formed while awake.

A study by Wagner and colleagues (2002), investigated how early or late-night nocturnal sleep affected performance on an implicit repetition priming memory task that used faces as stimuli. Similar to the Plihal and Born (1999) study mentioned previously, these researchers found that improved performance on an implicit memory priming task was associated with REM-dominated sleep more so than sleep dominated by SWS activity (Wagner et al., 2002). Contrary to these studies, another study observing the effects of a full night of sleep on priming in healthy older adults and older adults with amnesic mild cognitive impairment by Westerberg and colleagues (2012) found that priming effects were actually diminished after a full night of sleep. These results may be

specific to older adults, as the Plihal et al. (1999) and Wagner et al. (2002) designs had only young college-aged men as their participants.

There is no existing literature on the effects of sleep on antipriming. Therefore, the current study aims to not only add to the body of literature on the relationship between sleep and priming by examining how priming may be affected by sleep during an afternoon nap, but also to include for the first time how sleep may influence antipriming. Based on results obtained in prior studies examining the effects of sleep on priming, the hypothesis of this experiment is that synaptic consolidation during REM sleep may result in enhanced repetition priming effects and therefore should also simultaneously amplify antipriming effects.

II. METHOD

The data for this study was originally collected between 2012 and 2015 in the lab of Dr. Carmen Westerberg at Texas State University. In this experiment, priming and antipriming were examined for both visual objects and linguistic stimuli. Priming and antipriming for each stimulus type (visual objects, linguistic) were assessed across four phases, three of which occurred before a break that included a nap monitored with EEG to assess how long participants spent in each sleep stage, or wakefulness during the break. The order in which the first three phases for the two different stimulus types were given was counterbalanced across participants. In addition, the order in which the last phase was given for the two different stimuli types was also counterbalanced, such that if a participant completed the first three phases with the visual objects first before the break, the participant also completed the last phase with the visual objects first. The current analyses will focus only on visual object priming and antipriming.

Participants

Participants were undergraduate students at Texas State University between the ages of 18-29 that were compensated with either $30 or with extra credit points in psychology classes. Exclusion criterion was any current diagnosis of a sleep disorder. A total of 64 participants were randomly assigned to either a wake (n = 32, female=16, male=16) or sleep group (n = 32, female=16, male=16). However, due to various errors such as equipment failure, researcher error, or lack of adequate sleep recorded, not all of those who participated were included in the final analyses. The final number of included participants for each analysis are detailed in the results section.

Materials

Before the start of the experiment, participants completed several questionnaires. The Pittsburgh Sleep Quality Index is a questionnaire that gathers information on participant's sleep quantity and quality for the past month. There are 19 total questions divided into 7 components designed to assess sleep quality, latency, duration, habitual sleep efficiency, disturbances, the use of sleep medication, and daytime disturbances from the past month. Scores from these components are combined to create a global sleep quality score that range from 0 to 21, in which higher scores reflect a worse overall sleep quality (see Appendix A; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The validity and reliability of this questionnaire has been studied and replicated in both clinical and non-clinical populations (Mollayeva et al., 2016). The Karolinska Sleep Log gathers information about sleep quantity and quality from the preceding night through 13 questions. The format of this questionnaire is 6 free response questions followed by 7 questions that ask participants to rate their responses on a scale from 1-5 (Akerstedt, Hume, Minors, & Wayerhouse, 1994). As a part of the Vasterbotten Environmental Heath Study, the construct and criterion validity of this questionnaire was tested in a sample of over 3,000 Swedish citizens and found to be a reliable and valid measure of sleep quality and disturbances (Nordin, Akerstedt, & Nordin, 2013; see Appendix B). Following the break, all participants completed the Stanford Sleepiness Scale which evaluates current feelings of sleepiness. This scale includes a single response that asks participants to rate on a scale from 1 to 7, with lower responses relating to higher feelings of wakefulness and higher scores relating to feelings of sleepiness (see Appendix C; Hoddes, 1972). This questionnaire is valid and reliable when used either repeatedly or at

a uniform time in a study design as it only measures a participant's current state of sleepiness/wakefulness (Shahid, Wilkinson, Marcu, & Shapiro, 2011).

Experimental Stimuli

The stimuli used in this experiment were 250-line drawings of objects, referred to as object images, and 250 audio recordings of the names of those objects. For practice trials, an additional 9 objects and audio recordings were used. Using visual stimuli from this dataset ensured that objects presented in any of the separate conditions would not significantly overlap semantically or perceptually with other objects. A standard desktop computer was used for stimuli presentation. A microphone was used to collect voice-triggered response times.

General Procedure

The procedure of this experiment contained four phases. Stimuli were counterbalanced to assure that each object appeared an equal number of times across conditions and participants (see Figure 1 for an overview of the procedure).

In the first phase of the task, 50 auditory word recordings were presented, and participants were asked to report on a scale of 1-4, using the keyboard, how much they liked the object the word represented (1 = dislike very much, 4 = like very much).

