The Role of Sleep in the Selective Reconsolidation of Declarative Memories

by

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Abstract

While sleep has been shown to be involved in memory consolidation and the selective enhancement of newly acquired memories of future relevance (Wilhelm, et al., 2011), limited research has investigated the role of sleep or future relevance in processes of memory reconsolidation. The current research employed a list-method directed forgetting procedure in which participants learned two lists of syllable pairs on Night 1 and received directed forgetting instructions on Night 2. On Night 2, one group (Labile; n = 15) received a memory reactivation treatment consisting of reminders designed to return memories of the learned lists to a labile state. A second group (Stable, n = 16) received similar reminders designed to leave memories of the learned lists in their stable state. No differences in forgetting were found across the two lists or groups. However, a negative correlation between frontal delta (1 – 4 Hz) electroencephalographic (EEG) power during Early Stage 2 non-rapid eye movement (NREM) sleep and forgetting of to-be-remembered material was found exclusively in the Labile group (r = -.61, p < .05). Further, central theta (4 – 8 Hz) EEG power during rapid eye movement (REM) sleep was found to correlate with directed forgetting exclusively in the Labile group (r = .81, p < .001) and total forgetting in the Stable group (r = .50, p < .05). These observed relationships support the proposed hypothesis suggesting that sleep processes are involved in the reconsolidation of labile memories, and that this reconsolidation may be selective for memories of future relevance. A role for sleep in the beneficial reprocessing of memories through the selective reconsolidation of labile memories in NREM sleep and the weakening of memories in REM sleep is discussed.

Acknowledgements

Thank you, everyone.

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Introduction

This introduction to the current research provides a topical background of sleep and memory processes including consolidation, reactivation, and reconsolidaton. This is followed by a brief review of the neurophysiological characteristics of sleep that have been linked to sleep-dependent memory consolidation and that are pertinent to the current study. A prominent model of the function of sleep, the synaptic homeostasis hypothesis (Tononi & Cirelli, 2006), is considered along with recent research questioning some of the claims made in this hypothesis. A review on selectivity in memory processing is then provided with focus on the methodology of the research in forgetting and studies investigating the selectivity in sleep-dependent memory processing. A new hypothesis addressing the reprocessing of reactivated memories over sleep is then proposed before the current research is described in brief. Finally, this introduction ends with the specific predictions made regarding the outcomes of this study.

Sleep and Memory Background

Although it may appear to be simply a state of inactivity or rest, research has shown that sleep is much more complex in both its structure and function. Sleep has been traditionally separated into distinct stages defined by their physiological properties. Rapid eye movement (REM) sleep has been differentiated from four stages of non-REM (NREM) sleep in that electroencephalogram EEG recordings of REM sleep closely resemble those of a waking brain with the addition of theta frequency (4 – 8 Hz) brain activity, muscle atonia, and periods of saccadic eye movements (Berger, Olly, & Oswald, 1962; Cantero et al., 2003; Carskadon & Dement, 2011). Stage 1 NREM has been identified as a transitional stage between sleep and wakefulness, whereas stages 2, 3, and

4 of NREM sleep have been noted as being progressively deeper and containing increasingly more delta frequency (1 – 4 Hz) brain activity (Carskadon & Dement, 2011). This slow frequency EEG activity has been found to dominate stages 3 and 4, giving them the collective name of slow-wave sleep (SWS). Although they also occur during SWS, stage 2 sleep has been differentiated from other stages by its generally frequent, repetitive, and prominent bursts of sigma frequency (12 – 16 Hz) brain activity known as sleep spindles.

Over the course of a typical night, a sleeper will repeatedly go through a 90 – 120 minute cycle from relatively light sleep (stages 1 and 2) to deeper SWS, back to lighter sleep stages, and then to REM sleep before repeating this cycle anew (Carskadon & Dement, 2011). Typically, the majority of the night consists of stage 2 sleep. In addition, the first half of a typical night of sleep has been shown be relatively rich in non-REM sleep, whereas the second half has been conversely shown to be rich in REM sleep.

Despite spending approximately one third of our lives navigating through these states, there is a surprising lack of consensus on the function or, more likely, the functions of sleep. However, there has been evidence supporting the idea that sleep has a role in memory function since as early as 1924 with a study from Jenkins and Dallenbach suggesting that the rate of forgetting over sleep is slower than that over wakefulness. Since then, sleep has been linked to improvements in both non-declarative memories, which include memories that are not consciously recalled, and hippocampally-dependent declarative memories, which include memories for events and factual knowledge (Squire, 1992). Declarative memory improvements after sleep have been found for a variety of materials including word pairs (Plihal & Born, 1997), faces (Wagner, Hallschmid,

Verleger, & Born, 2002), short stories (Tilley & Empson, 1978), personal events (Aly & Moscovitch, 2010), and object-locations (Rasch, Büchel, Gais, & Born, 2007). Sleep, compared to wakefulness, has also been found to benefit memory by extracting the gist of encoded information (Payne et al., 2009), promoting insight into complex tasks (Wagner, Gais, Haider, Verleger, & Born, 2004), and increasing resistance to future interference (Ellenbogen, Hulbert, Stickgold, Dinges, & Thompson-Schill, 2006).

Thus, after decades of mostly confirmatory research in the area, there is widespread agreement that one function of sleep is to aid in memory consolidation, a term that collectively refers to the processes involved in stabilizing, enhancing and transforming memories (Diekelmann & Born, 2010). Two types of memory consolidation have been differentiated from each other. Synaptic consolidation refers to a shorter process that stabilizes and strengthens the synaptic changes associated with the memory, whereas system consolidation refers to a process that reorganizes memory representations and redistributes them from a localized and labile state to more stable neural networks of the brain for long-term storage (Born & Wilhelm, 2011; Dudai, 2004).

However, research in memory reactivation and reconsolidation has made it clear that these long-term memories are not permanent and may easily come out of storage (Sara, 2000). The reactivation of memories is thought to result from prediction errors in which strongly associated contextual cues are encountered without the items or events with which they are associated (Forcato, Rodríguez, Pedreira, & Robertson, 2011; Pedreira, Pérez-Cuesta, & Maldonado, 2004). The process of reactivation is thought to leave a memory trace in a labile state until the process of reconsolidation can occur and return it to its state of long-term storage. However, if reconsolidation does not occur, the

memory is not returned to its stable state of storage and it is lost over time. This sequence of events was first demonstrated by Misanin, Miller, and Lewis (1968) who reported that avoidance learning was lost in rodents that received electroconvulsive shock after contextual cues of the learning paradigm were given. Similar effects have been found using other types of memory and other amnesic treatments including the protein inhibitor anisomycin (Lewis, Bregman, & Mahan, 1972; Nader, Schafe, LeDoux, 2000). Evidence that reactivated memories are destabilized and in need of reconsolidation has been found using human subjects as well (Schiller et al., 2010; Walker, Brakefield, Hobson, & Stickgold, 2003). Further, both human and animal studies suggest this process of reactivation and subsequent reconsolidation may serve as a mechanism by which memories are updated with new and relevant information (Forcato, Rodríguez, Pedreira, & Maldonado, 2010; Lee, 2008).

Few studies to date have directly investigated a role for sleep in memory reconsolidation. In one study, it was found that six hours of REM sleep deprivation immediately after memory reactivation had no effect on the reconsolidation of either cued or contextual fear memories in rats (Tian et al., 2009). However, using six hours of total sleep deprivation instead of only REM sleep deprivation, a different study did find evidence for a role of sleep in memory reconsolidation. By injecting groups of rats with morphine in a distinct chamber, using that chamber to reactivate reward memories in some rats, and then later measuring preference for that chamber, Shi et al. (2011) were able to examine effects of memory reactivation and sleep deprivation on the strength of morphine reward memory. It was found that the groups receiving the combination of memory reactivation treatment and immediate sleep deprivation showed reduced

preference at test for the cage associated with morphine injection when compared to groups not receiving memory reactivation and groups that slept following reactivation treatment. Thus, it was concluded that sleep deprivation following memory reactivation disrupted memory reconsolidation processes, leading to degradation of the reactivated memory trace.

Neurophysiology of Sleep-Dependent Memory Consolidation

While there is a paucity of research on the role of sleep in reconsolidation, there has been a great deal of work conducted on sleep-dependent memory consolidation. Through this research, various aspects of sleep, including slow-wave (0.5 – 4 Hz) activity and sleep spindles in NREM sleep and theta activity in REM sleep, have been implicated in memory processing. Although the once popular dual-process hypothesis proposed that SWS was responsible for consolidation of declarative memories and REM sleep was responsible for consolidation of non-declarative memories (Diekelmann, Wilhelm, & Born, 2009; Plihal & Born, 1997, 1999), more recent evidence suggests that the different characteristics and stages of sleep may actually work together in the processing of declarative memories (Ackermann & Rasch, 2014; Diekelmann & Born, 2010).

SWS characterized by delta waves and slow oscillations (< 1 Hz) is widely thought to play a role in system consolidation processes through the reactivation of new memory traces (Diekelmann & Born, 2010; McClelland, McNaughton, & O'Reilly, 1995). In addition to a number of studies showing declarative memory benefits from early night sleep rich in slow-wave activity (Diekelmann et al., 2009; Plihal & Born, 1997; Yaroush, Sullivan, & Ekstrand, 1971), recordings of hippocampal and neocortical cells in rats have shown that spatiotemporal firing patterns during SWS closely resemble

the patterns recorded during prior learning of a maze (Ji & Wilson, 2007; Wilson & McNaughton, 1994). Both cortical gamma oscillations as well as slow-wave EEG activity may be involved in these reactivation patterns and the system consolidation of memories as both have been related to hippocampal sharp-wave ripple complexes that act as a method of communication between the neocortex and hippocampal cells (Chrobak & Buzsáki, 1996; Girardeau, Benchenane, Wiener, Buzsáki, & Zugaru, 2009; Le Van Quyen et al., 2010). Further supporting this view, using functional magnetic resonance imaging, Gais et al. (2007) found that sleep, compared to wakefulness, after learning was found to result in greater hippocampal-neocortical functional connectivity during a recall test 48 hours after learning. Memory reactivation during SWS has also been shown to have a casual and beneficial role in memory consolidation in an experiment measuring memory performance after presenting some participants with a scent during sleep to cue the memories encoded with that scent during the waking state (Rasch et al., 2007).

Sleep spindles have similarly been linked to sleep-dependent memory consolidation in a number of ways. Sleep spindles have been found to be positively correlated with hippocampal ripples and slow-wave activity, suggesting that they also play a role in hippocampal-neocortical communication during NREM sleep (Siapas & Wilson, 1998; Steriade, 2006). In addition, increases in sleep spindle density following learning have been found, and these increases are associated with better recall the following day (Gais, Mölle, Helms, & Born, 2002; Schabus et al., 2004). At the cellular level, simulated spindle firing patterns have been shown to alter synaptic plasticity and trigger long-term potentiation (Rosanova & Ulrich, 2005). While both slow sleep spindles in the 12 – 14 Hz range and fast sleep spindles in the 14 – 16 Hz range have

been studied, fast sleep spindles are thought to play the more direct role in declarative memory consolidation due to the temporal relationship between them and the slow oscillations of NREM sleep (Mölle, Bergmann, Marshall, & Born, 2011). Spindle activity is also predictive of performance on IQ tests and other measures of intelligence (Fogel, Nader, Cote, & Smith, 2007; Nader & Smith, 2001; Schabus et al., 2006). These results suggest that although spindle activity likely plays a role in memory processing, sleep spindles appear to have a more broad reach as marker of intelligence and learning ability (Fogel & Smith, 2011).

It has been proposed that while NREM sleep drives active system consolidation, REM sleep plays a complementary role by driving synaptic consolidation (Diekelmann & Born, 2010); however, the exact role of REM sleep in memory processing remains a mystery. Early research primarily using REM sleep deprivation methods strongly supported a role for REM sleep in memory processing (Smith, 1995). The additional stress placed on subjects in early REM deprivation studies have led to criticisms of this early research, but there is still ample evidence supporting a role for REM sleep in memory processing (Born & Gais, 2000). For example, greater amounts of REM sleep has been linked the enhancement of emotional memories, improved implicit face memory, and greater performance in a word-stem priming task (Payne, Chambers, & Kensinger, 2012; Plihal & Born, 1999; Wagner, Gais, & Born, 2001; Wagner et al., 2002). Supporting a role for REM sleep in synaptic consolidation, REM sleep has been found to be related to an upregulation of plasticity-related gene expression that supports long-term potentiation in areas of the brain active during prior learning (Diekelmann & Born, 2010; Ribeiro, Goyal, Mello, & Pavlides, 1999; Ribeiro et al., 2002). Theta activity during

REM sleep in particular has been implicated in memory processes as significant increases in REM sleep theta power in central regions were found following paired-associate learning (Fogel, Smith, & Cote, 2007). Furthermore, it has been suggested that theta waves of REM sleep may play a role in inducing long-term potentiation and that the high level of cholinergic activity globally during REM sleep may aid in the maintenance of long-term potentiation, thus enhancing synaptic consolidation (Diekelmann & Born, 2010; Pavlides, Greenstein, Grudman, & Winson, 1988).

Synaptic Homeostasis Hypothesis

An additional hypothesis regarding of sleep-dependent improvements in memory is the synaptic homeostasis hypothesis which proposes that the function of sleep is to regulate the synapses in the brain (Tononi & Cirelli, 2006). This hypothesis claims that wakefulness, and the accompanied learning, results in a great amount synaptic potentiation (Klintsova & Greenough, 1999), which is later relieved through a synaptic downscaling process tied to slow-wave activity during sleep. This hypothesis accounts for the well-documented homeostatic regulation of slow-wave activity (Borbély, 1982). Slow-wave activity has been shown to be regulated, at least partially, as a function of prior wakefulness and the learning (i.e., long-term potentiation) that took place during the period of wakefulness (Huber, Ghilardi, Massimini, & Tononi, 2004; Huber et al., 2008). This synaptic downscaling during SWS then, according to this hypothesis, results in benefits such as energy conservation as well as improved learning and memory (Tononi & Cirelli, 2006).

The benefits of sleep on later learning are hypothesized to come from a freeing of saturated neural connections in areas such as the hippocampus, allowing these areas to be

reused during subsequent learning (Tononi & Cirelli, 2006). This claim is supported by the finding that learned material is more resistant to interference from previously learned material when an intervening period of sleep is present (Sheth, Varghese, & Truong, 2012). This may be a result of the representation of the potentially interfering memories being, to some extent, removed during sleep from areas involved in encoding and tied to neocortical areas through system consolidation. Also consistent with this hypothesis,Van Der Werf et al. (2009) found that restricting slow-wave activity with a mild acoustic sleep disruption method resulted in a decreased ability to learn a visual memory encoding task the following day. Functional magnetic resonance imaging revealed that the induced shallow sleep was associated with less activity in the right anterior hippocampal formation during encoding, suggesting that the ability to encode new information in this region may have been compromised.

Improved memory and performance is hypothesized to result from synaptic downscaling during SWS through a process of increasing the signal-to-noise ratio (Tononi & Cirelli, 2006). An equal and global downscaling across synapses is proposed to reduce weak connections (noise) to insignificant and ineffective levels while also reducing stronger synapses (signal); however, due to their greater relative strength, these synapses remain a part of future memory processes. This function of slow-wave activity is supported by many studies associating SWS with beneficial effects on memory, including: Huber et al. (2004), who found slow-wave activity to be related to later performance on a memory test; Landsness et al. (2009), who found that visuomotor performance after learning was impaired after interrupting slow waves with audio stimulation; and Olcese, Esser, and Tononi (2010), which examined downscaling

specifically using a computational model.

Although the synaptic homeostasis hypothesis has gained favour as theoretical ground for sleep and memory processes, some criticisms and contradicting findings have been raised. Frank (2012) also explicitly criticized the synaptic homeostasis hypothesis for its lack of clarity on the physiological mechanisms that may underlie synaptic downscaling during sleep. Frank (2012) has suggested that oscillations in delta frequency range characteristic of SWS may actually serve to promote long-term potentiation rather than synaptic downscaling (Grover, Kim, Cooke, & Holmes, 2009). Furthermore, two studies examining retroactive interference, which is the case of newly acquired information interfering with associated memories previously formed, found evidence directly in contrast to the prediction that weak memories are decreased in strength due to synaptic downscaling. Replicating and building upon a study by Ekstrand (1967), Drosopoulos, Schulze, Fischer, and Born (2007) had participants learn a set of cued word pairings to a criterion of 90% before learning another list of words associated to the same cues (an A – B, A – C paradigm) all before a period of either sleep or wakefulness. A later cued-recall test found that memories weakened through retroactive interference were enhanced over sleep while stronger, more freshly encoded memories were unaffected. Similarly, in a supporting study, memories encoded less deeply were also given preferential enhancement over sleep. The fate of weak memories in these studies appear to contradict the predictions of the synaptic homeostasis hypothesis.