The second phase provided baseline measures of object naming accuracy and response times. A short trial period was introduced to accustom participants to the task, which consisted of 9 practice trails. The main task included 100 common objects images that were presented for 15 ms each while participants were instructed to identify the presented object image as quickly and as accurately as possible by speaking the name of each presented object into the microphone placed in front of them. A researcher in the

room compared participants responses against a printed list of the correct responses in order to record and grade accuracy. Object images were counterbalanced in their presentation location which was either 4.3 degrees above or below the central fixation point.

In the third phase, participants were presented with 50 new common object images in the center of the screen for 3 s each and asked to judge how much they liked what the object represented on a scale from 1-4 (1 = dislike very much, 4 = like very much).

Following the third phase, participants either remained in the lab and took a 90-minute nap (sleep group) or left the lab and were instructed not to sleep (wake group). Those in the sleep group were given a dark quiet room with a bed located in the lab while electroencephalography (EEG) was recorded.

In the fourth and final phase, after another short trial phase of 9 trails, the main task of 100 common object images were presented one at a time for 15 ms each and, like the second phase, participants were instructed to identify each presented object image as quickly and as accurately as possible by speaking into the microphone in front of them. A researcher in the room compared participants responses against a printed list of the correct responses in order to record and grade accuracy. Half of the object images presented in this phase were also presented during the third phase (primed). The remaining 50 object images were new (anti-primed). Object images were again counterbalanced in their presentation location which was either 4.3 degrees above or below the central fixation point.

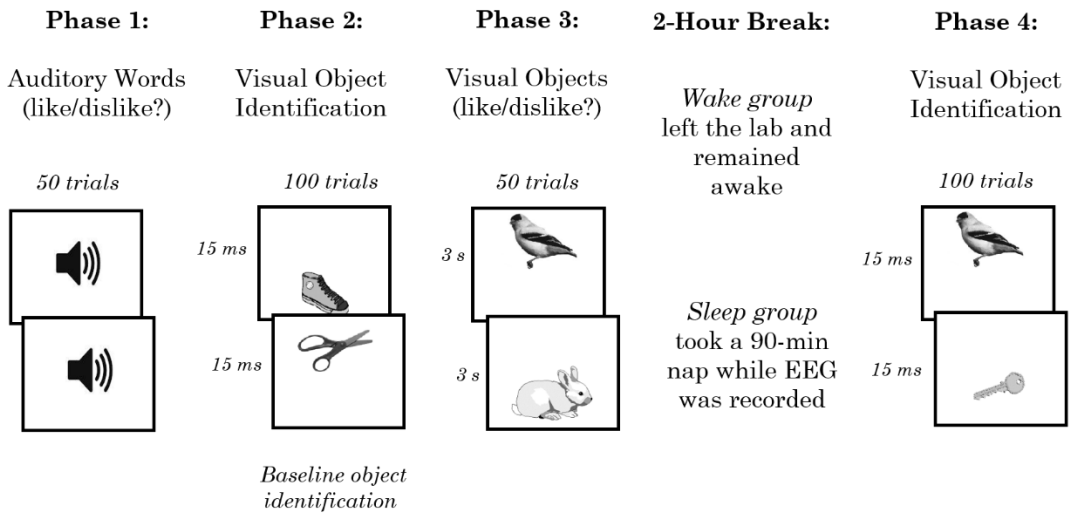

Figure 1. Procedure for experiment.

Dependent Measures

Measures of baseline performance were computed from the accuracy and response time values for the objects presented in the second phase of the experiment. Performance for primed material was computed *from* the accuracy and response time values from objects that were presented in the fourth phase that were primed in the third phase. Finally, performance for antiprimed material was computed from the accuracy and response times for naming the new object images included in the fourth phase.

Electroencephalogram

EEG equipment set up was completed for those in the sleep group before beginning the experiment. EEG was measured by placing seven electrodes embedded in a cap on the scalp at standard recording sites (C3, C4, Fpz, F3, F4, O1, O2). Two digitally-linked mastoid electrodes were used as an average reference online for each electrode. The data were digitally sampled with a 250 Hz sampling rate and amplified and band-pass filtered between 0.25-100 Hz (NuAmps amplifier, Compumedics Neuroscan).

Additionally, electrodes were places on the chest, chin, and next to the eyes for measures of electrocardiography, electromyography, and electrooculography to assist in assigning sleep stages.

Sleep stages were assigned offline for each 30 s epoch of EEG data based on standard criteria from the American Academy of Sleep Medicine (Iber et al., 2007).

III. RESULTS

Order of Experimental Procedures

To investigate whether the order of experimental tasks (visual versus linguistic) influenced response time or accuracy measures, two-tailed independent samples *t*-tests were completed on both response time and accuracy measures for the baseline, primed, and antiprimed conditions between those participants that received the linguistic task first and those who received the visual task first. No significant differences were found. A summary of the *t*-tests including *p* values are reported in Table 1.

Table 1

Average response times, proportion correct, and *t* and *p* values for comparisons between participants who completed the visual tasks first and participants who completed the linguistic tasks first by condition (baseline, primed, antiprimed). Standard deviations are in parentheses.

	Linguistic First	Visual First	*t* value	*p* value
Baseline Accuracy	0.85 (0.05)	0.85 (0.07)	0.19	0.85
Primed Accuracy	0.90 (0.07)	0.91 (0.07)	0.27	0.60
Antiprimed Accuracy	0.85 (0.08)	0.85 (0.10)	1.09	0.86
Baseline Response Time	850.72 (112.32)	861.24 (91.20)	0.60	0.72
Primed Response Time	796.22 (95.96)	830.14 (90.20)	-0.80	0.21
Antiprimed Response Time	855.34 (131.71)	887.59 (100.47)	-0.39	0.35

Response Times

There were 49 participants in the analysis of response times. There were 25 participants in the wake group (13 females and 12 males). Three participants were missing data from phase 2 or 4, and 4 participants were excluded because response times were not recorded for more than half of the trials during phase 2 or 4. There were 24 participants included in the sleep group (13 females and 11 males). Six participants were excluded for either failing to sleep or sleeping very little (i.e. < 20 mins) and 2 participants were excluded because response times were not recorded for more than half of the trials during phase 2 or phase 4. Additional data cleaning included excluding response times for individual trials under 300 ms and then deleting any trials that were beyond 2.5 standard deviations from the mean for each participant within their phase 2 (baseline condition) and phase 4 (primed/antiprimed conditions). After removing outlier response times, mean response times were calculated for each condition for each participant and then averaged across groups. The average response times and standard deviations for the baseline, primed, and antiprimed conditions for both the wake and sleep groups are reported in Table 2.

Table 2

Mean response times (ms) for baseline, primed, and antiprimed objects for wake and sleep groups. Standard deviations in parentheses.

	Baseline	Primed	Antiprimed
Wake Group	826.54 (119.81)	803.72 (113.45)	827.41 (133.94)
Sleep Group	834.39 (85.27)	787.55 (54.76)	842.42 (75.73)

The magnitude of the difference from baseline response times was computed separately for primed and antiprimed objects. To examine priming, mean response times from the primed condition were subtracted from the mean response times from the baseline condition for each participant. To examine antipriming, mean response times from the baseline condition were subtracted from the mean response times from the antiprimed condition for each participant. Mean values and standard deviations for the wake and sleep groups for these measures are reported in Table 3. These response time difference measures represent the size of priming and antipriming effects and were used as the dependent variable in a 2 x 2 mixed model ANOVA with group (wake or sleep) and condition (primed or antiprimed) as the independent variables. The was a significant main effect of group, $F(1, 47) = 5.72$, $p = 0.02$. This means that the magnitude of the priming and antipriming effects overall were significantly larger in the sleep group than in the wake group. The main effect of condition (primed or antiprimed) was not significant, $F(1, 47) = 2.37$, $p = 0.13$. This means that there was no difference between the magnitude of the priming effect and the magnitude of the antipriming effect. The condition X group interaction was not significant, $F(1, 47) = 0.18$, $p = 0.67$.

Table 3

Mean response time difference from baseline (ms) for primed and antiprimed conditions for the wake and sleep groups.

	Primed	Antiprimed
Wake Group	22.82 (61.46)	0.87 (63.78)
Sleep Group	46.84 (80.73)	8.03 (83.04)

Note: Standard deviations are in parentheses. Larger values reflect a greater difference from baseline response times.

To further investigate the main effect of group, two two-tailed independent samples *t*-tests were completed to compare the magnitude of the priming and antipriming effects between the wake and sleep groups. While the magnitude of the priming effect was numerically larger in the sleep group, the difference from the wake group was not significant, $t(47) = 1.18$, $p = 0.25$. Similarly, the magnitude of the antipriming effect was numerically larger in the sleep group, but it was not significantly different from the wake group, $t(47) = 0.34$, $p = 0.74$. These results suggest that the benefit of sleep for antipriming and priming is modest.

Additional two-tailed paired samples *t*-tests were conducted to see if the differences between baseline and primed or baseline and antiprimed conditions were significant within each group. In the wake group, mean response times from the primed condition were faster than those from the baseline condition, but only marginal in significance, $t(24) = 1.86$, $p = 0.08$. Mean response times from the antiprimed condition were not significantly different than those from the baseline condition, $t(24) = 0.07$, $p =$

0.95. In the sleep group, response times from the primed condition were significantly faster than those from the baseline condition $t(23) = 2.84$, $p = 0.009$. However, mean response times from the antiprimed condition were not significantly different than those from the baseline condition, $t(23) = 0.47$, $p = 0.64$. These results indicate that priming effects were present in the sleep group and to a lesser extent, in the wake group. However, no significant antipriming effects were present in either group.

Additional t-tests were completed to assure that the difference between priming and antipriming effects between groups were not due to initial differences in baseline performance. A two-tailed independent samples *t*-test compared response times for the baseline condition and found no significant difference between the wake and sleep groups, $t(48) = 0.26$, $p = 0.79$. This lack of difference between baseline response times confirms that the larger priming and antipriming effects found in the sleep group are not due to ceiling or floor effects in the wake group.