Additional reports also call into question whether SWS is the best candidate mechanism for synaptic downscaling during sleep. Grosmark, Mizuseki, Pastalkova, Diba, and Buzsáki (2012) found that firing rates of hippocampal neurons increased over

periods of NREM sleep, and then experienced a more rapid decrease over REM periods. REM sleep theta power specifically was found to correlate with a decrease in firing rates and an increase in synchrony. During wakefulness, stimulation of hippocampal afferents during the peaks of theta oscillations reliably induce long-term potentiation, while stimulation during the troughs of theta oscillations can result in long-term depotentiation of synapses (Huerta & Lisman, 1995). Poe, Nitz, McNaughton, and Barnes (2000) recorded cellular activity in rats' REM sleep after each ran through both a previously learned track and a novel track. It was found that hippocampal place cells exclusively associated with the familiar track fired during the troughs of REM sleep theta oscillations and cells associated with the novel track fired during the peaks of theta oscillations, Considering both Huerta and Lisman's (1995) findings and the differentiation in firing patterns during REM sleep, Poe et al. (2000) suggested that REM sleep could serve synaptic plasticity by strengthening memories of recent experience and weakening more remote memories. So, while the logic behind the necessity of synaptic downscaling may be sound, these results suggest that REM sleep rather than SWS may perform the task, and further, REM sleep may add selectivity to the downscaling process.

Selective Memory Processing

The degradation or weakening of memories is closely linked to another aspect of memory: forgetting. Although commonly considered a fault of memory, it may be more accurate to consider some types of forgetting as playing an essential role in memory efficiency (Storm, 2011). The previously described interference task can be used as an example. If one's goal was to remember the second A – C list of words, strong memory of the initial A – B pairs may cause interference and influence the accuracy of memory.

Throughout daily life as well, some cues will inevitably be paired with multiple memories, and the ability to forget weak memory traces as well as those that are no longer relevant is crucial. Without this ability, the sight of a large parking lot might trigger multiple memories of previously used parking spaces, making remembering the current placement of a car much more difficult.

Research investigating forgetting has typically done so using one of two paradigms. A phenomenon known as retrieval-induced forgetting is typically studied using the retrieval-practice paradigm (Anderson, Bjork, & Bjork, 1994). In this method, participants are required to learn lists of words sorted by their categories (e.g., fruits or sports). They are then asked, through cuing, to retrieve some items from one of these lists from memory. When tested for recall later, these items are better recalled than items from the non-practiced list; however, memory for the unpracticed items from the practiced list is the worst of the three groups. This phenomenon has been shown for a variety of stimuli and is thought to occur because of the inhibition applied to the unpracticed items to prevent their retrieval when the cue calls for other items from the same list (Levy & Anderson, 2002). This effect provides evidence that inhibition processes occur during wakefulness. This may be an adaptive measure to prevent overwhelming levels of interference.

A second method through which forgetting has been studied is the directed forgetting paradigm in which participants typically learn lists of items that are paired with cues to either remember or forget the particular items. This usually results in the to-be-forgotten (TBF) items being remembered more poorly than to-be-remembered (TBR) items, a pattern known as the directed forgetting effect (for review, see MacLeod, 1998).

Directed forgetting is studied using both the item-method in which the cue indicating that this item is to be remembered is given after each individual to-be-remembered item, and the list-method in which a cue is typically placed halfway through a list asking the participant to forget previous items and remember subsequent ones. When tested for recall, the effects of the two types of instruction are similar; however, when tested for recognition, the list-method does not typically result in a direct forgetting effect (Basden, Basden, & Gargano, 1993; MacLeod, 1999). The directed forgetting effect found using the item-method has been attributed to a lower degree of rehearsal being given to TBF items compared to TBR items, whereas the directed forgetting effect found using the list-method has been attributed to an inhibition of TBF items after learning that may limit the accessibility of these memories without necessarily weakening their strength (Bjork & Bjork, 2003). Furthermore, the directed forgetting effect is often found to be limited or completely absent for emotionally charged material when included in both item-method (Bailey & Chapman, 2012; Otani et al. 2012; Nowicka, Marchewka, Jednoróg, Tacikowski, & Brechmann, 2011) and list-method paradigms (Payne & Corrigan, 2007). This is understandable given that memory of emotional material is typically stronger than neutral material (LaBar & Cabeza, 2006).

Crick and Mitchison (1983) postulated that the role of REM sleep may be to weaken select memory traces through a reverse learning mechanism. In contrast, Jenkins and Dallenbach (1924) suggested that sleep prevents forgetting through its passive prevention of interference. Despite these classic theories of sleep and memory and the emerging focus on memory reconsolidation and synaptic downscaling during sleep, only a few studies have specifically examined the role of sleep in forgetting. Instead of either

just strengthening or just weakening memories, it might be more appropriate to consider that sleep may have a more general role in optimizing memory efficiency. In this sense, sleep may serve to downscale or forget memories that are no longer important while preserving and enhancing other memories, thus improving memory accuracy and efficiency when interference is a possibility.

Studies examining the role of sleep in retrieval-induced forgetting have yielded mixed results. Racsmány, Conway, and Demeter (2010) reported increases in retrieval-induced forgetting over a night of sleep compared to wakefulness. However, this effect was not found in two studies employing similar methods. Abel and Bäuml (2012) found that sleep, compared to wakefulness, only enhanced memories of unpracticed control items and did not result in increased forgetting of items inhibited through retrieval-induced forgetting. However, it is interesting to note that only the memories of control items, and not those of the inhibited items were enhanced by sleep. When Baran, Wilson and Spencer (2010) investigated the influences of particular sleep stages in retrieval-induced forgetting, they found no evidence of enhanced forgetting over either a night of sleep or a 90-minute nap opportunity. In fact, it was found that the items weakened from the retrieval-induced forgetting procedures were actually better remembered over sleep and that this enhancement was positively correlated with time spent in REM sleep.

These tests of forgetting, however, were investigating a type of selectively that was not necessarily beneficial to memory efficiency and accuracy. Instead, a role for motivation to remember certain material in sleep-dependent memory consolidation has been shown using both between-subjects designs (Wilhelm et al., 2011) and within-subjects designs (van Dongen, Thielen, Takashima, Barth, & Fernández, 2012). First, the

different experiments conducted by Wilhelm et al. (2011) had participants learn word pair associates, an object-location task, and a finger tapping sequence. For each task, half of the participants were informed of the next-day memory test after learning the material. The other half of the participants were instead told they would be asked to perform a completely different task. The participants informed of the future memory tests showed both an increase in the proportion of stage 4 SWS and the number of sleep spindles in SWS compared to the unsuspecting participants. For all three tasks, it was found that only when memories were expected to be of future relevance did post learning sleep result in enhancement of the relevant memory. Furthermore, the declarative memory improvements on the object-location task seen in the informed participants were positively correlated with increased slow oscillations and spindle activity of SWS. These results were conceptually replicated by van Dongen et al. (2012) who showed using a picture-location task that only sleep and not wakefulness led to a selective memory improvement for material told to be on a future test compared to material participants believed would not to be tested again.

Motivation to specifically suppress and inhibit memories over sleep using a think/no-think paradigm, however, was not shown to be effective (Fischer, Diekelmann, & Born, 2011). Participants in this study were asked to actively suppress some target words from their minds when presented with their cue words while rehearsing the target words presented with other cue words. Although this study found a general memory improvement for rehearsed words, sleep did not facilitate the forgetting of suppression pairs when tested the next day. Similar to Baran et al. (2010), a supplementary experiment suggested that late-night sleep rich in REM sleep compared to early-night

SWS actually led to better memory for words meant to be suppressed when tested for 30 minutes after wakening. Therefore, effort to actively inhibit memories from sleep-dependent consolidation may paradoxically result in a stronger memory trace the following day.

Studies examining directed forgetting over sleep have attempted to address the possible role of sleep in beneficial forgetting by examining how the sleeping brain treats TBR and TBF items. Rauchs et al. (2011) had participants follow a typical item-method directed forgetting paradigm and then subjected half of the participants to a night of sleep deprivation. When tested for recognition after two recovery nights, participants deprived of sleep for the first post-learning night remembered more of the TBF words than participants that slept for three consecutive nights. This effect was observed despite equal performance between groups in recognition of TBR words. Imaging data showed that during encoding, there was more hippocampal activity for TBR items than TBF items, and even further, more hippocampal activity was seen in TBR items that were successfully remembered compared to those that were not. Additionally, a similar item-method directed forgetting study found that a 100-minute nap opportunity led to a selective benefit in remembering TBR words over TBF words and that this directed forgetting effect was positively correlated with a count of fast sleep spindles over the nap opportunity (Saletin, Goldstein, & Walker, 2011). Although the nap did not appear to result in any active forgetting effects at the level of behavioural performance, this result serves to echo the selectivity in the memory consolidation process previously reported by Wilhelm et al. (2011).

The list-method directed forgetting procedure used by Abel and Bäuml (2013)

offers a different approach to the question of the role of sleep in directed forgetting. In this study, all participants learned a list of sixteen words, but while one group was told after learning that they should remember this list, another group was told they were presented the wrong list and should forget what they had just seen. Participants were further divided into groups receiving either a twelve-hour delay containing sleep or a twelve-hour delay containing only wakefulness before the test. A third twenty-minute delay group was also included to control for time-of-day effects on recall. It was found that when sleep was included in the twelve-hour delay, it served to eliminate the directed forgetting effect found in the other groups. Thus, these findings support the claim that sleep may actually serve to enhance weaker memory traces and rescue them from inhibition (Baran et al., 2010; Fischer et al., 2011).

However, it is important to consider when discussing these directed forgetting methods that the memories being forgotten may not be comparable to stable memories that have been previously consolidated over sleep. If one considers forgetting to be the loss of a memory once had, the item-method directed forgetting paradigm suffers to some extent from a lack of external validity. Being given the cue to forget material so quickly after presentation likely halts any deep encoding of that material, an idea reflected by the lack of hippocampal activity during encoding reported by Rauchs et al. (2011). In these cases, the TBF items are not strong memories that need to be forgotten, but instead are just a source of noise that should be ignored. The list-method procedure used by Abel and Bäuml (2013) addresses forgetting more closely as participants learn the TBF material as if it was going to be on the test before knowing that it should be forgotten. However, there is also a need to investigate the forgetting of memories that have previously been

consolidated over sleep as it is likely fundamentally different from the forgetting of material learned only moments earlier.

Current Research

Based upon the research reviewed here, it is hypothesized that sleep may function to beneficially reprocess labile memories both through reconsolidation processes selective for future relevance and through downscaling mechanisms that hasten the degradation of memories without future relevance. Selectivity in memory reconsolidation may naturally serve to replace memories that are no longer relevant. If, while learning new material, related but no longer relevant memories are reactivated, these older memories would return to a labile state that requires reconsolidation to avoid degradation (Sara, 2000). Then, if reconsolidation processes are selective for future relevance as sleep-dependent memory consolidation has been shown to be (Saletin et al., 2011; van Dongen et al., 2012; Wilhelm et al., 2011), the memories of no future relevance would not receive reconsolidation and instead be degraded. A similar process in which only some components of a memory trace reconsolidated while other reactivated components are replaced with new information and left to degrade could explain how memories may be updated during sleep. Regulation of memory resources and energy resources could hypothetically be achieved through synaptic downscaling mechanisms proposed to be global in nature and taking place during SWS (Tononi and Cirelli, 2006). However, it may be more likely that this process of synaptic plasticity would be more selective in nature and take place during REM sleep (Grosmark et al., 2012; Poe et al., 2000). Thus, sleep is hypothesized to play a role in maintaining an efficient use of resources through its reprocessing of reactivated memories. Labile memories of future relevance may be

retained or strengthened through selective memory reconsolidation during NREM sleep, while other labile memory traces are weakened through degradation and active downscaling mechanisms taking place in subsequent periods of REM sleep.

The current research examined this proposed role of sleep in the reprocessing of previously consolidated memories by employing a modified list-method directed forgetting paradigm. All participants were asked to learn two lists of five syllable pairs for a future memory test. Although previous research on directed forgetting over sleep administered the directed forgetting instructions either during or shortly after learning the material, the current research allowed participants a full night of sleep to consolidate their memories of the lists before they were given the directed forgetting instructions. Each syllable pair list was learned in combination with a specific context that was used to remind participants of these lists the following evening. To investigate the proposed forgetting process of selective memory reconsolidation, participants were randomly assigned to two groups. One group received a specific type of reminder shown to reactivate memories (the Labile group) while the other group received reminders shown to keep memories in their stable state (the Stable group; Forcato et al., 2011). On this same day, both groups received directed forgetting instructions informing participants which of the lists would be on the test and which would not be on the test. All participants were then allowed a second night of sleep before being tested for cued-recall of the syllable pair lists the following morning.

The directed forgetting instructions were not expected to have an effect on the Stable group as their memories were not expected to receive reprocessing during the second night of sleep. This is in contrast to the Labile group whose memories were

reactivated and in need of reconsolidation. It was hypothesized that the overnight reconsolidation processes expected to take place in this Labile group would act to selectively reconsolidate the memories of relevant TBR material while memories of irrelevant TBF material would be forgotten more quickly through downscaling processes of sleep.

Within the Labile group, various features of sleep physiology were predicted to play a role in the second-night reprocessing of these memories. Considering the documented role of SWS in sleep-dependent memory consolidation (Diekelmann & Born, 2010), it was predicted that delta power during NREM sleep would positively correlate with memory of TBR items. Secondly, theta power during REM sleep was predicted to positively correlate with forgetting of TBF items as it has been proposed to play a role in both synaptic depotentiation and the downscaling of neuronal firing rates (Poe et al., 2000; Grosmark et al. 2012). Finally, based on Saletin et al. (2011), it was expected that the directed forgetting effect would be positively correlated with stage 2 fast sigma power in the Labile group. Again, these relationships were not expected in the Stable group that did not receive the memory reactivation treatment as their memories were not expected to undergo substantial reprocessing during the night. A summary of these predictions is available in Table 1.

Method

Participants

Participants were recruited from the university population at Brock University through posters displayed on campus, the psychology department's participant recruitment website, and visits to psychology classrooms. All potential participants were asked to phone the Sleep Research Laboratory to learn more information about the study and to undergo a short screening interview (Appendix A). Those deemed eligible after the phone interview were given a more detailed, web-based screening questionnaire package that included a sleep/wake and health questionnaire, the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), the Epworth Sleepiness Scale (Johns, 1991), and a 30-item Fatigue Questionnaire (Yoshitake, 1978). Only individuals between 18 and 30 years of age, who learned English before age 8, and reported typical daily sleep from approximately 23:00/00:00 hr to 07:00/08:00 hr were eligible to participate in the study. Further, all of those scoring high on either the depression, sleepiness, or fatigue measures; those reporting evidence of sleep disorders or head injury; and those indicating that they currently smoke cigarettes, take medications, or have worked shift work in the previous six months were excluded from participation.

Of the 58 individuals who completed the web-based screening questionnaires, 6 lost interest or were unable to participate in the study, 5 were excluded due to high scores (>9) on the Beck Depression Inventory, 2 were excluded due to poor sleep patterns, and 1 was excluded due to a reported head injury. After an off-protocol adaptation and polysomnographic screening night in the laboratory, an additional 3 individuals lost interest in participating, another 3 were excluded due to frequent arousals associated with

periodic limb movements, and 1 was excluded due to a seizure in the laboratory.

A sample of 37 participants passed all screening procedures and completed the study. Of these, 3 participants were removed from the analyses due to poor sleep efficiency (< 80%) on the second experimental night. An additional 3 participants were removed because their learning performance in the memory task was below the 60% criterion; this criterion was based on previous research using this memory task (Forcato et al., 2011). Data collected from pre-sleep and post-sleep questionnaires given at each session of laboratory-recorded sleep revealed that 3 participants admitted to consuming caffeine during the experiment. In each case, the participant reported consuming only a single serving of coffee or tea at least five hours prior to coming to the lab. For this reason, these participants were not excluded. No other irregularities emerged from the pre-sleep and post-sleep questionnaires. Participants were given either an honorarium of 25 dollars or course credit for participation in the adaptation and screening night and were given an additional 50 dollars for completion of the full study.

The final sample used for data analysis included a total of 31 participants. Random assignment was used to place participants into either the Stable or Labile groups, defined by the memory reactivation treatment each would receive during the experiment. This was done separately for both men and women in an effort to maintain comparable sex distributions in the two groups. The Stable group included 16 participants (11 women) ranging from 18 to 25 years of age (M = 19.69, SD = 2.24), and the Labile group included 15 participants (8 women) ranging from 18 to 24 years of age (M = 20.80, SD = 1.97). Ten participants in each group reported in a feedback questionnaire (Appendix B) given after debriefing that they either suspected or knew the TBF material would be on the

memory test. Although concerning, this number may be inflated due to a possible hindsight bias present in participants when responding after being debriefed. These participants were included in the sample to preserve the total sample size. Groups did not differ significantly in either verbal or performance intelligence as measured by the Multidimensional Aptitude Battery-II (Jackson, 1998).

Materials

Memory task. The task used to assess memory performance was based on a paired-syllables memory task shown to be effective in previous research on memory reactivation and reconsolidation (Forcato et al., 2011). The task was delivered to each participant on a desktop computer with a monitor displaying all images in colour and desktop speakers playing all sounds at a comfortable preset volume. Participants were administered three lists of syllable pairs with each list being delivered in one of three blocks for them to learn. Participants were tested via cued-recall on all three blocks on the final day of the three-day experimental protocol (Figure 1). The three blocks of the task were identified to participants as Block P, Block A, and Block B. To fit a list-method directed forgetting paradigm, participants learned all the material before instructions identified Block A as TBF and Block B as TBR. Block P was identified as a practice block. With these instructions, memory could be tested for learned material cued TBR, learned material eventually cued TBF, and also learned material that was never intended to remembered.