Accuracy

There were 55 participants included in the accuracy analyses. There were 29 included from the wake group (15 females and 14 males). Three participants were excluded because accuracy data was not recorded. There were 26 participants included from the sleep group (14 females and 12 males). Six participants were excluded because they failed to sleep, or they slept very little (< 20 mins). The exclusion criterion was failure to correctly identify at least half of the object images and was applied separately to phase 2 and 4. Mean accuracy values were calculated for each condition for each participant and then averaged across group. Mean accuracy values and standard deviations for each condition for each group are reported in Table 4.

Table 4

Mean proportion correct for baseline, primed, and antiprimed conditions for wake and sleep groups. Standard deviations are in parentheses.

	Baseline	Primed	Antiprimed
Wake Group	0.82 (0.09)	0.90 (0.05)	0.82 (0.09)
Sleep Group	0.84 (0.08)	0.91 (0.06)	0.82 (0.11)

The magnitude of the difference from baseline proportion correct was computed separately for primed and antiprimed objects. To compute the size of the priming effect for accuracy, mean baseline proportion correct was subtracted from mean proportion correct from the primed condition for each participant. To compute the size of the antipriming effect, mean proportion correct from the antiprimed condition was subtracted from mean proportion correct for the baseline condition for each participant. Means and standard deviations for these difference measures are reported in Table 5. Similar to the response time analysis, these values were used as the dependent variable in a 2 x 2 mixed model ANOVA with group (wake or sleep) and condition (primed or antiprimed) as the independent variables. There was a significant main effect of condition, $F(1, 53) = 24.77$, $p < 0.001$. The magnitude of the priming effect was significantly larger than the magnitude of the antipriming effect. There was not a significant main effect of group, $F(1, 53) = 0.11$, $p = 0.75$, indicating there is no difference in the magnitude of priming and antipriming between wake and sleep groups. There was also not a significant group X condition interaction, $F(1, 53) = 1.39$, $p = 0.25$.

Table 5

Mean difference from baseline proportion correct and standard deviations for the primed and antiprimed conditions for the wake and sleep groups. Standard deviations are in parentheses.

	Primed	Antiprimed
Wake Group	0.08 (0.08)	0.00 (0.06)
Sleep Group	0.07 (0.06)	0.02 (0.07)

Note: Larger values reflect a greater difference from baseline proportion correct.

To further investigate the main effect of condition, follow up paired-samples *t*-tests were completed to compare the magnitude of priming and antipriming within groups. For the wake group, the magnitude of the priming effect was significantly larger than the magnitude of the antipriming effect, $t(28) = 4.18$, $p < 0.001$. Similarly, in the sleep group, the magnitude of the priming effect was significantly larger than that of the antipriming effect, $t(25) = 2.87$, $p = 0.008$. This result is consistent with past literature in which the priming effect is more robust than the antipriming effect (Marsolek, 2008).

Additional two-tailed paired samples *t*-tests were conducted to see if the differences between baseline and primed conditions or baseline and antiprimed conditions were significant in each group. These tests confirmed that the in the wake group, proportion correct from the primed condition was significantly higher than baseline proportion correct, $t(28) = 5.61$, $p < 0.001$. However, baseline proportion correct was not significantly different from antiprimed proportion correct, $t(28) = 0.21$, $p = 0.83$. The sleep group followed a similar pattern, such that primed proportion correct was

significantly higher than baseline proportion correct, $t(25) = 6.26$, $p < 0.001$. However, there was not a significant difference in proportion correct between the baseline and the antiprimed conditions, $t(25) = 1.27$, $p = 0.21$. These results show that there was a significant priming effect in both groups but there was no significant antipriming effect found in either group.

To investigate the possibility of initial baseline differences between groups, an independent two-tailed *t*-test was conducted and found no significant difference between baseline accuracy proportion correct between the wake and sleep groups, $t(54) = 0.75$, $p = 0.46$. This lack of difference in baseline performance indicates that the observed pattern of priming and antipriming effects across groups were not contaminated by initial differences between groups.

Sleep

In the sleep group, time spent in stage 1, stage 2, slow-wave sleep (SWS), REM, and total sleep time were computed for each participant. Time spent in non-REM (NREM) sleep was then computed as the sum of time spent in stage 2 and SWS. Within the sleep group, sub-groups were created to represent those who achieved REM sleep (REM group, n=9) and those who had no significant recorded REM sleep (5 minutes or less; No-REM group, n=15). The motivation behind this sub-grouping was based on previous research that suggests that REM sleep in particular may enhance priming effects (Plihal & Born 1999; Wagner et al., 2002). Mean time spent in NREM, REM, and total sleep time for the No-REM and REM groups are reported in Table 6.

Table 6

Average time spent in each sleep stage (min) for No-REM and REM groups. Standard deviations in parentheses.