The syllable list assigned to each block was randomly selected for each participant from the three lists of five syllable pairs (e.g., SAN – DEM; for complete lists, see Appendix C). All syllables used in these lists were created for this study to be natural

English syllables in the form of a three-letter stream (consonant – vowel – consonant) that were not words, popular acronyms, or expressions on their own. The three syllable pair lists did not differ significantly in cued-recall difficulty in a pilot of 21 participants (Appendix D). Each block was randomly assigned one of three context pairs used to build stronger associations with memories of the syllable pair lists so that they could later be reactivated through the introduction of prediction errors. Each of the two sets in a context pair had a unique border colour, background image, and piece of music. For example, one context set in a pair had a border colour of orange, a background image of a forest in autumn, and classical piano music, while its alternative set had a blue border, a city background image, and smooth jazz music (for all context sets and pairings, see Appendix E). Background images were acquired from various online sources of free-to-use desktop backgrounds, and music samples were acquired from the Freeplay Music LLC music libraries and edited using Audacity® audio editor to fit the needs of the task. The task was programmed by K. MacDonald using AutoHotkey scripting language and GUI utility (C++ source code).

The two context sets in each block were used by participants to differentiate two types of syllable trials, namely Syllable Trials and Foil Trials (Figure 2). Syllable Trials contained the list of syllable pairs. Foil Trials did not contain the syllable pairs and were implemented to ensure participants paid attention to all aspects of the trials. Both types of trials began with a context period that presented a colour border for two seconds, the colour border and a background image together for the next two seconds, followed by the colour border, background image, and music for six seconds. All Syllable Trials in each block were specifically paired with one of the context sets for that block (e.g., blue

border, city background, and smooth jazz music). The context for all Foil Trials was randomly selected in each trial and included at least one element from the alternative context set (e.g., orange border, forest background, or classical piano music). To ensure participants were attending to the context period of each trial, they were asked to enter a prediction during the last three seconds of the context period as to whether or not the trial would contain syllable pairs. They were asked to press the right arrow key (marked Y) to predict "yes, there will be syllable pairs" or the left arrow key (marked N) to predict "no, there will not be syllable pairs."

In Syllable Trials, the ten-second context period was followed by the five syllable pairs presented one-by-one on the monitor (Figure 2). First, one cue syllable was shown on the left side of the screen for five seconds alongside a blank response box on the right side of the screen. Participants were asked to fill in this blank by typing in the cue syllable's paired associate. After five seconds, the response was recorded and the correct answer was shown for four seconds before the process was repeated for the next syllable pair. Syllable Trials ended after all five syllables pairs were presented. If the response entered did not match the correct answer, an error was scored. The number of errors made in each Syllable Trial was recorded. Foil Trials ended immediately after the ten-second context period (i.e., no syllable pairs were presented). The trials of each block were presented in a pseudorandom order that did not allow for more than three consecutive Syllable Trials. Four seconds of a blank black screen separated each trial.

The memory task was divided into a learning session, a reminder session, and a test session with each session taking place on a separate day of the experiment (Figure 1). Participants were also given an interactive demonstration of the memory task prior to the

experimental protocol to ensure all participants understood the task before coming to the learning session.

Learning session (Day 1, 21:00 hr). To begin the learning session, participants were reminded of the task instructions and told that they were going to be given three blocks of the memory task. The first block (Block P) was said to be a practice block, but they were asked to try their best at it regardless. They were then told that they should learn the next two blocks (Block A and Block B) for the upcoming test session. However, they were told that only one of these blocks would actually be on the test, that they were not going to be told which block it was, and that they should learn both to the best of their ability (for the script of the instructions, see Appendix F). Each block in the learning session was composed of eight Syllable Trials and sixteen Foil Trials. To keep memories of the syllable list from each block separate and free from interference, a two-minute break and three-minute reaction time task was placed after each block. Participants were told the reaction time task was used to measure their alertness, although it was merely a filler task. No responses were collected for the first Syllable Trial of each block because participants had not yet seen the syllable pairs. Including instructions, the learning session was approximately 60 minutes in duration.

Reminder session (Day 2, 21:00 hr). Both the within-group directed forgetting manipulation and the between-group memory stability manipulation were administered during the reminder session. Trials given in the reminder session differed from those in the other sessions and differed between the Stable and Labile groups as well (Figure 3). The Stable group was given Cue-Response Reminders that were similar to regular Syllable Trials with two exceptions. First, the correct answer was not shown after the

participant's response was submitted, and second, only one syllable pair was presented before a notice appeared indicating the end of the reminder. The Labile group was given Cue-Only Reminders designed and shown to trigger memory reactivation and reconsolidation through the introduction of a strong prediction error (Forcato et al., 2011). Cue-Only Reminders differed from the Stable Group's Cue-Response Reminders in that the notice ending these reminders appeared either two seconds into the response window or as soon as the participant attempted to type in a response (whichever came first). This abrupt ending is thought to drive memory reactivation by introducing a prediction error and preventing participants from completing their response process. All participants were given two reminders of Block A followed by two reminders of Block B. To prevent participants from being shocked by the abruptness of the reminders, all participants were told at the start of the session that the reminders would be shorter than the trials in the learning session. A five-minute drawing task was given immediately after each reminder. Although it simply served to separate the reminders and prevent rumination over each trial, participants were told this task would be used as a measure of creativity.

The directed forgetting manipulation occurred before the first reminder of Block B. After the five-minute drawing task, all participants were shown a notice on the screen indicating that they were to receive special instructions (for the script of the instructions, see Appendix F). These instructions alerted participants that they were selected to be in the group that gets to know which block of syllable pairs would be on the test. In fact, all participants were told that they would be tested on Block B, that they would not be tested on Block A, and that there was no need to remember Block A. The reminder session then

continued with the Block B reminders. The reminder session was approximately 30 minutes in duration.

Test session (Day 3, 09:00 hr). The test session closely resembled a shorter version of the learning session. Participants were tested on all three blocks with each block containing four Syllable Trials and eight Foil Trials. Again, a two-minute break and a three-minute reaction time task were given between the blocks. In this session, participants were first tested on Block A, then Block B, and finally Block P. Participants were instructed to try their best to remember the material from each block regardless of previous instructions. The test session was approximately 25 minutes in duration.

Outcome variables. To account for the individual variability in learning performance, forgetting scores were calculated to measure memory performance at test session relative to performance in the learning session. Also, because the syllable lists were repeated across the multiple test trials, only the first Syllable Trial from each block was used to measure forgetting. Thus, a forgetting score was calculated for each block by subtracting the number of errors made in the block's last Syllable Trial of the learning session from the number of errors made in the block's first Syllable Trial of the test session. To reflect the directed forgetting instructions, forgetting of Block A was scored as TBF forgetting, and forgetting of Block B was scored as TBR forgetting. Because Block P was not cued as either TBR or TBF, forgetting of Block P can be considered a measure of the natural degradation of memory. A directed forgetting score was calculated by subtracting TBR forgetting scores from TBF forgetting scores. In this way, higher directed forgetting scores reflect a tendency to remember TBR items and forget TBF items. In addition, a total forgetting score was calculated as the sum of a participant's

TBR and TBF forgetting scores to reflect the amount of overall forgetting independent of the TBR and TBF cues.

As an alternative measure of memory, the second, third, and fourth Syllable Trials of each block during the test session were used to explore the difficulty of relearning the syllable pairs. The total number of errors made in these relearning trials was calculated for each block to yield a score for the relearning errors made in the practice block, the TBR block, and the TBF block. Similarly, a score for directed relearning errors, reflecting a greater difficulty in relearning TBF items than TBR items was calculated by subtracting the relearning errors made in the TBR block from the relearning errors made in the TBF block. Finally, a score of total relearning errors was calculated as the sum of a participant's TBR and TBF relearning errors to reflect the overall difficulty in relearning independent of the TBR and TBF cues.

Polysomnography

All sleep electrophysiology was recorded using SynAmps2 amplifiers with Neuroscan SCAN 4.5 software (Compumedics Inc., Abbotsford, Australia). Gold-plated silver electrodes were fixed to the skin each night using both surgical tape, cotton gauze and Ten20 Conductive Paste (Weaver and Company, Aurora, CO, USA). Electrical impedances were below 5KΩ at all sites prior to recording. Data were recorded at a sample rate of 1000 Hz filtered DC to 200 Hz with an additional notch filter at 60 Hz to filter out sources of high-frequency noise.

Sleep EEG was recorded from twelve scalp sites placed according to the International 10 – 20 system for electrode placement at F3, Fz, F4, C3, Cz, C4, P3, Pz, P4, O1, Oz, and O2 (Pivik et al., 1993). Two electrodes placed on the outer canthi of the

right and left eyes were used to record electrooculography, and chin electromyography was recorded using a bipolar channel created from two electrodes placed under the chin. Data were referenced online to an electrode placed at Fpz and grounded to an electrode placed at AFz. Sleep records were scored using primarily sites F4, C4, and O4. Sites F3, C3, and O3 were used when the signal from the primary channel was obscured. Electrodes placed at the right and left mastoids were used for offline re-referencing of scalp EEG and electrooculography channels to the contralateral mastoids for sleep scoring and to the average of the two mastoids for quantitative EEG analyses.

Sleep records were scored by two trained scorers in accordance with the standard criteria defined by the American Academy of Sleep Medicine for Night 2 only (Iber, Ancoli-Israel, Chesson, & Quan, 2007). The first scorer initially scored all of the records before the second sleep scorer reviewed them for agreement. Points of disagreement were resolved through discussion. A display filter removing frequencies below 0.3 Hz and above 30 Hz was applied at all scalp sites to aid in sleep scoring by removing slow sweat artifact and high-frequency movement artifact respectively. Sleep records were divided into 30 second epochs of either Wake, Stage 1 sleep, Stage 2 sleep, SWS, or REM sleep. Sleep efficiency, the time spent in each stage of sleep, the percentage of total sleep spent in each stage of sleep, and the latency to each stage of sleep from lights out were calculated for each participant.

Power spectral analysis of sleep EEG was conducted using Fast Fourier Transformation (FFT) techniques with Neuroscan 4.5 software (Compumedics Inc., Abbotsford, Australia) on five-minute segments from each stage of sleep. Early Stage 2 and SWS were sampled by selecting the first consecutive five-minute period of each

stage that did not include any arousals or transitional stages. Because the first REM period typically occurring approximately 90 minutes into sleep was generally transient and sometimes non-existent, the REM sleep segments were sampled using the same method but from the more well-defined REM period beginning approximately 180 minutes into sleep. To investigate whether potential relationships between Stage 2 sleep and forgetting were specific to the beginning of the night, a Late Stage 2 sleep segment was sampled by selecting the last consecutive five-minute period of Stage 2 sleep that did not include any arousals or transitional stages. Artifacts in the EEG (e.g., movement) were detected through visual inspection of the sleep records and removed prior to FFT analysis, which was conducted using two-second Hanning windows with 75% overlap. The full spectral power distribution resulting from FFT was reduced into absolute power values reflecting the average power ($\mu V^2/Hz$) found in conventional frequency bands including delta (1 – 4 Hz), theta (4 – 8 Hz), alpha (8 – 12 Hz), slow sigma (12 – 14 Hz), fast sigma (14 – 16 Hz), beta (16 – 30 Hz), and gamma (30 – 70 Hz). To achieve normal distributions, band power values were log10 transformed.

FFT analyses were carried out at all twelve sites for each of the conventional frequency bands; however, to avoid inflation of the type I error rate, further statistical analyses were performed with a focused approach using previous research on sleep and memory to guide the selection of specific frequency bands from NREM and REM sleep and the scalp regions from which to measure them. Inspection of the data revealed no clear evidence of lateralization, and without theoretical rationale for lateralization, it was decided to divide the twelve scalp sites into frontal, central, parietal, and occipital regions of interest by averaging across the three sites from each scalp region. Delta power and

fast sigma power in NREM sleep were selected to test the predictions that delta power would relate to better memory of TBR items and fast sigma power would relate to greater directed forgetting. Fast sigma power was measured from the parietal region because it is thought to reflect the activity of centro-parietal fast sleep spindles (Doran, 2003). NREM delta power was chosen to be measured in the frontal region as previous research has directly linked frontal delta EEG to consolidation of declarative memories (Oudiette, Antony, Creery, & Paller, 2013). REM theta power, selected to test the prediction that it would relate to forgetting to TBF items, was measured centrally as REM theta power in this region has been shown to be influenced by the learning of paired-associates (Fogel et al., 2007). Finally, because recent research has suggested a temporal and functional link between gamma activity and NREM slow-wave activity, NREM gamma power was also measured frontally and examined in statistical analyses to explore its potential role in memory reconsolidation (Valderrama *et al*., 2012).

Procedure

The full study procedure was cleared by the Research Ethics Board at Brock University and began with an off-protocol sleep screening and adaptation night. Participants came to the laboratory at 21:00 hr to start the screening and adaptation night. Participants were given a tour of the laboratory, and informed consent was collected from all participants at this time (Appendix G). Participants were then given a twenty-minute demonstration of the memory task that included detailed instructions, four trials for them to observe, and eight trials for them to complete. Experimenters were available for questions throughout the demonstration and ensured all participants completely understood the task. Following the demonstration, participants were prepared for standard

clinical polysomnography measuring respiratory airflow and chest effort, chin and leg electromyography, electrocardiography, electrooculography, and EEG from scalp sites C3, C4, O1, and O2. Participants were then given a sleep opportunity from 23:00 hr to 07:00 hr. These recordings were screened for evidence of poor sleep quality and evidence of possible sleep disorders. The various electrodes and sensors were removed in the morning, and participants were offered breakfast options of oatmeal, granola bars, and juice. At 08:00 hr, participants completed the Multidimensional Aptitude Battery-II, which took participants approximately 90 minutes to complete and yielded scores on various measures of intellectual functioning grouped into a verbal and performance category (Jackson, 1998).

Following the screening and adaptation night, participants were scheduled to participate in the main study. Participation was always scheduled for at least two days after the adaptation night and typically within the following seven days. The main protocol took place over three days and contained the three sessions of the memory task (learning, reminder, and test) as well as two nights of polysomnographically recorded sleep in the laboratory (Figure 1). All participants had their own bedroom for the duration of the study, and were asked to refrain from reading prior to sleep or using cell phones or other electronic devices that might distract their attention or interfere with the procedure. All participants were also asked to refrain from taking naps or consuming alcohol or caffeine for the duration of the study.

On the first day of the protocol, participants came to the sleep laboratory at 21:00 hr, were shown to their bedroom, and then given instructions for the learning session of the memory task. All participants completed the learning session at the desktop computer

in their assigned bedroom. When the task completed at approximately 22:00 hr, participants were taken to a separate room where the electrodes for polysomnography were applied. Participants were then taken to their bedroom where they could relax while the experimenter set up the recording equipment. This whole process generally took between 30 and 50 minutes. If the participant was ready for lights out more than 15 minutes early, they were offered a deck of playing cards to pass the time. At 22:55 hr, participants were given the pre-sleep questionnaire used to further screen participants for compliance with instructions and unusual events that may have happened during their day. Lights out occurred 23:00 hr, and participants were left undisturbed to sleep until 07:00 hr the following morning.

Upon awakening, participants were given the post-sleep questionnaire asking them to assess their night of sleep and current subjective state with regard to fatigue, sleepiness, and pain. All electrodes were then removed, and breakfast was again offered to all participants. They were then allowed to leave the laboratory and go about their day before returning to the laboratory at 21:00 hr. At this time, participants were given the instructions for the reminder session and taken to their bedroom to complete the task. The procedure for the remainder of the Night 2 was identical to that of the Night 1.

Participants were woken up at 07:00 hr on the third day of the protocol and given another copy of the post-sleep questionnaire to complete. Electrodes were removed, and participants were offered breakfast. Participants were asked to wait in the laboratory until the start of the test session at 09:00 hr. To pass the time until the test, they were given a selection of movies they could watch on a television set. At the end of the test session, participants were debriefed about the nature of the study and given a short questionnaire

asking them if they expected either Block A or Block P to be included in the test session and what strategies they used when learning the syllable pairs (Appendix B). Participants were then thanked for their participation in the study.

Statistical Analyses

Key variables from the memory task and power spectral analyses are defined and presented with their relevant predictions in Table 1.

Test assumptions. Before analyses could be conducted on the forgetting scores, the relearning scores, and the sleep variables of interest, the distributions of such scores were checked for normality. This was first done through visual inspection of the histograms and normal probability plots of each distribution. Skewness and kurtosis were assessed by calculating a *z*-score for both skewness and kurtosis and evaluating them at a critical *z* score of 1.96 ($p < .05$). Distributions found to deviate significantly from normality are identified in the results. If such distributions could not be made approximately normal through transformation, non-parametric tests were used. For all between-group comparisons using a *t*-test or analysis of variance (ANOVA), Levene's test for equality of variances was used to test the assumption of homogeneity of variance. When it was found that this assumption was not met, the unpooled estimate of the error term was used, and degrees of freedom were adjusted using the Welch-Satterthwaite method (Satterthwaite, 1946). For ANOVAs with repeated measures, Mauchly's test of sphericity was used to test the assumption of sphericity, and if this assumption was not met, the Greenhouse-Geisser correction was used to adjust the degrees of freedom (Greenhouse & Geisser, 1959).

Assumptions underlying multiple regression analyses were examined at the final

model predicting each dependent variable in each group. Specifically, histograms and normal probability plots of residuals as well as *z*-tests for skewness and kurtosis were used to determine if the model met the assumption of normality in residuals. Plots of standardized residuals versus standardized predicted values were used to identify heteroscedasticity, and tolerance values less than 0.25 were used to identify collinearity. For all reported models, the distributions of residuals were found to be approximately normal and no evidence of heteroscedasticity or collinearity was found. Cases were also examined for their influence in the regression model, using leverage values over 0.5 to identify extremity on the predictors, externally studentized residuals over 2 to identify extreme discrepancy, Cook's *D* values over 1 to identify cases with high influence on the regression coefficient, and standardized DFBETA values over 1 to identify high influence on individual predictors.

Outlier treatment. Each variable was scanned for statistical outliers ($|SD| > 3$). One case was found to be an outlier in Early Stage 2 gamma power. This case was inspected and found to lie well outside the otherwise normal distribution of Early Stage 2 gamma values. However, it also appeared to be a legitimate measure of gamma power and was not removed from analyses for this reason. Because such an extreme case could have a large impact on statistical estimates, the decision was made to winsorize this case to the 95th percentile on Early Stage 2 gamma power. No other statistical outliers were found within either group separately or the sample as a whole for any other of the variables presented here.

Preliminary analyses. In initial inspection of the data, the paired-samples *t*-tests revealed learning performance in Block P to be significantly worse than learning

performance in the TBF Block A (t (30) = 3.17, p = .004) and the TBR Block B (t (30) = 4.16, p < .001). Further, an additional four participants did not meet the 60% criterion in the last Syllable Trial of Block P. This difference in learning performance between blocks is a confound in the comparisons that can be made between Block P and the TBR and TBF blocks. For this reason, Block P was removed from all main analyses. Analyses concerning Block P performance were conducted on a reduced sample reflective of the 60% criterion; these analyses are reported in Appendix H.

Independent *t*-tests were planned for direct comparisons between the Stable and Labile groups on the number of errors made during learning, Night 2 sleep architecture, and Night 2 power spectral values. The primary use of such comparisons was to assess whether the groups differed on variables expected to be similar between groups due to random sampling. Similarly, paired-samples *t*-tests were planned for within-group or full sample comparisons of the errors made during learning the TBR and TBF blocks.

Hypothesis testing.

Forgetting scores. Forgetting scores were analyzed using a 2 ×2 Group by Block mixed-model ANOVA to compare performance on the TBR and TBF blocks between the Stable and Labile groups. Due to the properties of a 2 x 2 mixed-model ANOVA and the variable definitions used, a test of the main effect of Group is equivalent to a between-group comparison of total forgetting scores, and a test of the interaction is equivalent to a between-groups test of directed forgetting scores (Anderson et al., 1980). In the event of a significant interaction, follow-up paired-samples *t*-tests were planned to examine the simple main effect of Group on performance in each Block.

Relearning errors. Relearning errors were planned to be analyzed with the same

methods used for the forgetting scores. However, nearly 40% of the sample did not make any errors during either the TBR or TBF relearning trials. To allow for group comparisons with such low variability in relearning errors, dichotomous variables were created. Participants were split into those who did make at least one relearning error in a particular block and those that made no relearning errors in that block. A similar dichotomous variable was created to separate those that did not make a relearning error in either the TBR block or the TBF block from the participants that did make at least one relearning error in these blocks. Finally, to address a possible directed forgetting effect, the participants were divided into those that made more errors on the TBR block than the TBF block, those that made more errors on the TBF block than the TBR block, and those that made the same number of errors on each of these blocks. Chi-squared tests were used to test whether the relative proportions of participants making relearning errors differed between Groups or Blocks.

Sleep and forgetting. The predicted relationships between the forgetting scores and the power spectral variables of interest were tested by calculating the Pearson's correlation coefficient for each pair of forgetting score and power spectral variable separately within each group. Similar correlations between the forgetting scores and the percent time spent in each stage of sleep were also conducted. To test whether or not correlations between forgetting scores and power spectral variables of interest showed specificity for either the Stable or Labile group, significant correlations were followed with a regression model predicting the particular forgetting score from the interaction of group membership and the particular power spectral variable of interest. These interaction terms were tested for significance at the second step of a hierarchical model

that accounts for the variance of both the unique effects group membership and the sleep variable on the first step.

Because multiple processes of sleep likely contribute to affect memory performance, stepwise regression analyses were employed to better understand the relationships between the power spectral variables of interest and the forgetting scores in each group. A stepwise regression predicting a particular forgetting score was conducted in a particular group when at least one significant bivariate correlation was found in that group between that forgetting score and the power spectral variables of interest. In each step of the stepwise regressions, the strongest predictor that could significantly ($p < .05$) account for the most variance in the criterion unaccounted for by variables already in the model was entered. The variables in the model were then reanalyzed, and any variables that no longer significantly ($p \geq .05$) accounted for a unique amount of variance in the criterion were removed from the model. To reduce the likelihood of collinearity among predictors and because functional roles of delta power, fast sigma power, and gamma power would likely be comparable across all NREM sleep samples, only Early Stage 2 delta, fast sigma, and gamma power were included in the regression analyses. Early Stage 2 measures were chosen over SWS and Late Stage 2 measures because they generally showed stronger bivariate correlations with the forgetting scores.

Results

Preliminary Analyses

Baseline learning performance. Participants were randomly assigned to the Stable and Labile groups, and therefore performance during the learning session should not have differed between groups. Similarly, syllable lists in Block A (later given TBF instructions) and Block B (later given TBR instructions) were intended to be equivalent, and therefore performance should not have differed for these blocks before the directed forgetting instructions were given. Thus, the errors made during the learning session were examined to identify any unexpected baseline differences in how well participants were able to learn the TBF Block and TBR blocks prior to receiving the group manipulation or directed forgetting instructions. From the detailed analyses of learning performance (to follow), it can be concluded that the degree to which the lists were learned prior to the group manipulation or directed forgetting instructions was approximately equal across groups and blocks. Further, the calculation of the forgetting scores was designed to take into account any marginal differences present at the end of the learning session.

In general, the performance of both groups in these blocks progressed to near ceiling over the eight Syllable Trials (Figure 4). Special attention was paid to the number of errors made on the last Syllable Trial of each block because these were the counts used in calculation of the forgetting scores and indicate the strength of memory for each list at the end of the learning session. Performance in these learning session trials was near ceiling with 81% of participants making zero errors in the last Syllable Trial of the TBR block and 77% of participants making zero errors in the last Syllable Trial of the TBF block, indicating that in both blocks the majority of participants completely learned the

list of syllable pairs. Both the total number of errors made in baseline learning of the TBR and TBF blocks and the number of errors made in just the last learning session Syllable Trials of each block were found to be positively skewed. For the total number of errors, the distribution was normalized with a log10 transformation; however, for the number of errors made in the last Syllable Trial of each block, the distributions could not be made approximately normal, and non-parametric tests were conducted.

A Group (Stable vs. Labile) by Block (TBR vs. TBF) mixed-model ANOVA was conducted to investigate whether baseline learning performance differed across groups or blocks before these experimental manipulations took place. The main effect reflecting later group membership was found to be non-significant ($F (1, 29) = 1.79$, $p = .191$, $\eta^2 = .058$). Both the main effect of Block ($F (1, 29) = 4.03$, $p = .054$, $\eta^2 = .122$) and the Group by Block interaction ($F (1, 29) = 3.93$, $p = .057$, $\eta^2 = .119$) were found to be marginally significant. The absence of significant effects suggest that learning performance across groups and blocks were approximately equal; however, to further understand any possible confounds in the interpretation of the results, the marginally significant Group by Block interaction was followed with tests of the simple main effect of Block. Doing so found that the Labile group made significantly more errors in learning of the TBF block ($M = 9.73$, $SD = 5.23$) compared to the TBR block ($M = 6.27$, $SD = 4.04$; $t (30) = 2.73$, $p = .016$) despite the fact that no group or block manipulations had been implemented at the time of the learning session. The Stable group, on the other hand, showed similar baseline learning performance between the TBF block ($M = 5.88$, $SD = 3.88$) and the TBR block ($M = 6.31$, $SD = 4.59$; $t (30) = 0.02$, $p = .984$).

While the difficulty in learning each block is important to consider, the degree to

which participants learned the blocks of syllable pairs by the end of the learning session is of greater importance when examining how many syllable pairs were forgotten over the course of the procedure. In comparisons of only the last learning session Syllable Trial of each block, the Wilcoxon signed-rank test found no significant differences between the number of errors made in the TBR and TBF blocks in analyses of the full sample (Z = -0.97, p = .344), the Stable group alone (Z = -0.82, p = .414), or the Labile group alone (Z = -1.73, p = .084). However, the latter group did maintain a marginally significant difference of more errors in the TBF block than the TBR block. In between-group comparisons, the Mann-Whitney-U test found no significant group differences in the number of errors made in the last learning session Syllable Trial of the TBR block (U = 106, p = .419) or the TBF block (U = 92, p = .130). Thus, while the Labile group may have shown a tendency to make relatively more errors in learning the TBF block, these results suggest that the two groups did not differ greatly in the strength to which they learned either the TBR or TBF blocks prior to receiving the group manipulation or directed forgetting instructions.

Sleep architecture and EEG band power. Before examining the relationships between sleep and forgetting in Stable and Labile groups, it is important to compare these groups for differences in sleep architecture and EEG band power during sleep. Because the only thing separating the two groups in the experiment was the rather subtle memory reactivation manipulation, group differences in sleep characteristics were not expected.

Basic sleep architecture from the second experimental night and the related between-group comparisons are reported in Table 2. In summary, groups did not differ significantly in wake time; total sleep time; sleep efficiency; stage 1, stage 2, or SWS

onset latency; the time spent in stage 1, stage 2, or SWS; or the percentage of total sleep time spent in stage 1, stage 2, or SWS. However, compared to the Stable group, the Labile group showed greater pressure for REM sleep, including a significantly greater amount of REM sleep, a significantly greater percentage of REM sleep, and a significantly shorter REM sleep latency on average. Although the groups did not significantly differ in measures of SWS, inspection of the group means for minutes in SWS and the percentage of SWS suggests that the Labile group experienced relatively more REM sleep at the cost of less SWS. As only the second night of sleep was scored according to sleep stages, it is not clear whether this greater pressure for REM sleep in the Labile group was driven by the between-group memory reactivation manipulation or pre-existing group differences.

Comparisons of the Stable and Labile group means for the Night 2 power spectral variables of interest are reported in Table 3. Group means and comparisons for Night 2 EEG power in all frequency bands, measured globally across all twelve scalp sites, are reported in Appendix I. Concerning the power spectral variables of interest, the Stable group, in comparison to the Labile group, was found to have significantly greater REM theta power and Early and Late Stage 2 delta power. Groups did not differ significantly in delta, fast sigma, or gamma power during SWS or fast sigma or gamma power during Early or Late Stage 2 sleep. From these data, it cannot be certain as to whether these group differences reflected stable individual differences between the groups, were the result of the experimental manipulations, or emerged by chance through other sources of variability such as the sampling method used or measurement error. The subtle group manipulation was not expected to significantly impact sleep, and, because of random

sampling, baseline group differences in sleep characteristics were not anticipated. *Post hoc* analyses (reported in their own section) were conducted to further explore these group differences in Night 2 sleep.

Hypothesis Testing

Forgetting scores. To test the prediction that the Labile group would show stronger directed forgetting compared to the Stable group, a Group (Stable vs. Labile) by Block (TBR vs. TBF) mixed-model ANOVA was conducted. Contrary to the prediction, the two groups did not differ significantly in their directed forgetting scores as the Group by Block interaction was found to be non-significant ($F (1, 29) = 1.54$, $p = .225$, $\eta^2 = .050$). The main effect of Group was also found to be non-significant, indicating that the Stable and Labile groups did not differ significantly in total forgetting scores either ($F (1, 29) = 1.48$, $p = .703$, $\eta^2 = .005$). Finally, there was also no evidence for greater TBF forgetting than TBR forgetting in general as the main effect of Block was found to be non-significant as well ($F (1, 29) = 4.36$, $p = .514$, $\eta^2 = .014$). Means for forgetting scores by group are reported in Table 4.

Relearning errors. The first Syllable Trials of each block in the test session measured the forgetting of the syllable lists and also revealed the correct syllable pairs to the participants. The remaining three Syllable Trials in each block were used as a second and unique measure of memory strength as they measured the difficulty for each participant to relearn the syllable pairs of each block. The numbers of errors made during these relearning trials were thus examined for evidence of either the memory stability or directed forgetting manipulations.

In general, relearning errors were minimal for both the Stable and Labile groups for

both the TBR and TBF blocks. In both groups combined, only 45% of participants made an error in relearning the TBR block, and only 42% made an error in relearning the TBF block. Combining both blocks, it was found that 39% of participants did not make a single relearning error, and an additional 36% of participants made only one or two errors across the TBR and TBF trials. Thus, while participants did show forgetting of the syllable pairs of both lists, the majority quickly relearned the material with only one presentation of the correct pairs.

Chi-squared tests showed that for the TBR block, the TBF block, and both blocks combined, the proportion of participants making at least one error in the relearning trials did not differ significantly between the Stable and Labile groups (all *p*s > .832). These groups also did not differ significantly in their distributions of participants who made more relearning errors in the TBR block than the TBF block, participants who made more relearning errors in the TBF block than the TBR block, and participants who made the same number of relearning errors in each block ($p = .774$). Further, a one-sample chi-squared test found that the number of participants in each of these three cells did not differ significantly from those expected by chance ($p = .303$). Together, these results suggest that, like the forgetting scores, there were no effects of either the memory stability or directed forgetting manipulations on the relearning errors made.

Sleep and forgetting. No effect of the memory reactivation treatment was found in between-group comparisons of forgetting; however, this does not preclude characteristics of sleep from having had an effect on forgetting of TBR and TBF items either similarly or differently in the two groups. Thus correlations between sleep characteristics and the four forgetting scores were examined in both the Stable and Labile groups. It was predicted

that, in the Labile group, NREM delta power would negatively correlate with TBR forgetting, REM theta power would positively correlate with TBF forgetting, and NREM fast sigma power would positively correlate with directed forgetting. Correlation coefficients for these predicted relationships and all other relationships between the four forgetting scores and the power spectral variables of interest for both groups are reported in Table 5. Correlation coefficients for relationships between the four forgetting scores and the percentage of total sleep time spent in each of the stages are reported in Table 6. Findings of significant correlations between power spectral variables of interest and forgetting scores in particular groups were followed with stepwise regressions predicting forgetting scores from Early Stage 2 delta, fast sigma, and gamma power and REM theta power. Intercorrelations between these predictors are reported in Table 7. Finally, supplementary correlation coefficients for the relationships between the four forgetting scores and the EEG power in each frequency band at each scalp site during each sleep stage sampled are reported across tables in Appendix J.

TBR forgetting. TBR forgetting was predicted to be negatively correlated with NREM delta power in the Labile group. This prediction was found true as a significant negative correlation was found between Early Stage 2 delta power and TBR forgetting in the Labile group (Figure 5A). The correlation between TBR forgetting and delta power was not significant in either SWS or Late Stage 2 sleep in the Labile group. Although it was not specifically predicted, a significant negative correlation found between TBR forgetting and the percentage total sleep time spent in SWS is in line with the predicted relationship between TBR forgetting and NREM delta power as SWS was defined by the presence of delta EEG activity. None of the sleep stage percentages or power spectral

variables of interest were found to be significantly correlated with TBR forgetting in the Stable group. A regression analysis testing the Group by Early Stage 2 delta power interaction as a predictor of TBR forgetting was conducted to determine whether the relationship between Early Stage 2 delta power and TBR forgetting was exclusive to the Labile group. When accounting for unique effects of both Group and Early Stage 2 delta power, this interaction term was found to be a significant predictor of TBR forgetting (β = 4.66, t (27) = -2.65, p = .013), indicating that the relationship between Early Stage 2 delta power and TBR forgetting was specific for the Labile group.

TBR forgetting was also found to be negatively correlated with gamma power during both Early and Late Stage 2 sleep but not SWS. As with Early Stage 2 delta power, separate regression analyses testing the interactions of these variables with group membership showed that both the interaction between Group and Early Stage 2 gamma power (β = -4.09, t (27) = -2.75, p = .010) and the interaction between Group and Late Stage 2 gamma power (β = -0.52, t (27) = -2.40, p = .024) were significant predictors of TBR forgetting. This suggests that, like Early Stage 2 delta power, the relationship between TBR forgetting and Stage 2 gamma power is dependent on whether the memories were reactivated before sleep.