	TST	NREM	REM
No-REM Group (n = 15)	60.33 (12.55)	50.07 (9.84)	0.33 (1.29)*
REM Group (n = 9)	71.56 (15.29)	47.83 (12.85)	15.44 (5.65)

Note: TST = Total Sleep Time, NREM = Non-REM.

*Includes one participant who had 5 minutes of recorded REM sleep. All other participants in this group had no REM sleep.

Accuracy and response time measures were analyzed across the No-REM and REM groups to investigate if REM sleep during a nap had a significant effect on priming and antipriming. Two 2 x 2 mixed model ANOVAs were completed to investigate whether there were any significant differences in the magnitude of priming and antipriming between the No-REM and REM sleep groups, using the response time and accuracy difference measures described above as the two dependent variables.

Response Times

The first ANOVA used the subtracted values that represented the magnitude of difference in response times from baseline as the dependent measure, with condition (antiprimed, primed) and group (REM, Non-REM) as the independent variables. The mean and standard deviations of these values for each condition for both groups are reported in Table 7. There was not a significant main effect of condition, $F(1, 22)= 1.48$, $p = 0.24$. While the numerical magnitude of the priming effect was larger than the

magnitude of the antipriming effect, this difference was not significant. There was not a significant main effect of group, $F(1, 22) = 0.74$, $p = 0.40$, nor a significant group X condition interaction, $F(1, 22) = 0.08$, $p = 0.77$.

Table 7

Mean response time difference from baseline (ms) and standard deviations for primed and antiprimed conditions for the No-REM and REM groups. Standard deviations are in parentheses.

	Primed	Antiprimed
No-REM Group	40.19 (87.58)	8.71 (91.91)
REM Group	57.90 (71.35)	6.90 (70.97)

Note: Larger values reflect a greater difference from baseline response times.

Accuracy

The second 2 x 2 mixed model ANOVA was completed to investigate any differences in accuracy between REM and no-REM groups. This ANOVA used the subtracted measures that represented the magnitude of difference in proportion correct from baseline as the dependent measure, with condition (antiprimed, primed) and group (REM, No-REM) as the independent variables. Mean values and standard deviations of these values for both groups are reported in Table 8. There was a significant main effect of condition, $F(1, 22) = 6.31$, $p = 0.02$, such that the priming effect was significantly larger than the antipriming effect. There was not a significant main effect of group, $F(1,$

22) = 0.00, p = 0.95, and the group X condition interaction was also not significant, F(1, 22) = 0.05, p = 0.83.

Table 8

Mean difference from baseline proportion correct and standard deviations for primed and antiprimed conditions for the No-REM and REM groups. Standard deviations are in parentheses.

	Primed	Antiprimed
No-REM Group	0.07 (0.06)	0.01 (0.05)
REM Group	0.06 (0.06)	0.02 (0.09)

Note: Larger values reflect a greater difference from baseline proportion correct.

Follow up two-tailed paired samples t-tests were completed within groups using the subtracted values to compare primed and antiprimed conditions. In the no-REM group, the magnitude of the priming effect was significantly larger than the magnitude of the antipriming effect, t(14) = 2.41, p = 0.02. In the REM group, the magnitude of the priming effect was not significantly larger than the magnitude of the antipriming effect although a numerical trend was present, t(8) = 1.24, p = 0.25.

Correlations

Within the sleep group, additional analyses investigated if there were any relationships between sleep and priming or antipriming effects. In these analyses, similar to those from the response time and accuracy analyses, subtracted values that reflect the magnitude of the priming and antipriming effects were used. Pearson's correlations

between the subtracted measures for response times and proportion correct for the primed and antiprimed conditions and total sleep time, non-REM sleep time, and REM sleep time were conducted. With multiple comparisons being made, a Bonferroni correction was calculated by dividing an alpha of 0.05 by the total number of comparisons (12) to get a corrected critical alpha of 0.004. These analyses found no significant correlations between response time and accuracy priming or antipriming effects and total sleep time, time spent in non-REM sleep, or time spent in REM sleep. Correlations for REM sleep only included participants who had more than 5 minutes of REM sleep (n=9). A summary of the correlations can be found in Table 9.

Table 9

Correlation coefficients between time spent in Non-REM sleep, REM sleep, and total sleep time (min) and priming and antipriming values. *P* values are in parentheses.

	TST	NREM	REM
Antipriming accuracy	0.38 (0.06)	0.36 (0.09)	-0.2 (0.59)
Antipriming response times	0.27 (0.20)	0.09 (0.66)	0.41 (0.24)
Priming accuracy	0.01 (0.98)	0.04 (0.30)	0.11 (0.76)
Antipriming response times	-0.07 (0.78)	0.06 (0.78)	-0.22 (0.55)

Note: TST = total sleep time, NREM = Non-REM

Questionnaires

Sleep questionnaires were analyzed to determine if there were any significant differences between sleep characteristics of the sleep and wake groups. For the Pittsburg