A stepwise regression was conducted in the Labile group to better understand the relationships between Early Stage 2 sleep characteristics and the forgetting of reactivated TBR items. At the first step of this analysis, Early Stage 2 gamma power was entered as the strongest significant predictor of TBR forgetting (R^2 = .51, F (1, 13) = 13.76, p = .003). At the second and final step, Early Stage 2 delta power was also entered as a unique predictor of TBR forgetting (R^2 = .66, F (2, 12) = 11.57, p = .002; Figure 5B). In this

final model, both Early Stage 2 gamma power (β = -0.57, t = -3.16, p = .008) and Early Stage 2 delta power (β = -0.41, t = -2.25, p = .044) served as significant predictors with gamma power uniquely accounting for 28% and delta power uniquely accounting for 14% of the total variance in TBR forgetting. Thus, in the Labile group, both greater gamma and greater delta frequency EEG during Early Stage 2 sleep were uniquely associated with greater memory of TBR items.

TBF forgetting. It was hypothesized that REM theta power has an active role in forgetting processes, and thus a positive correlation between REM theta power and TBF forgetting in the Labile group was predicted. Although no significant correlations were found between TBF forgetting and any of the power spectral variables of interest in either group, this predicted positive correlation did reach marginal significance. Further, a regression model conducted to follow up on a significant Group by REM theta power interaction predicting directed forgetting scores (reported under directed forgetting) found unexpected results with regard to REM theta power and TBF forgetting. It was found that REM theta power was a significant and unique predictor of TBF forgetting when the analysis was conducted in the full sample controlling for individuals' group membership (i.e., "the main effect of REM theta power;"r = .399, β = 0.48, t (27) = 2.48, p = .020; Figure 6). Although this was not a predicted result, it suggests that REM theta power may have a role in the forgetting of previously consolidated memories regardless of whether or not they were reactivated prior to sleep.

Total forgetting. No predictions were made regarding total forgetting in either the Stable or Labile groups, and no significant correlations were found between total forgetting and any of the sleep stage percentages or the power spectral variables of

interest in the Labile group. However, an unexpected significant negative correlation was found in the Stable group between total forgetting and the percentage of total sleep time spent in Stage 1 sleep. This finding may be spurious or may indicate that deeper stages of sleep have a role in the forgetting of stable memories generally. A second unexpected significant correlation was also found as REM theta power was positively correlated with total forgetting in the Stable group (Figure 7). While neither this finding nor the previously mentioned relationship between REM theta power and TBF forgetting were predicted, these results together suggest that REM theta power may indeed play a general role in the forgetting previously consolidated memories. In a follow-up regression model, the Group by REM theta power interaction term was not a significant predictor of total forgetting ($\beta = -0.38$, $t\ (27) = -1.49$, $p = .148$).

A stepwise regression using Early Stage 2 delta, fast sigma, and gamma power and REM theta power as potential predictors of total forgetting in the Stable group found no significant predictors of total forgetting beyond REM theta power. In the only step of this regression, REM theta power emerged as the only significant predictor accounting for 25% of the variance in the total forgetting scores of the Stable group ($\beta = .50$, $F\ (1, 13) = 4.78$, $p = .046$; Figure 5B). A single case with a large discrepancy from its predicted value (externally studentized residual = 2.45) was identified as having a high degree of influence on the REM theta power coefficient (standardized DFBETA = -1.24). Reanalysis with the exclusion of this case also yielded a significant model with REM theta power as the only predictor, this time accounting for 47% of the variance in total forgetting ($\beta = .68$, $F\ (1, 13) = 11.41$, $p = .005$).

Directed forgetting. A positive correlation between directed forgetting and NREM

fast sigma power was predicted in the Labile group. This relationship reached statistical significance in both Early Stage 2 sleep (Figure 8A) and SWS, but not Late Stage 2 sleep. While this predicted result may indicate a relationship between NREM fast sigma activity and the selectivity of memory reconsolidation, it is important to note that significant positive correlations with directed forgetting in the Labile group were also found with REM theta power (Figure 8B) and Early Stage 2 gamma power. None of the sleep stage percentages or power spectral variables of interest were found to be significantly correlated with TBR forgetting in the Stable group. The interactions of group membership and Early Stage 2 fast sigma power ($\beta = 0.52$, t (27) = 1.64, $p = .114$), SWS fast sigma power ($\beta = 0.42$, t (27) = 1.74, $p = .093$), and Early Stage 2 gamma power ($\beta = 2.74$, t (27) = 1.79, $p = .084$) were all non-significant as predictors of directed forgetting when tested in separate regression models. However, REM theta power ($\beta = 0.69$, t (27) = 3.15, $p = .004$) was found to have a significant interaction with group membership in predicting directed forgetting scores.

To understand whether this interaction was driven by an association between REM sleep theta power and TBR forgetting or REM theta power and TBF forgetting, regression models testing the Group by REM theta power interaction were conducted within each block. As reported under TBF forgetting, greater REM theta power was associated with greater forgetting of TBF material in the full sample. In contrast the Group by REM theta power interaction was found to be a significant predictor of TBR forgetting as it was being be driven by two marginally significant bivariate correlations showing greater REM theta power associated with more TBR forgetting in the Stable group and less TBR forgetting in the Labile group. Thus, all these results come together

to suggest that REM theta power may play a role in the weakening of all previously consolidated memories except those that are both recently reactivated and of future relevance.

A stepwise regression predicting directed forgetting in the Labile group was conducted to better examine the contributions of Early Stage 2 sleep characteristics and REM theta power in directed forgetting scores. REM theta power emerged as the strongest significant predictor in the only step of this regression model accounting for 63% of the variance in directed forgetting scores of the Labile group ($\beta = .81$, $F(1, 13) = 24.31$, $p > .001$; Figure 5C). Although one case with a high discrepancy from the predicted value (externally studentized residual = 2.61) was identified, it did not have a high degree of influence on the REM theta power coefficient (DFBETA = 0.27) and therefore was not removed.

***Post Hoc* Analyses**

The Stable group was found to show greater Early and Late Stage 2 delta power and REM theta power during Night 2 sleep. These group differences were unanticipated and were further explored by calculating intraclass correlation coefficients to measure the night-to-night reliability of power measures and by conducting a Group (Stable vs. Labile) by Night (1 vs. 2) mixed-model ANOVA on each power spectral variable of interest to investigate possible effects of the experimental manipulations.

According to accepted thresholds for interpreting reliability estimates (Landis & Koch, 1977), REM theta power showed almost perfect reliability over the two nights in both the Stable and Labile groups. Although the Labile group showed substantial reliability in Early Stage 2 delta power, Early Stage 2 delta power in the Stable group and

Late Stage 2 delta power in both groups were not found to be very reliable over the two recorded nights. Intraclass correlation coefficients for night-to-night reliability of all power spectral variables of interest for each group are reported in Table 3. Night-to-night stability among individuals appears high for most of these variables. Night-to-night variability, measurement error, or treatment effects may contribute to the poor reliability found for some of the power spectral variables.

In all of the mixed-model ANOVAs conducted on the power spectral variables of interest, no significant Group by Night interactions were found (all *p*s > .105). A significant Night effect was only found for Early Stage 2 gamma power with this effect indicating a general decrease in power from Night 1 to Night 2 (F (1, 29) = 9.00, p = .006, η^2 = .105; all other *p*s > .317). Significant effects of Group were only found for Early Stage 2 delta power (F (1, 29) = 5.63, p = .024, η^2 = .163) and REM theta power (F (1, 29) = 10.83, p = .003, η^2 = .272) with both effects indicating greater power in the Stable group (all other *p*s > .125). Together with the intraclass correlations, these results suggest that the group differences in Night 2 REM theta power were likely the result of preexisting and stable individual differences, while additional sources of variability were involved in the group differences in Night 2 Early and Late Stage 2 delta power.

Discussion

It was hypothesized that one function of sleep would be to beneficially reprocess labile memories through the selective reconsolidation of memories of future relevance during NREM sleep and the weakening of memories of no future relevance during REM sleep. The study was designed to address this hypothesis by comparing overnight forgetting of both stable and labile memories cued to be either remembered or forgotten and relating these forgetting outcomes back to individuals' sleep neurophysiology. While only some of the specific predictions made were found true in this experiment, the pattern of results found do offer support to the proposed hypothesis. In review of the reported correlations between forgetting scores and sleep neurophysiology, the results suggest that NREM sleep is associated with the retention of reactivated memories of future relevance and that REM sleep is associated with the forgetting of previously consolidated memories.

Predictions

Directed forgetting instructions were expected to only have an effect in the Labile group because the proposed mechanism of selective memory reconsolidation would hypothetically only act on labile memories in need of reconsolidation. Thus, it was predicted that the Labile group would show greater directed forgetting than the Stable group. This effect was not found. The groups did not differ in either directed forgetting or total forgetting nor did forgetting differ between the TBR and TBF lists overall. This could indicate that the directed forgetting and memory reactivation manipulations had no effect on forgetting of the syllable pairs. It should be noted, however, that there was a large amount of individual variability within the limited range of TBR and TBF

forgetting scores. Participants varied a great deal in how well they generally retained memories of the syllable pairs regardless of list or group membership. Although significant proportions of this variability were accounted for within the groups by characteristics of sleep, it is likely that the high variability within a small possible range of scores resulted in between-group analyses that lacked sufficient power to identify effects at the group level.

However, when examining data at the individual level using regression, it was found that group membership interacted with NREM and REM sleep characteristics to predict both TBR forgetting and directed forgetting scores, respectively. These interactions were reflective of significant correlations in the Labile group between sleep and forgetting scores indicating memory selectivity for future relevance without such correlations in the Stable group. This suggests that although no group differences in forgetting scores emerged between the Stable and Labile groups, the memory reactivation treatment was effective in allowing sleep processes to treat TBR and TBF memories differently. Considering one sleep variable specifically, the results suggest that REM theta power acted on TBR and TBF memories differently in the Labile group, but not the Stable group, in which REM theta power only correlated with the total forgetting score.

Three specific predictions were made regarding forgetting scores and sleep neurophysiology in the Labile group. First, based on the documented role of SWS and slow-wave activity in memory consolidation (Diekelmann & Born, 2010) and the proposed role of NREM sleep in the selective reconsolidation of labile memories, NREM delta power was expected to negatively correlate with forgetting of TBR items. This relationship was indeed found, although it was only significant for delta power of Early

Stage 2 sleep and not SWS or Late Stage 2 sleep. As this relationship was only found in the Labile group, and not the Stable group, it is supportive of the hypothesis that the process of memory reactivation and reconsolidation is a phase in which memories can be beneficially reprocessed for future relevance. Further, the finding that NREM sleep characteristics delta and gamma power were associated with less forgetting suggests that NREM sleep characteristics active during the second-night reprocessing of memories may be similar to those typically active during first-night consolidation of memories.

It was also found that the relationship between delta frequency EEG and better memory of TBR items was limited to or at least most robust during the earliest part of sleep. Although this particular finding appears inconsistent with the proposed importance of SWS in memory consolidation (Diekelmann & Born, 2010), the results regarding TBR forgetting and delta power in general are not surprising. First, the percentage of total sleep time spent in SWS was found to negatively correlate with forgetting of TBR material in the Labile group, suggesting that SWS does play a role in the retention of these memories. Further, the learning-driven changes often found in SWS, such as increased delta and sigma activity, have similarly been found during Stage 2 sleep in other studies (Ruch et al., 2012). It has also been suggested that changes in neural circuitry can take place from brief bursts of activity and that longer durations of sleep may be more important for the integration of new memories with those that have been previously consolidated (Bi & Poo, 1998; Poe, Walsh, & Bjorness, 2010).

Because of its proposed role in synaptic plasticity and regulation of memory resources (Poe et al., 2000; Grosmark et al. 2012), REM theta power was predicted to positively correlate with the forgetting of TBF material in the Labile group. In this group,

memories of the TBF material were proposed to be left to degrade due to selectivity in memory reconsolidation. REM theta activity was proposed to play a role in this weakening of the memories. This correlation did reach marginal significance, but more interesting results regarding REM theta power emerged as well. An interaction between REM theta power and group membership was significant in predicting directed forgetting. Overall, this interaction appears driven by a tendency for REM theta power to be associated with the forgetting of all memories except those that were both recently reactivated and of future relevance.

It was also predicted that directed forgetting scores would positively correlate with NREM fast sigma power in the Labile group. This prediction was based on the finding from Saletin et al. (2011) showing, in an item-method paradigm, a positive correlation between the directed forgetting effect and the number of fast sleep spindles. Consistent with these findings, fast sigma power during both Early Stage 2 sleep and SWS were found to correlate with directed forgetting scores in the Labile group. Although this correlation was not significant in the Stable group, the lack of a significant interaction of group membership and Early Stage 2 fast sigma power on directed forgetting indicates that the apparent specificity of this relationship for labile memories is not a statistically significant result. However, REM theta power was found to not only show this statistically significant specificity for the Labile group in predicting directed forgetting scores, but it also emerged as the only significant predictor of directed forgetting in the stepwise regression analysis.

Early Stage 2 fast sigma power did not significantly predict directed forgetting over and above REM theta power. This, however, is not in contradiction with the finding of

Saletin et al. (2011) that fast sleep spindles were correlated with the directed forgetting effect as many differences separate their methodology from the methodology of the current research. Most notably, the current research administered list-method directed forgetting instructions a full twenty-four hours after the memories were initially encoded, whereas Saletin et al. (2011) employed item-method directed forgetting instructions at the time of encoding. In addition, the current research did not contain a measure of fast sleep spindle density and instead relied on the measure of fast sigma power during NREM sleep to indirectly measure fast sleep spindles. While fast sigma activity in the 14 to 16 Hz range may serve as a useful approximation of fast sleep spindle activity, non-spindle EEG activity in this frequency range was also included in the measure of fast sigma power. For these reasons, the findings reported here do not address the role of fast sleep spindle activity in the selectivity of memory consolidation.

These reported results should also not rule out NREM fast sigma power or fast sleep spindles from having a role in the reprocessing of memories. Although it may be the case that the significant correlation between Early Stage 2 fast sigma power and directed forgetting is simply the result of the high intercorrelation between this predictor and REM theta power, it may also be the case that these two predictors are related, share similar processes, and thus account for the same variance in directed forgetting scores. Further, the emergence of REM theta power as the single significant predictor may not be a stable result. The exact magnitude of the correlations between the power spectral variables and directed forgetting scores may not be very stable from sample to sample. Although REM theta power emerged as the strongest predictor of directed forgetting in this sample, Early Stage 2 fast sigma power could emerge as a stronger predictor in a

different sample. Finally, measures of fast sleep spindles more pure than fast sigma power can be obtained by visually counting fast sleep spindles and calculating fast sleep spindle density. If fast sleep spindles play a strong role in memory efficiency and directed forgetting, these direct measures of spindles will likely correlate more strongly with directed forgetting than fast sigma power could. Thus, one should not preclude a role for fast sigma activity or fast sleep spindles in either reconsolidation or directed forgetting merely from the data presented here. Direct analyses of spindle activity instead of NREM fast sigma power are underway and may help to separate roles for both fast sleep spindles and REM theta power in the reprocessing of memories.

Integration of Major Findings

The first major finding of this study is the significant relationship between NREM sleep characteristics Early Stage 2 delta and gamma power and TBR forgetting. Both of these features of Early Stage 2 sleep showed specificity for the Labile group in their association with less forgetting of material they were informed would be on the test, and together they accounted for 66% of the variance in TBR forgetting in the Labile group. In this experiment, the only difference separating the Stable group from the Labile group is the prediction error of the Cue-Only reminder trials thought to trigger memory reactivation in the Labile group. Given that the reactivation manipulation appeared to allow the association between NREM sleep characteristics and TBR forgetting, that reconsolidation processes would hypothetically only act on reactivated memories, and that the same NREM sleep characteristics have been associated with memory consolidation, it seems likely that Early Stage 2 delta and gamma power are a reflection of reconsolidation processes acting selectively on memories of future relevance. As stated

previously, this process of selective reconsolidation of labile memories may naturally serve to stabilize updates to complex memories that took place during wakefulness. Components of the memories that still have future relevance are selectively reconsolidated while components that are no longer relevant can be passed over and left to degrade to prevent future interference.

The finding that REM theta power significantly accounted for 25% of the variance in total forgetting scores in the Stable group and 63% of directed forgetting scores in the Labile group is another key outcome from this study. This finding supports and expands upon the hypothesized role for REM sleep proposed by Poe et al. (2000). It was proposed that REM sleep selectively strengthens recently acquired memories through reactivation during the peaks of the theta rhythm while also depotentiating older memories through reactivation during the troughs of the theta rhythm. The results reported here suggest that it is not only new memories that are selectively strengthened but also recently reactivated memories thought to be of future relevance. In contrast, recently reactivated memories thought to have no future relevance as well as stable memories both with and without future relevance all appear to be weakened by these REM sleep processes. While it was predicted that REM theta activity would play in role in forgetting TBF memories in the Labile group, the memories of the Stable group were expected to be largely exempt from processing during sleep. The data suggest this was not the case and that all memories, or at least those that have been recently accessed, are subject to further processing.

Given that the forgetting associated with theta EEG activity during REM sleep was general in the Stable group and not specific to memories that should be forgotten, it should be noted that the apparent function of REM sleep may not always be beneficial for

performance. This is not unprecedented. Recent research from Oudiette et al. (2013) investigating the role of sleep in the retention of memories of varying monetary value found that greater time spent in REM sleep over a 90-minute nap was related to weaker memories for low-value items. As these items still had some monetary value associated with them, there was no direct benefit from forgetting low-value items. Importantly, the relationship between REM sleep and weakening of low-value memories was not found when externally-cued reactivation was used to increase the consolidation of low-value items during NREM sleep. This is interesting as results from the current research suggest that theta activity during REM sleep led to forgetting of all material except TBR material in the labile group, for which greater NREM delta and gamma power were associated with better memory performance. Thus, it may be the case that rather than having an active role in strengthening memories based on relevance or value, REM sleep functions more generally to weaken all memories not specifically consolidated (Oudiette et al., 2013) or reconsolidated (the current research) during NREM sleep.

Considered this way, the results of the current study offer partial support to the logic of the synaptic homeostasis hypothesis (Tononi & Cirelli, 2006). This hypothesis is based on the assumption that sleep must contain a downscaling mechanism to counteract the net increase in total synaptic potentiation that takes place during wakefulness. Slow-wave activity during NREM sleep was originally proposed as a means for synaptic downscaling. However, recent evidence suggests that theta activity during REM sleep may play a greater role in synaptic plasticity (Grosmark et al., 2012), and Tononi and Cirelli (2012) maintain that the downregulation of synapses could be achieved through a number of candidate processes. The data presented here do give support for the proposed

existence of a downscaling mechanism during sleep, and, given the observed relationships between REM theta power and forgetting, more support is given to the idea that this synaptic downscaling process takes place during REM rather than NREM sleep.

In the synaptic homeostasis hypothesis, benefits in memory strength are proposed to be driven primarily through increases in the signal-to-noise ratio occurring when weak synapses are downscaled to ineffectual levels and strong synapses, also downscaled, remain relatively strong in comparison. This is presented as a more indirect process than the active process of memory consolidation. In regard to NREM sleep specifically, it is noted that the positive associations between TBR forgetting and both delta and gamma power were not accompanied by any increases in forgetting in the Labile group. This pattern is consistent with other findings suggesting that slow-wave and gamma activity of NREM sleep strengthen memories of future relevance through an active process, and not one of general downscaling (Diekelmann & Born, 2010; Le Van Quyen et al., 2010; Wilhelm et al., 2011). However, results regarding theta activity during REM sleep again suggest that it is REM sleep that best fits the characteristics of the synaptic downscaling mechanism proposed by Tononi and Cirelli (2006). In terms of the synaptic homeostasis hypothesis, REM theta power may have served to downscale all memories indiscriminately but increase memory efficiency (i.e., directed forgetting scores) in the Labile group as the recently reconsolidated TBR memories became greater in relative strength compared to the competing TBF memories that did not receive reconsolidation.

In summary, the results of the current research generate some clarifications and modifications for the initial hypothesis that one function of sleep may be to selectively reconsolidate labile memories of future relevance during NREM sleep and weaken labile

memories of no future relevance during REM sleep. It is suggested that both NREM and REM sleep factors play important roles in both memory processing and reprocessing. Memory reactivation in concert with NREM slow-wave activity may serve to both consolidate newly acquired relevant memories and reconsolidate labile relevant memories (Rasch et al., 2007). Memories may then undergo a second process taking place during REM sleep in which all memories are weakened to regulate memory and energy resources through a downscaling mechanism that may function within the troughs of the theta rhythm (Poe et al., 2000; Tononi & Cirelli, 2006). These processes are suggested to be primary in reconsolidation and forgetting, but there are likely complementary roles for fast sigma activity in NREM sleep in memory processing and and REM sleep theta activity in the strengthening of memories. At present, it is unclear what memories are subject to downscaling processes. Future research should continue to investigate the role and mechanisms of REM sleep processes in both the strengthening and weakening of memories, perhaps through the induction of memory reactivation, to determine if and how REM sleep may act differently on old, new, and reactivated memories.

Limitations and Future Directions

In discussion of this proposed role for sleep, it is important to consider the limitations of the current research. First, the study had only limited power for comparisons of forgetting scores, and it is not clear whether or not differences between the Stable and Labile groups may have been found in different conditions. Participants of both groups showed considerable individual differences within a limited range of possible forgetting scores. This variability resulted in a large amount of error variance in between-

group comparisons, and in this small sample size, group means may have more so reflected the specific individuals randomly assigned to the groups rather than an effect of the reactivation manipulation. Further, perhaps due to the small number of items within each block, the actual range in the directed forgetting scores was small. If participants did not show equal forgetting of each block, they generally forgot only one or two more syllable pairs from one compared to the other. To find group differences using these specific tests of forgetting, a larger sample size would likely be necessary. Alternatively, the sensitivity of this paradigm could be improved by adding more items to the syllable pair lists or altering the design to allow for a within-subjects memory reactivation manipulation instead of the between subjects manipulation used in the current research. Finally, the syllable pair memory task could be replaced with a type of picture-location task shown to be sensitive in similar sleep studies (Oudiette et al., 2013; van Dongen et al., 2012). It may be the case that individuals naturally vary much more in their ability to remember syllable pairs than in their ability to remember picture and location combinations.

Again, it should be noted that although no group differences were found in forgetting scores in between-group comparisons, interaction effects of group membership with NREM delta and gamma power and REM theta power suggest that the memory reactivation treatment was effective in allowing differential reprocessing of TBR and TBF memories in the Labile group. While causal links between sleep and the forgetting of memories cannot be directly determined through these correlations, the fact that these results support hypotheses built from previous experimental research does lend favourably to the more causal interpretations proposed.

The hypothesis that sleep acts to selectively reconsolidate memories of future relevance relies on the assumption that reconsolidation processes take place, predominately at least, during sleep. This study did not test this assumption directly as all participants slept on each night of the study. Reconsolidation could have taken place during wakefulness in the time between reactivation and sleep onset. However, previous research in rodents has suggested that although REM sleep is not necessary for memory reconsolidation to take place, sleep in general, compared to wakefulness, is critical in the reconsolidation of reward memories (Shi et al., 2011; Tian et al., 2009). These findings are consistent with negative correlations found between NREM delta and gamma activity and TBR forgetting in the Labile group. Further, these findings are also consistent with the proposed hypothesis that reconsolidation takes place predominately during NREM sleep. Further research should be conducted to test this hypothesis experimentally for human declarative memories.

A final point to consider is the nature of the directed forgetting instructions used in the current study. The participants were not explicitly asked to try to actively forget the TBF material they had already learned. In what could be considered directed remembering rather than directed forgetting instructions, participants were told they should try to remember Block B and that there was no need to remember Block A (Appendix F). Thus, the TBF material in the current study did not address the fate of memories intended to be forgotten. This modification to typical directed forgetting instructions was made because, in pilot testing, many participants reported suspicion upon hearing the explicit instruction to “forget.” Even with this modification, approximately two thirds of the participants in both groups reported on the feedback

questionnaire (Appendix B) that they either suspected or knew that Block A would be on the test. Because of this reported suspicion, an interpretation of the results that does not require the role of future relevance is needed. It may be the case that memories for TBF material were relatively weaker prior to the instructions regardless of suspicion. Practice effects, retroactive interference at learning, and increased attention after the TBR cue could all cause this as the TBF block was always presented before TBR block. Even if this was the case for some or all of the participants, the interpretation of the results would be similar. Relatively stronger memories, if labile, may receive preferential reconsolidation during NREM sleep while relatively weaker memories, both stable and labile, are further weakened through synaptic downscaling during REM sleep.

Future research could further clarify this hypothesis of selective reconsolidation in a number of ways. First, because a number of potentially interesting correlations and effects only reached marginal significance and only a few would have met conservative values of alpha in this study, direct and conceptual replications of these effects are needed to increase the confidence that can be placed in these findings. Future research could also explore more explicit forget instructions that could be used to directly examine the fate of memories intended to be forgotten instead of those that are simply not cued TBR. This change would be necessary to determine if sleep plays an active inhibitory role to selectively forget memories. This future research should be conducted with a large enough sample size to allow the exclusion of participants who were suspicious that the TBF material would appear on the test. Alternatively, selectively in memory reconsolidation could be further examined by using similar instructions to those given in the recent research from Oudiette et al. (2013) that replaced standard directed forgetting

instructions by increasing the incentive to remember some items over others. If participants are informed during a memory reactivating reminder session that some items are worth more money than others during the test, selectivity in memory reconsolidation could be addressed without the confound of having some participants believe in the TBF instructions while others are more suspicious.

Implications

Whether driven by relative strength, motivation, or directed forgetting, evidence of selective remembering and forgetting of material has implications in clinical areas of research. Although not examined here, REM sleep has been implicated in both the processing of emotional memories as well as post-traumatic stress disorder (Mellman, Pigeon, Nowell, & Nolan, 2007; Walker, 2010). A recent study comparing power spectral analysis sleep EEG between trauma-exposed individuals with and without post-traumatic stress disorder found that those with resilience had greater theta power during REM sleep compared to those who had developed post-traumatic stress disorder (Cowdin, Kobayashi, & Mellman, 2014). An understanding of the role of theta activity during REM sleep in forgetting may be essential in further understanding and treating this disorder. Further, greater knowledge of reconsolidation and forgetting processes in general may be of benefit for the treatment of other conditions in which problem memories may play a role. As suggested by Shi et al. (2011), preventing the reconsolidation of drug-related reward memories through sleep deprivation could potentially serve as an ancillary method to prevent relapse in those suffering from addiction.

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Table 1

Definitions and Associated Predictions of Forgetting Scores and Power Spectral Variables of Interest

Variable	Definition	Prediction
TBR forgetting	Errors made in first Syllable Trial of Block B at test - Errors made in last Syllable Trial of Block B at learning.	Negative correlation with NREM delta power in the Labile group.
TBF forgetting	Errors made in first Syllable Trial of Block A at test - Errors made in last Syllable Trial of Block A at learning.	Positive correlation with REM theta power in the Labile group.
Total forgetting	TBF forgetting + TBF forgetting; higher scores reflect greater forgetting regardless of TBR and TBF cues.	
Directed forgetting	TBF forgetting – TBR forgetting; higher scores reflect more forgetting of TBF items and less forgetting of TBR items.	Greater in the Labile group than the Stable group, and positively correlated with NREM fast sigma power in the Labile group.
NREM delta power	1 – 4 Hz EEG power (μV^2/Hz) during NREM sleep measured frontally in Early Stage 2 sleep, Slow-wave sleep, and Late Stage 2 sleep.	Negative correlation with TBR forgetting in the Labile group.
NREM fast sigma power	14 – 16 Hz EEG power (μV^2/Hz) during NREM sleep measured frontally in Early Stage 2 sleep, Slow-wave sleep, and Late Stage 2 sleep.	Positive correlation with directed forgetting in the Labile group.
NREM gamma power	30 – 70 Hz EEG power (μV^2/Hz) during NREM sleep measured frontally in Early Stage 2 sleep, Slow-wave sleep, and Late Stage 2 sleep.	
REM theta power	4 – 8 Hz EEG power (μV^2/Hz) during REM sleep measured centrally.	Positive correlation with TBF forgetting in the Labile group.

Note. TBR = to-be-remembered; TBF = to-be-forgotten; NREM = non-rapid eye movement; EEG = electroencephalographic; REM = rapid eye movement.

Table 2

Comparisons of Stable and Labile Group Means for Measures of Night 2 Sleep Architecture

	Stable (n = 16)		Labile (n = 15)			
Measure	*M*	*SD*	*M*	*SD*	*t* (29)	*p*
Wake time	27.28	13.86	31.87	22.35	0.69	.495
Total sleep time	450.19	16.57	449.13	25.03	0.14	.890
Sleep efficiency	93.22	3.36	93.19	4.72	0.02	.982
Stage 1 time	26.63	8.40	32.37	10.54	1.68	.103
Stage 2 time	237.06	38.83	232.97	28.87	0.33	.740
SWS time	103.22	41.37	80.87	32.18	1.67	.105
REM time	84.19	19.79	103.83	24.25	2.48	**.019**
Stage 1 percentage	5.93	1.89	7.28	2.59	1.66	.108
Stage 2 percentage	52.44	8.56	51.72	6.24	0.26	.793
SWS percentage	22.93	9.30	17.98	7.24	1.64	.111
REM percentage	18.71	4.36	23.02	4.86	2.60	**.014**
Stage 1 latency[a]	13.03	11.82	13.53	10.97	0.41	.688
Stage 2 latency[a]	17.00	13.12	19.40	16.17	0.64	.531
SWS latency[a]	28.66	12.72	29.53	16.02	0.05	.957
REM latency[a]	121.91	41.15	89.73	23.78	2.49	**.019**

Note. Significance values of $p < .05$ are in boldface. SWS = slow-wave sleep; REM = rapid eye movement. Time and latency variables measured in minutes. Percentage variables measured as a percentage of total sleep time.

[a] Positively skewed data received log10 transformation prior to *t*-test. Reported means and standard deviations are of the untransformed data.

Table 3

Comparisons of Stable and Labile Group Means for Night 2 EEG Band Power and Night-to-Night Reliability for the Power Spectral Variables of Interest

	Stable (n = 16)			Labile (n = 15)				
Measure	*M*	*SD*	*ICC* [95% CI]	*M*	*SD*	*ICC* [95% CI]	*t* (29)	*p*
Early Stage 2								
Delta	2.02	0.21	.34 [-.16, .70]	1.87	0.17	.69 [.31, .88]	2.10	**.045**
Fast sigma	0.52	0.34	.75 [.42, .90]	0.43	0.38	.95 [.87, .98]	0.71	.487
Gamma	-1.46	0.17	-.13 [-.56, .38]	-1.52	0.18	.03 [-.47, .51]	0.96	.344
SWS								
Delta	2.57	0.23	.89 [.72, .96]	2.53	0.17	.82 [.54, .93]	0.61	.550
Fast sigma	0.31	0.34	.91 [.77, .97]	0.20	0.35	.92 [.79, .97]	0.88	.389
Gamma	-1.64	0.21	.59 [.16, .83]	-1.56	0.31	.27 [-.25, .67]	-0.90	.375
REM								
Theta	0.84	0.20	.82 [.58, .93]	0.64	0.18	.90 [.74, .97]	3.00	**.005**
Late Stage 2								
Delta	1.99	0.21	.44 [-.04, .76]	1.75	0.26	.59 [.15, .84]	2.91	**.007**
Fast sigma	0.37	0.41	.42 [-.07, .75]	0.32	0.38	.89 [.72, .96]	0.35	.730
Gamma	-1.57	0.26	.62 [.21, .85]	-1.60	0.21	.12 [-.39, .58]	0.26	.793

Note. Significance values of $p < .05$ are in boldface. *ICC* = intraclass correlation coefficient; SWS = slow-wave sleep; REM = rapid eye movement. Electroencephalographic band power variables have been log10 transformed at each scalp site. Delta and gamma power measured frontally as an average over F3, Fz, and F4. Fast sigma power measured parietally as an average over P3, Pz, and P4. Theta power measured centrally as an average over C3, Cz, and C4. *ICC* reflects the reliability between Night 1 and Night 2 sleep for each power spectral variable of interest.

Table 4

Stable and Labile Group Means for Each Forgetting Score

	Stable (n = 16)			Labile (n = 15)		
Forgetting	M	SD	95% CI	M	SD	95% CI
TBR	1.44	1.26	[0.76, 2.11]	1.87	1.19	[1.21, 2.52]
TBF	1.88	1.26	[1.20, 2.55]	1.73	1.16	[1.09, 2.38]
Total	3.31	2.21	[2.13, 4.49]	3.60	1.92	[2.60, 4.60]
Directed	0.44	1.21	[-0.21, 1.08]	-0.13	1.36	[-0.88, 0.62]

Note. TBR = to-be-remembered; TBF = to-be-forgotten.

Table 5

Correlations Between Forgetting Scores and Power Spectral Variables of Interest in the Stable and Labile Groups

	Stable (n = 16)				Labile (n = 15)			
Measure	TBR	TBF	Total	Directed	TBR	TBF	Total	Directed
Early Stage 2								
Delta	.28	.02	.17	-.28	**-.61***	-.16	-.48^{+}	.40
Fast sigma	-.09	-.03	-.07	-.06	-.27	.44	.10	**.61***
Gamma	.23	.24	.27	.01	**-.72****	-.01	-.45^{+}	**.62***
SWS								
Delta	.05	.48^{+}	.30	.44^{+}	-.41	-.06	-.29	.30
Fast sigma	-.08	-.05	-.07	.04	-.34	.34	.01	**.61***
Gamma	.02	-.01	.01	-.03	-.41	-.05	-.28	.31
REM								
Theta	.48^{+}	.40	**.50***	-.09	-.48^{+}	.45^{+}	-.02	**.81*****
Late Stage 2								
Delta	.22	.06	.16	-.17	-.44	.02	-.26	.40
Fast sigma	.16	.19	.20	.04	-.33	.20	-.09	.46^{+}
Gamma	.17	.11	.16	-.06	**-.63***	-.09	-.45^{+}	.48^{+}

Note. Coefficients with significance values of $p < .05$ are in boldface. TBR = to-be-remembered; TBF = to-be-forgotten; SWS = slow-wave sleep; REM = rapid eye movement.

*$p < .05$. **$p < .01$. ***$p < .001$.