Sleep Quality Index (PSQI), participants responded to a range of questions relating to their sleep habits within the past month. Scores from each question were combined to create a global PSQI score that ranged from 0 to 21, in which higher scores reflected worse overall quality of sleep. A two-tailed, independent-samples *t*-test was conducted and showed that there was no significant difference in global sleep quality scores between the wake and sleep groups (Table 10). The Karolinska Sleep Log (KSL) collected information on how participants slept the night immediately preceding the experiment (see Table 11). Participant responses to the 13 questions about sleep quantity and quality were compared between groups using two-tailed, independent-samples *t*-tests and no significant differences were present between the wake and sleep groups. Mean values, standard deviations, *t* values, and *p* values are reported in Table 11. Finally, for the Stanford Sleepiness Scale, participants selected a single response that best reflected their current level of sleepiness. Scores from this questionnaire ranged from 1 to 7, in which lower scores reflected a higher feeling of wakefulness and higher scores reflected an increased feeling of sleepiness. To compare responses between groups, a two-tailed, independent-samples *t*-test was conducted. No significant difference in sleepiness level after the break between the wake and sleep groups was found. Means, standard deviations, and *t*-test values are reported in Table 11.

Table 10

Mean values, standard deviations, *t* value and *p* value for the Pittsburgh Sleep Quality Index global scores for wake and sleep groups.

	Wake Group	Sleep Group	*t* value	*p* value
Global PSQI Score	11.65 (6.86)	8.4 (6.08)	1.9	0.12

Note: Global PSQI scores may range from 0 to 21, where higher scores indicate worse sleep quality.

Table 11

Mean responses, *t* values, and *p* values for each Karolinska sleep log question for the wake and sleep groups.

	Wake Group	Sleep Group	*t* value	*p* value
What time did you go to bed last night?	12:30 am	1:30 am	0.54	0.59
What time did you wake up this morning?	7:30 am	7:45 am	0.66	0.52
How long did you sleep? (hours)	6.75 (1.32)	6.44 (1.32)	0.75	0.45
How long did it take you to fall asleep? (in minutes)	30.52 (33.45)	19.00 (17.39)	1.36	0.18
How many times did you wake up while you slept?	0.75 (0.79)	0.58 (1.07)	0.60	0.55
How many minutes were you awake after falling asleep? (in minutes)	19.25 (61.40)	6.11(12.51)	0.92	0.36
How did you sleep? (1=very poor/5=very well)	3.75 (0.74)	4.21 (0.85)	1.86	0.07
Did you feel refreshed after your sleep? (1=not at all/5=yes very)	3.04 (1.07)	3.21 (0.85)	0.56	0.58
Did you sleep soundly? (1=very restless/5=very soundly)	3.71 (1.04)	4.00 (1.00)	0.93	0.36
Did you sleep throughout the time allotted? (1=woke up too early/5=slept until the end)	4.00 (1.29)	4.53 (1.02)	1.47	0.15
How easy was it for you to wake up? (1=very easy/5=very difficult)	3.17 (0.96)	3.37 (1.21)	0.60	0.55
How easy was it for you to fall asleep? (1=very easy/5=very difficult)	2.38 (1.28)	2.16 (1.17)	0.57	0.57
How much did you dream? (1=none/5=a lot)	2.42 (1.41)	2.68 (1.29)	0.63	0.53

Table 12

Mean sleepiness level, *t* value, and *p* value from the Stanford Sleepiness Scale for the wake and sleep groups.

	Wake Group	Sleep Group	*t* value	*p* value
Total score	2.50 (1.10)	3.16 (1.21)	1.80	0.10

Note: Scores may range from 1 to 7, with lower scores reflecting increased alertness, and higher scores reflecting increased sleepiness.

IV. DISCUSSION

The goal of the current experiment was to investigate if priming and antipriming effects would be influenced by sleep-dependent memory consolidation processes during an afternoon nap. It was hypothesized that an afternoon nap would enhance both priming and antipriming effects. Supporting this hypothesis, priming and antipriming effects in the response time measure were larger in the sleep group than in the wake group. This finding replicates previous work (Plihal & Born, 1999; Wagner et al., 2002) showing larger priming effects following periods of nocturnal sleep compared with periods of wakefulness. This result expands the literature by demonstrating enhanced priming following sleep during an afternoon nap compared with an equivalent period of wakefulness while using visual objects as primed/antiprimed stimuli, while previous designs used other forms of stimuli, (i.e., facial features or word stems). This is also the first study to demonstrate that sleep-dependent memory consolidation enhances antipriming.

Another hypothesis in this study was that within the sleep group, those who achieved REM sleep would show larger enhancements in priming and antipriming compared with sleep participants who had little or no REM sleep. Previous experiments investigating the effects of sleep on priming (Plihal & Born, 1999; Wagner et al., 2002) demonstrated that overnight REM sleep mediated enhanced priming effects. These experiments used a split night testing design in which participants were tested on two nights. On one-night, participants went through a learning phase before they went to sleep and then were tested after the first three hours of sleep with this period of sleep being dominated by SWS activity. On the other night, participants went through a

learning phase after three hours of sleep and then were tested after sleeping another three hours, this later period being more dominated by REM sleep. The researchers found that the testing that followed the later night REM dominated sleep period had enhanced priming effects compared to testing that followed the earlier night SWS dominated sleep period. This finding is consistent with the theory that synaptic consolidation within circumscribed brain areas primarily takes place during REM sleep (Diekelmann & Born, 2010) and may be the mechanism through which priming is enhanced.