$^{+}p < .10$

Table 6

Correlations Between Forgetting Scores and Percentage of Total Sleep Time Spent in Each Sleep Stage in the Stable and Labile Groups

	Stable (n = 16)				Labile (n = 15)			
Percentage of TST	TBR	TBF	Total	Directed	TBR	TBF	Total	Directed
Stage 1	-.38	-.49^{+}	**-.50***	-.11	.00	-.23	-.14	-.20
Stage 2	.03	.16	.11	.13	.42	.02	.27	-.36
SWS	.03	-.00	.02	-.03	**-.54***	-.19	-.45^{+}	.30
REM	.04	-.09	-.03	-.13	.26	.39	.39	.11

Note. Coefficients with significance values of $p < .05$ are in boldface. TBR = to-be-remembered; TBF = to-be-forgotten; TST = total sleep time; SWS = slow-wave sleep; REM = rapid eye movement.

*$p < .05$.

$^{+}p < .10$

Table 7

Intercorrelations Between the Power Spectral Variables used as Predictors of Forgetting in the Stable and Labile Groups

Measure	1	2	3	4
1. Early Stage 2 delta	-	.12	.36	.25
2. Early Stage 2 fast sigma	.37	-	**.54***	**.83****
3. Early Stage 2 gamma	.25	.16	-	**.68****
4. REM theta	**.66****	.09	**.75****	-

Note. Coefficients with significance values of $p < .05$ are in boldface. Intercorrelations for the Stable group are presented below the diagonal, and intercorrelations for the Labile group are presented above the diagonal.
*$p < .05$. **$p < .01$. ***$p < .001$.

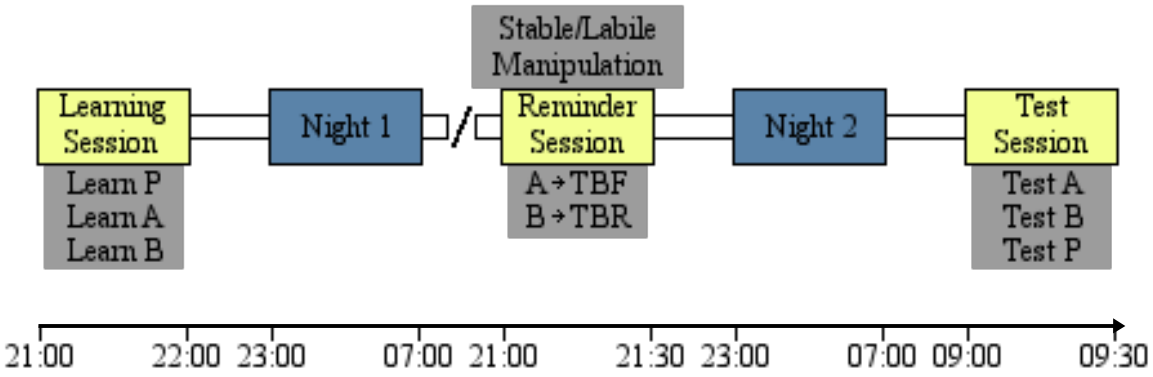

Figure 1. Three-day experimental protocol. Three blocks of syllable pairs (P, A, and B) were learned during the learning session. During the reminder session, Block A was cued to-be-forgotten (TBF) and Block B was cued to-be-remembered (TBR). The Stable / Labile manipulation created a Stable memory group and a Labile memory group using two different types of reminders. Both groups were tested on all blocks during the test session. Night 1 and Night 2 sleeps were polysomnographically recorded.

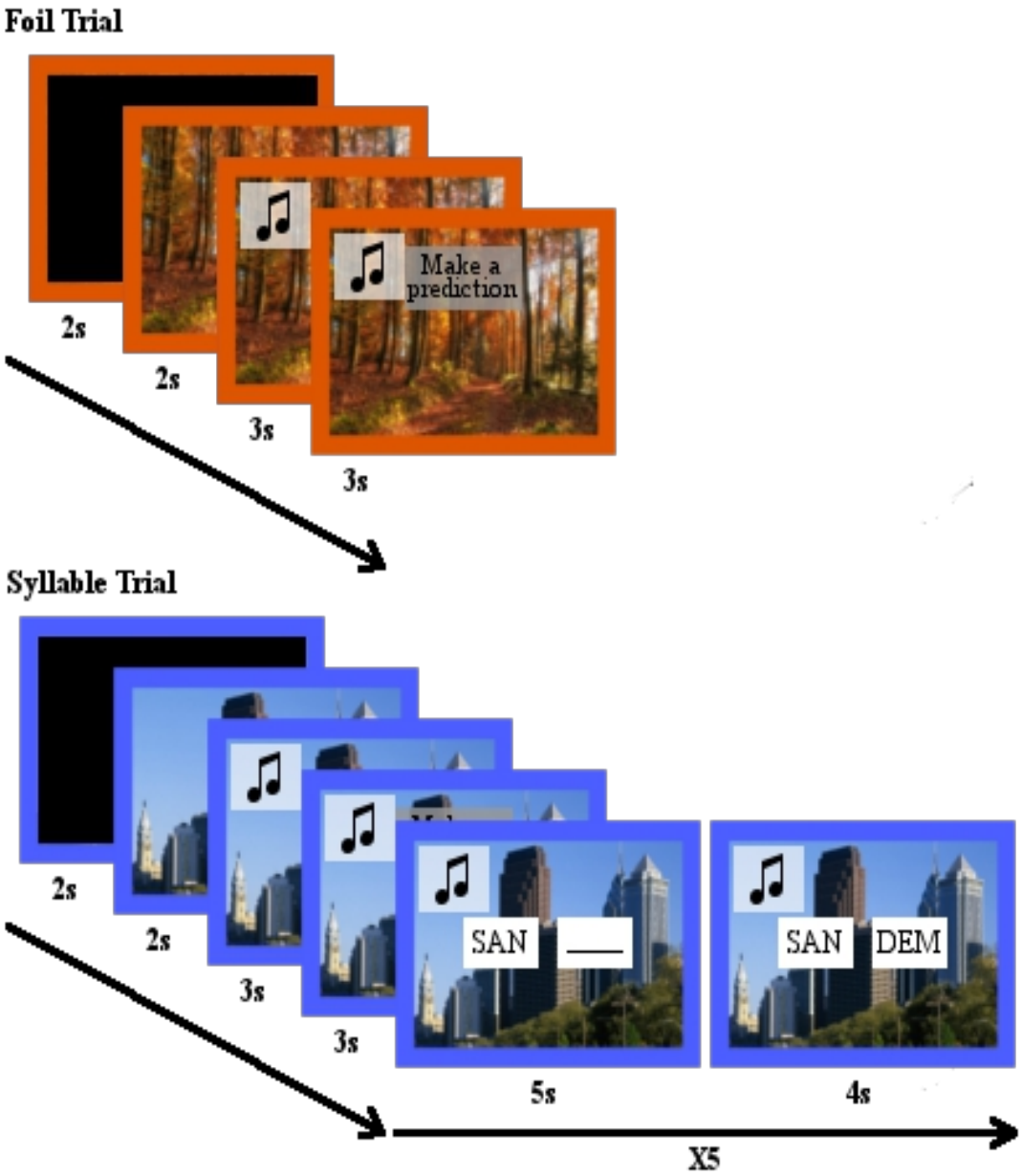

Figure 2. Foil Trials and Syllable Trials from the learning and test sessions. Both types of trial contained a ten-second context period in which a border colour, background image, and piece of music were presented, and participants were asked to make a prediction in each about whether syllables would also be presented. Within each block, Syllable Trials always contained all three matching elements of a specific context set while Foil Trials contained at least one element of the opposite context set for that block. Participants were given five seconds to respond to each of the five cue syllables before its pair was shown.

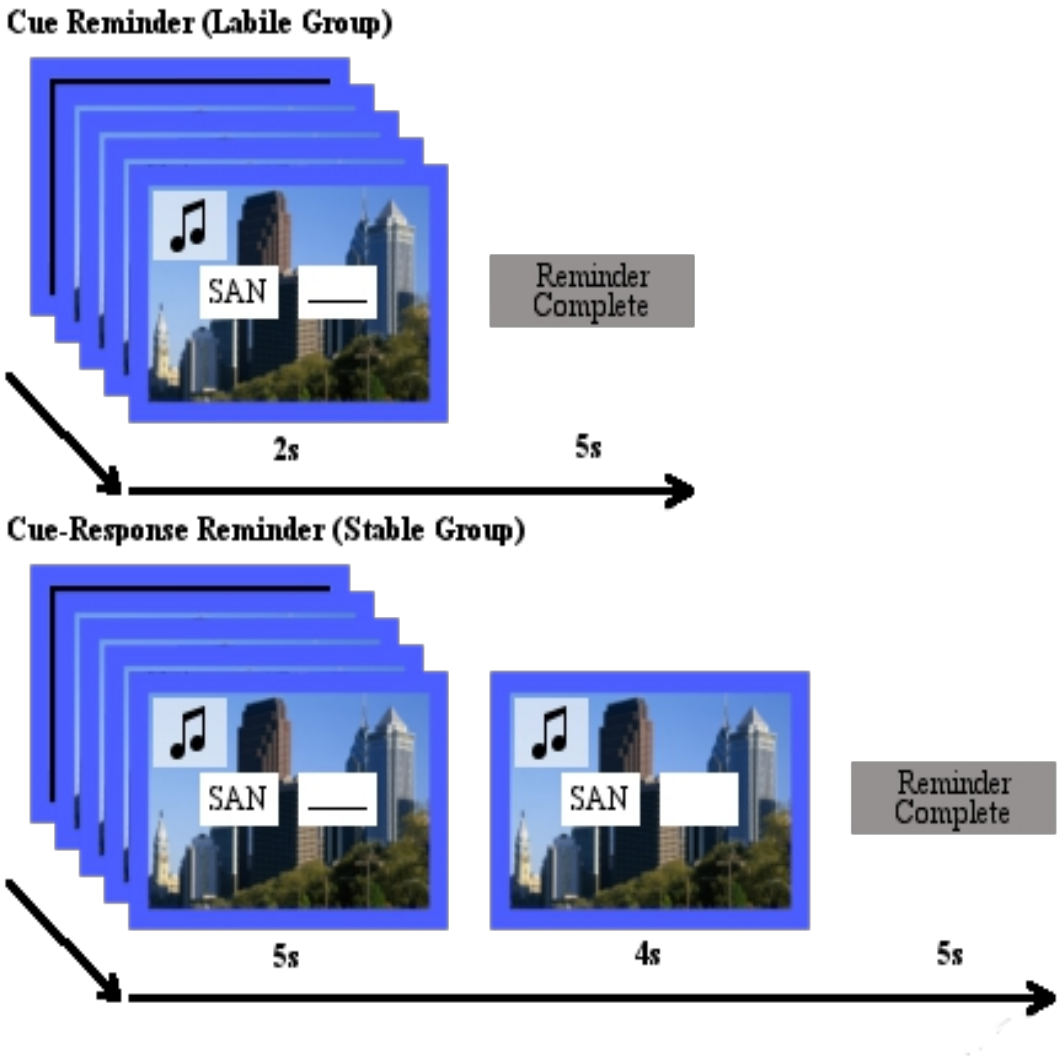

Figure 3. Cue-Only Reminders and Cue-Response Reminders from the reminder session. The Labile group was given Cue-Only Reminders in which the context period from the Syllable Trials was followed by a single cue syllable and then terminated before the participant could respond. The Cue-Response Reminders were given to the Stable group and allowed participants to respond to the cue syllable shown. Only Cue-Only Reminders, not Cue-Response Reminders, have been shown to trigger memory reactivation.

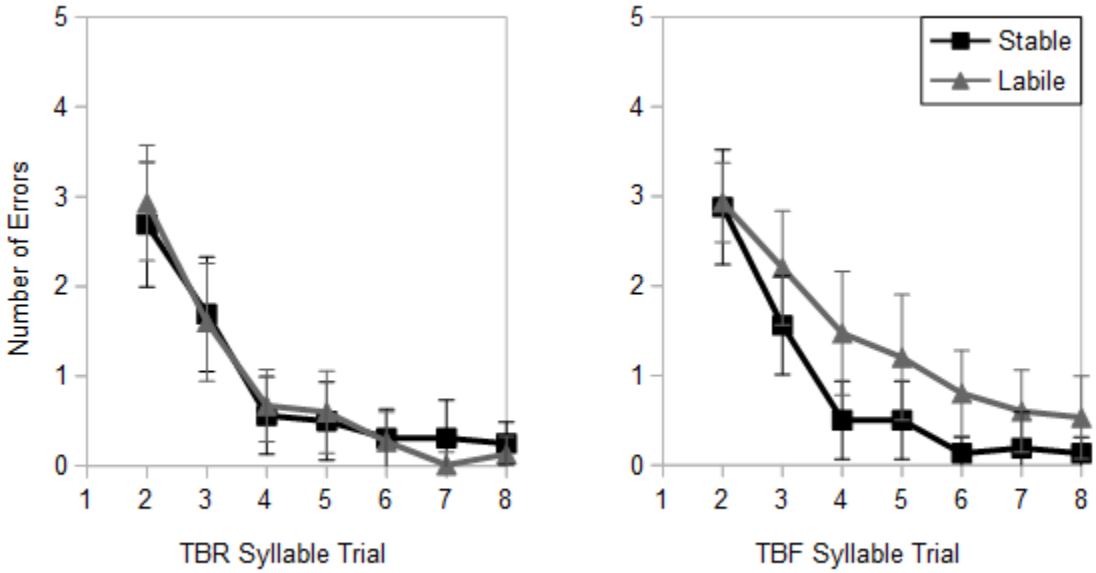

Figure 4. Trial-by-trial learning performance for the syllable pairs in the to-be-remembered (TBR) and to-be-forgotten (TBF) blocks in the Stable and Labile groups. Error bars represent 95% confidence intervals around means. No responses were collected from the first Syllable Trial in each block.

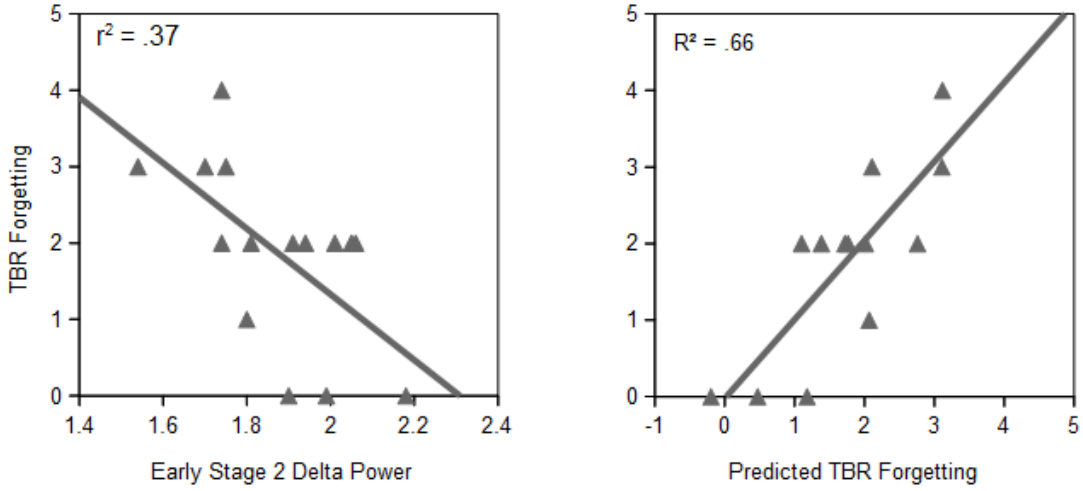

Figure 5. Prediction of to-be-remembered (TBR) forgetting scores in the Labile group (n = 17). (A) Correlation between Early Stage 2 delta power and TBR forgetting in the Labile group (r = -.61, p < .05). (B) Scatter plot of to-be-remembered (TBR) forgetting scores versus the predicted TBR forgetting scores in the Labile group based on the regression model using Early Stage 2 gamma power (β = -.57, p = .008) and Early Stage 2 delta power (β = -.41, p = .044) as predictors. Average power spectral variables have been log10 transformed at each scalp site. Delta and gamma power measured frontally as an average over F3, Fz, and F4.

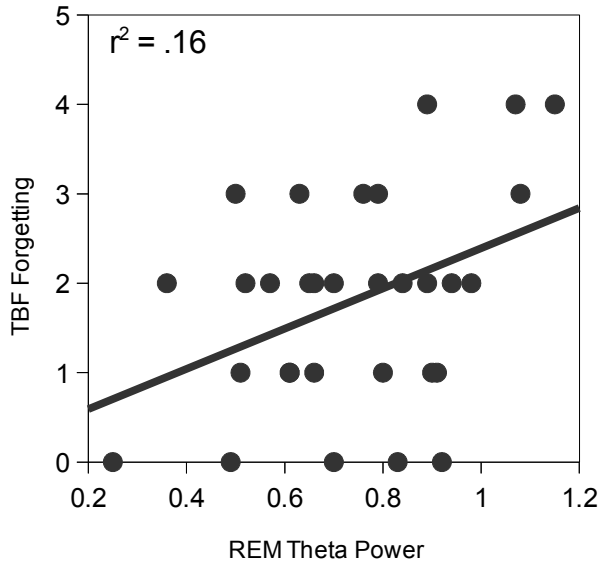

Figure 6. Correlation between rapid eye movement (REM) theta power and to-be-forgotten (TBF) forgetting scores in the full sample (n = 31; r = .40, p < .05). Average power spectral variables have been log10 transformed at each scalp site. Theta power measured centrally as an average over C3, Cz, and C4.