In the current study, the size of priming and antipriming effects did not differ between those who achieved REM sleep during the nap and those who did not. There are multiple reasons why REM sleep may not have been implicated in enhanced priming and antipriming in the current study. In this experiment, stimuli included visual objects, whereas previous studies used words or faces as stimuli (Plihal & Born, 1999; Wagner et al., 2002). The difference in results may indicate that REM sleep may be more relevant for priming with words and facial features than the visual object stimuli that were used in the current design. Another difference in this study compared with past studies was that not all participants achieved REM sleep. Within the sleep group, only nine participants achieved more than 5 minutes of REM sleep. In previous studies by Plihal and Born (1999) and Wagner et al., (2002), all participants in the sleep groups achieved REM sleep and included between 20 and 30 participants. Therefore, the current experiment may have lacked the power to detect any changes between REM and No-REM groups that may be present. Also, time spent asleep in the current study differed greatly from past research in which participants slept at least 3 hours between learning and testing phases, as participants were allowed only 90 minutes of sleep in the current study. Another

possibility is that REM-mediated influences on priming are only apparent with longer periods of time spent in REM. A final reason that REM sleep was not implicated in enhanced priming in the current experiment but was in past experiments may be that there could be a functional difference between sleep-dependent memory consolidation during a daytime nap compared with during nocturnal sleep. A previous study by Payne et al., (2012) found evidence that across nocturnal sleep periods, REM sleep activity was associated with enhanced memory for negative objects. However, a later work by Payne et al., (2015) showed that this same enhancement for negative memories was actually related to SWS activity during an afternoon nap. Further exploring the effects of an afternoon nap on emotionally salient information, a study by Alger et al., (2018) found similar results to the Payne et al., (2015) study in which SWS activity was found to be more related to enhanced recall of negatively salient stimuli, and further adding evidence that this effect was correlated to the total number and density of sleep spindle activity in SWS. While these studies were investigating more explicit forms of memory, they may indicate that aspects of sleep that are critical for enhancing priming effects may differ across daytime naps and nocturnal sleep. Further analyses of SWS and spindle activity in the current data may implicate other aspects of sleep in enhanced priming and antipriming effects in the sleep group.

It was also hypothesized that sleep-related enhancements in priming and antipriming effects would be observed in both response time and accuracy measures. In the analysis of accuracy, priming effects were significantly larger than antipriming effects, replicating previous work from Marsolek et al., 2006. However, there was no group difference observed in the accuracy measure. The lack of a group difference in

priming and antipriming accuracy could be related to the high accuracy rates overall for this sample. In a previous study by Marsolek et al., (2006) the average proportion correct ranged between 0.50-0.80 while the current experiment had proportions ranging from 0.81-0.90. The near-ceiling effect found in the current experiment could have reduced the sensitivity of the current experiment to detect priming and antipriming effects in the accuracy measure.

It should be noted that in both the accuracy and response time measures, significant antipriming effects were not observed in either group. However, accuracy was numerically lower for antiprimed compared with baseline objects and response times were numerically slower for antiprimed than for baseline objects, showing that an antipriming trend was present. While it should be noted that antipriming effects are typically smaller in magnitude than priming effects, past work has shown significantly worse performance for antiprimed than for baseline stimuli (Marsolek et al., 2006, 2010; Zhang, Fairchild, & Li, 2017). One reason for the lack of significant antipriming in either group in the present study may be the two-hour time break between phases 3 and 4. In previous experiments, phase 4 normally took place immediately following phase 3 (Marsolek et al., 2006, 2010). Given that antipriming effects are already smaller in magnitude than priming effects, this delay may account for the even smaller difference between antiprimed and baseline conditions in the current study. A delay of any kind could introduce increased interference that was not present in previous designs. A previous experiment by McKone (1998) tested the effects of time delay between encoding and testing on priming effects. In this design they tested time delays that included no additional tasks (time-only delay) and time delays that also included

additional tasks (task and time delays). This researcher found that both time-only delays and task and time delays worked independently to reduce short-term implicit memory priming effects (McKone, 1998). Therefore, the time delay in the current experiment may have diminished the already fragile antipriming effects to a point that significance was not found where it was in previous designs (Marsolek et al., 2006, 2010; Zhang, Fairchild, & Li, 2017).

The current experiment adds to a small body of literature that addresses the question of how sleep influences repetition priming. It is the first experiment to show that an afternoon nap enhances both repetition priming and antipriming. Previous studies that investigated the relationship between priming effects and sleep using overnight sleep periods were replicated in the current design in which priming, as well as antipriming, were enhanced following an afternoon nap. Unlike overnight sleep experiments, REM sleep was not a significant mediator of priming (or antipriming) enhancements, suggesting that sleep-dependent memory consolidation processes may operate differently during daytime napping and nocturnal sleep. Additional analyses of sleep physiology (e.g. sleep spindles, slow-wave power) may uncover mechanisms responsible for enhanced priming and antipriming in the current experiment. This experiment also further contributes to the perspective that antipriming effects may be more fragile than priming effects as first characterized by the Marsolek studies (2006; 2010). Additional research examining how delays between the study and test phases may influence priming and antipriming are necessary to more fully understand the fragility of antipriming effects. In conclusion, this study contributes to a wider understanding of how visual representations undergo changes during sleep and adds new considerations for future studies in this area.

APPENDIX SECTION

Appendix A: Pittsburg Sleep Quality Index

Name_____ Date_____

Sleep Quality Assessment (PSQI)

What is PSQI, and what is it measuring?

The Pittsburgh Sleep Quality Index (PSQI) is an effective instrument used to measure the quality and patterns of sleep in adults. It differentiates "poor" from "good" sleep quality by measuring seven areas (components): subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction over the last month.

INSTRUCTIONS:

The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

During the past month,

1. When have you usually gone to bed? _____
2. How long (in minutes) has it taken you to fall asleep each night? _____
3. What time have you usually gotten up in the morning? _____
4. A. How many hours of actual sleep did you get at night? _____
   B. How many hours were you in bed? _____

5. During the past month, how often have you had trouble sleeping because you	Not during the past month (0)	Less than once a week (1)	Once or twice a week (2)	Three or more times a week (3)
A. Cannot get to sleep within 30 minutes				
B. Wake up in the middle of the night or early morning				
C. Have to get up to use the bathroom				
D. Cannot breathe comfortably				
E. Cough or snore loudly				
F. Feel too cold				
G. Feel too hot				
H. Have bad dreams				
I. Have pain				
J. Other reason (s), please describe, including how often you have had trouble sleeping because of this reason (s):				
6. During the past month, how often have you taken medicine (prescribed or "over the counter") to help you sleep?				
7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				
8. During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?				
9. During the past month, how would you rate your sleep quality overall?	Very good (0)	Fairly good (1)	Fairly bad (2)	Very bad (3)

Scoring

Component 1	#9 Score	C1 _____
Component 2	#2 Score (<15min (0), 16-30min (1), 31-60 min (2), >60min (3)) + #5a Score (if sum is equal 0=0; 1-2=1; 3-4=2; 5-6=3)	C2 _____
Component 3	#4 Score (>7(0), 6-7 (1), 5-6 (2), <5 (3)	C3 _____
Component 4	(total # of hours asleep) / (total # of hours in bed) x 100 >85%=0, 75%-84%=!, 65%-74%=2, <65%=3	C4 _____
Component 5	# sum of scores 5b to 5j (0=0; 1-9=1; 10-18=2; 19-27=3)	C5 _____
Component 6	#6 Score	C6 _____
Component 7	#7 Score + #8 score (0=0; 1-2=1; 3-4=2; 5-6=3)	C7 _____

Add the seven component scores together _____ *Global PSQI* _____

A total score of "5" or greater is indicative of poor sleep quality.
If you scored "5" or more it is suggested that you discuss your sleep habits with a healthcare provider

Appendix B: Karolinska Sleep Log

KAROLINSKA SLEEP LOG **Date:**_____

1. At what time did you go to bed and turn the light off last night? _____PM or AM
2. At what time did you arise this morning? _____PM or AM
3. How long did you sleep? _____hours and _____minutes
4. How long did it take you to fall asleep? _____hours and _____minutes
5. How many awakenings did you have last night? _____
6. How many total minutes were you awake after falling asleep last night? _____minutes

(Don't include time in bed before falling asleep)

Circle one per question only:

7. How did you sleep?

1	2	3	4	5
Very Poorly				Very Well

8. Did you feel refreshed after you arose this morning?

1	2	3	4	5
Not at all				Completely

9. Did you sleep soundly?

1	2	3	4	5
Very Restless				Very Soundly

10. Did you sleep throughout the time allotted for sleep?

1	2	3	4	5
Woke up much too early				Slept thru the night

11. How easy was it for you to wake up?

1	2	3	4	5
Very Easy				Very Difficult

12. How easy was it for you to fall asleep?

1	2	3	4	5
Very Easy				Very Difficult

13. How much did you dream last night?

1	2	3	4	5
None				Much

Appendix C: Stanford Sleepiness Scale

Participant #_____ Date_____

Stanford Sleepiness Scale

Circle the number that best describes how you are feeling right now.

1 Feeling active, vital, alert, or wide awake
2 Functioning at high levels, but not at peak; able to concentrate
3 Awake, but relaxed; responsive but not fully alert
4 Somewhat foggy, let down
5 Foggy, losing interest in remaining awake; slowed down
6 Sleepy, woozy, fighting sleep; prefer to lie down
7 No longer fighting sleep, sleep onset soon; having dream-like thoughts

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