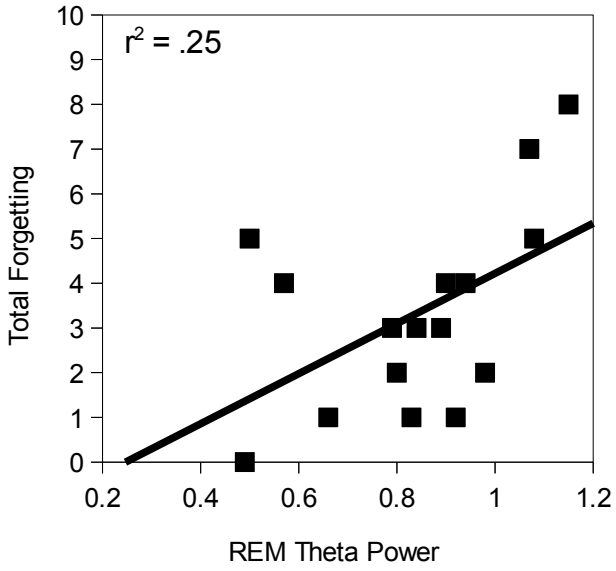

Figure 7. Correlation between rapid eye movement (REM) theta power and total forgetting scores in the Stable group (n = 15; $r = .50$, $p < .05$). Average power spectral variables have been log10 transformed at each scalp site. Theta power measured centrally as an average over C3, Cz, and C4.

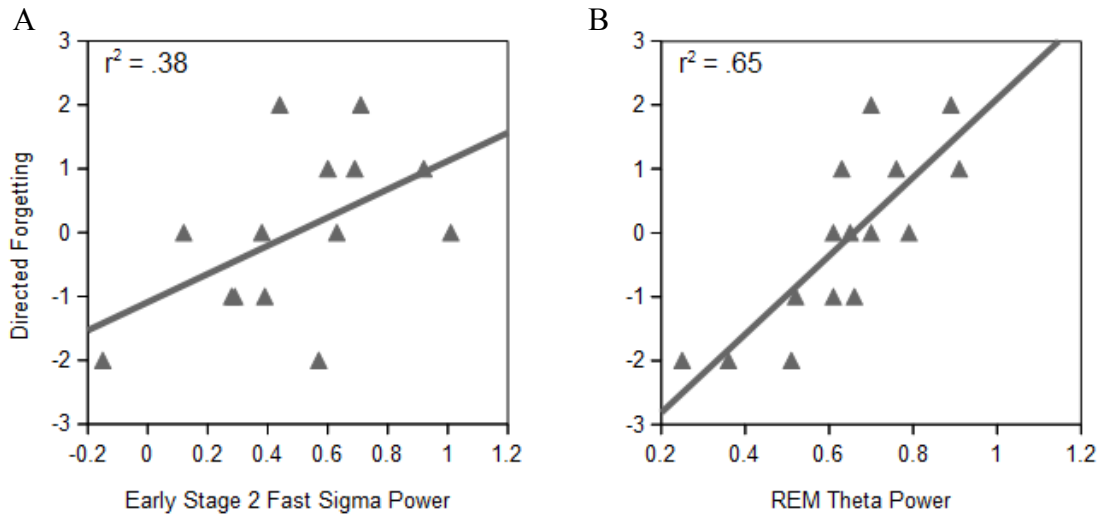

Figure 8. Prediction of directed forgetting scores in the Labile group (n = 17). (A) Correlation between Early Stage 2 fast sigma power and directed forgetting in the Labile group ($r = .61$, $p < .05$). (B) Correlation between rapid eye movement (REM) theta power and directed forgetting in the Labile group ($r = .81$, $p < .001$). Average power spectral variables have been log10 transformed at each scalp site. Fast sigma power measured parietally as an average over P3, Pz, and P4. Theta power measured centrally as an average over C3, Cz, and C4.

Appendix A

Telephone Interview

Date: **Time:** **ID CODE:**

I. DESCRIBE STUDY:

We are interested studying the relationship between sleep and memory. In this study, you will be asked to perform a three part memory study over three days and have your sleep recorded with electrodes in the Brock University Sleep Laboratory over the two intervening nights.

Here are the details of what would be expected of you:

1. First, we will get some information from you on the phone today to see if you meet our basic criteria for participation. This interview will include simple demographic questions as well as questions about your sleep behaviours, health, and substance use.
2. Next, we will ask you to complete some on-line screening questionnaires to see if you are eligible (this will take about 30 minutes). You will be asked questions about physical and mental health, sleep habits, substance use (e.g., alcohol, caffeine, medications, and drugs), and personality.

3. **If you meet the criteria in these questionnaires,** we will then ask you to attend the over night sleep screening session where you will tour the Sleep Lab, complete more questionnaires, and sleep from 11PM – 7AM while sensors are used to monitor your brain activity, respiration, muscle movement, and heart rate. You will also be given a full consent form when you enter the lab for this session – this provides more details about the study – and you can decide whether or not you are interested in full participation.
4. **If at any point during the screening questionnaires or overnight sleep screening session you are deemed ineligible to participate in the study**, you will be informed of the reason and all the data collected from you to that point will be destroyed.

5. **For the main part of the study**, you will be scheduled for two overnight sessions in the Sleep Laboratory to take part in the memory task and have your sleep recorded. The first session will begin at 9PM one evening with the memory task lasting approximately 50 minutes. After the task, the electrodes for recording your sleep will be applied and you will be asked to sleep in the laboratory from 11PM – 7AM once again. You will be allowed to leave the lab in the morning and go about your day.
6. You will be asked to return to the lab that evening at 9PM for the second portion of the memory task taking approximately 20 – 30 minutes. After this, electrodes will be applied and you will again be asked to sleep from 11PM – 7AM.
7. The final portion of the memory task will begin at 9AM that morning. Again, this will take approximately 30 minutes. This test will conclude your participation in the study.

For completing participation of the main part of the study, you will be given an additional $50 honorarium, bringing the total compensation value to $75 or $50 and course credit.

Are you interested? [yes] – OK, I have a few questions for you to make sure you are suitable for the study. You may decline to answer any of these questions; however, this may exclude you from this study.

II. INCLUSION CRITERIA:

What nights would you be free to participate from about 9PM to 9:30AM the next morning (indicate schedule):

_____

Age (18-30): _____

Weight (indicate kg or lbs): _____

Gender: M / F_

Smoker: Y / N [no]

Handedness: R / L [right] _____

How many caffeinated drinks do you typically have in a day [min - moderate, <3]:_____

Is English your first language (if not, did you learn before age 8): _____

Do you have any difficulties with hearing? [no, in both ears]: _____

III. Questions on SLEEP:

1. Do you consider yourself to be a good sleeper? [yes]:_____
2. What are your usual sleeping times [approx 23:00-07:00]:_____
3. How does this change on weekends? [sleeping-in a bit is OK]_____
4. Do you have difficulty *falling* asleep at night [no]: _____
5. Do you *wake up* often during the night and are unable to return to sleep [no]: __ ____
6. Have you ever been diagnosed with a Sleep Disorder [no]:_____
7. Have you ever been told you kick your legs all-night long or stop breathing during the night? [no] _____
8. Do you experience restless legs or a "creepy crawling" sensation before bed each night? [no]: _____
9. Would you describe yourself as *excessively* tired during the day [no]: _____
10. Do you currently work shift work [no]; any history of shiftwork?_____
11. Do you take daytime naps? Y / N
    How frequently (# / week) _____ Duration for each _____
12. Have you ever pulled an all-nighter? How often/how many times etc. _____

IV. Questions on HEALTH:

1. Are you presently in good health [yes]: _____
2. Taking any medications [no]: _____
3. Any history of depression, anxiety or schizophrenia [no]:_____ _____
4. Any history of head injury (e.g., car accident, stroke, loss of consciousness), epilepsy, or other neurological condition [no]: _____
5. Any history of chronic pain [no] _____
6. Any history of heart disease or cardiac abnormalities [no]: _____

Appendix B

Feedback Questionnaire

FEEDBACK QUESTIONS **ID: _____**

- Circle the most appropriate answer to fill in the blank
  - I _____ I would be tested on the Block A material.
    didn't know suspected knew
  - I _____ I would be tested on the Block P (Practice Block) material.
    didn't know suspected knew
- If you knew or suspected that the material from either of these blocks would be on the test, please explain your reasoning below.

_____

_____

_____

- What strategy (if any) did you use to remember the syllable pairs?

_____

_____

_____

☐ Check this box to indicate that you have read and understood the Letter of Appreciation / Debriefing.

☐ Check this box if you still consent to use of the data collected from you in this study as outlined in the original consent form. All personal data will be kept strictly confidential, and all data collected from the memory task and questionnaires are tied only to the ID code assigned to you.

Appendix C

Syllable Pair Lists

Demonstration List

Cue Syllable	Pair Syllable
LAV	PER
GEN	LIF
MUR	DOM
GOG	NEM
PUM	HOB

List X

Cue Syllable	Pair Syllable
PED	VEN
HIN	MAR
JUM	NEM
FEN	VIS
DER	NOM

List Y

Cue Syllable	Pair Syllable
TER	LUM
SUS	GIB
MEL	JER
SAN	DEM
KER	BON

List Z

Cue Syllable	Pair Syllable
ZEL	NER
WAN	PEL
CEN	DEP
FAM	LUX
HAP	TEM

Appendix D

Pilot Results: Difficulty of Syllable Pair Lists

The three lists of syllable pairs are identified as List X, List Y, and List Z (Appendix C). Analyses were conducted on a pilot of 21 participants to determine whether the syllable pair lists differed in difficulty of cued-recall. As in the main experiment, the lists in the pilot study were randomly assigned to the three blocks of the memory task. Although not perfectly counterbalanced, random assignment resulted in similar distributions of the lists among the three blocks (Table D1). This was important as an order effect was found showing that participants made significantly more errors learning the first block of the task (Block P; $M = 15.14$, $SD = 8.32$) compared to the second block (Block A; $M = 6.05$, $SD = 4.13$; $t\ (20) = 5.39$, $p < .001$) and third block (Block B; $M = 5.67$, $SD = 5.10$; $t\ (20) = 5.24$, $p < .001$). The number of errors made in learning did not differ significantly between the second and third blocks ($t\ (20) = 0.46$, $p = .651$).

In learning of the three syllable pair lists, participants made an average of 7.62 errors ($SD = 6.34$) on List X, an average of 11.14 errors ($SD = 9.50$) on List Y, and an average of 8.10 errors ($SD = 5.89$) on List Z. A repeated measures ANOVA was conducted to investigate the number of errors made across these three lists regardless of the order in which they were delivered. The effect of list on the number of errors made was found to be non-significant ($F\ (2, 40) = 1.53$, $p = .229$), indicating that the number of errors made during learning did not differ significantly across lists X, Y and Z.

To further investigate the difficulty of the syllable pair lists, the number of errors made in each list's last Syllable Trial of the learning session were examined. Performance

on these trials was high with 76%, 62%, and 76% of participants not making a single error in the last Syllable Trials of List X, Y, and Z, respectively. Because of these highly skewed distributions, the non-parametric Friedman test was conducted to test whether the number of errors made in these trials varied across the lists. Again it was found that the number of errors made did not vary significantly across lists X, Y, and Z (χ^2 (2) = 1.68, p = .431)

Although still non-significant, it can be noted in both the full learning session and its last Syllable Trials that participants were more prone to make errors in learning List Y. This tendency is thought to be the result of the previously noted order effect as List Y was most often assigned to the first presented Block P. Thus, it was concluded from this pilot that lists X, Y, and Z did not differ in difficulty of cued-recall.

Table D1

Frequency of the Placement of Each List in the Three Blocks of the Memory Task

List	Block P	Block A	Block B
X	5	9	7
Y	9	6	6
Z	7	6	8

Note. For each participant (N = 21), Lists X, Y, and Z were randomly assigned to Blocks P, A, and B of the memory task. The delivery order of the blocks was always Block P first, Block A second, and Block B third with five minutes separating each block.

Appendix E

Context Set Pairs

Demonstration Pair

Traditional Japanese Music

Rock Music

Pair X

Classical Piano Music

Smooth Jazz Music

Pair Y

Bluegrass Music

Folk Violin Music

Pair Z

Latin Jazz Music

Traditional Arabic Music

Appendix F

Memory Task Instructions

Learning Session

In this session, we ask that you try to learn the material presented to you. There will be 3 separate blocks of this task, and each will have different material for you to learn. There will be 24 short trials in each block that will be used to teach you this material.

All of these trials will contain a colour border, an image, and a short piece of music. In addition, some of the trials will feature a set of 5 syllable pairs that we would like you to memorize. On these trials, the syllable pairs will be presented to you on the screen one at a time after 6 seconds of the music.

One of your tasks will be to determine which trials will contain these syllable pairs and which will not. You should make your prediction about whether or not the trial will feature syllables by pressing the appropriate key on the keyboard when prompted. Press the Right Arrow Key to predict that the syllables will be shown, and press the Left Arrow Key to predict that they will not be shown.

Your other task will be to memorize these syllable pairs. The syllables presented on the left are the cue syllables and will always being given to you. The first time you see a syllable set, the paired syllables will be presented on the right after a short delay. Try to remember these pairs.

On all other syllable trials you will be shown a response box to the right of the cue syllable for 5 seconds. Type the cue syllable's pair in this box. After 5 seconds, the response entered into the box is recorded, and the correct answer is shown. This procedure is repeated for each syllable pair until all 5 in the set are shown.

The first of the 3 blocks, Block P, is a practice block. The following 2 blocks, Block A and Block B, will teach you the material needed for the memory test that will be given on Day 3. Only one of these blocks will appear on the test, but you won't know which block it will be until Day 3. Therefore, you should try your best to learn both of them at this time.

Directed Forgetting Instructions (Within the Reminder Session)

As you already know, the memory test tomorrow will only test you on one of the blocks of syllable pairs you learned yesterday. You were also informed that you wouldn't get to know which block will be on the test until you see the test.

However, we are using this experiment to study the efficiency and capacity of memory, and we do this by telling one group of participants which syllable pairs will be on the test and which will not. Random group selection has placed your ID, ___ into this group.

You will be tested on your memory of the Block B syllable pairs. This is the block you will be reminded of in the next two reminder trials. You should do your best to remember

these syllable pairs.

You will not be tested on the Block A syllable pairs. This is the block you were just reminded of in the previous two reminder trials. There is no need to remember these syllable pairs.

Remember: some participants will not be given this extra information about the test. Because we are interested in learning whether memory performance differs when participants know which pairs will be on the test and which will not, we ask that you please keep these instructions a secret from any other participants you may talk to.

Appendix G

Letter of Information / Consent Form

LETTER OF INFORMATION / CONSENT FORM
BROCK UNIVERSITY SLEEP RESEARCH LABORATORY
PSYCHOLOGY DEPARTMENT

Title of Study: Investigating the Relationship between Sleep and Context Memories
Principal Investigators: Kimberly A. Cote, Ph.D., & Kevin MacDonald, MA student

This letter of information/consent form is provided to you for your information on the website of the Brock University Sleep Research Laboratory. You should carefully read this form to understand all aspects of participation in the research study prior to completing the on-line eligibility questionnaires. By completing the on-line questionnaires, you are acknowledging that you have read and understood this form and you are providing consent to participate in the full research study. You will be asked to sign this form and be given a copy during your next visit to the Sleep Laboratory.

If you have questions about the details of this study prior to completing the on-line questionnaires, please call the Sleep Laboratory at 905-688-5550, ext.3795.

Name of Participant:_____
(*Please print your name in the space above - on the paper copy only*)

PART A: INFORMATION ABOUT THE STUDY

I understand that I am being invited to participate in a research study investigating the relationship between sleep and memory performance. This study will be of benefit to the scientific community and contribute to the understanding of the complexities of memory and its relationship with different aspects of brain function during sleep.

I understand that participation has five phases:

1) On-line questionnaires including questions about personality, sleep, health, and substance use (e.g., alcohol, caffeine, medications, and drug use).
2) An overnight orientation session and sleep screening in the Sleep Lab, where I will tour the facilities, complete more questionnaires, and sleep from 11PM to 7AM while aspects of my sleep are recorded through a number of electrodes and sensors placed on my head, face, and body.
3) An overnight session that begins with learning the material for the memory task (40 – 50 minutes) at 9PM and ends once all electrodes are taken off after a second

night of recorded sleep in the laboratory from 11PM to 7AM.

4) A second, consecutive overnight session beginning again at 9PM with the second portion of the memory task (20 – 30 minutes) followed by another night of recorded sleep in the laboratory from 11PM to 7AM.
5) A memory test (30 – 40 minutes) where I will be asked to recall the previously learned material at 9AM following the second night of recorded sleep in the experimental procedure. I will be asked to remain in the laboratory for the two hours between the 7AM wake-up time and the 9AM recall test.

On-line screening questionnaires will ask questions about physical and mental health, sleep habits, and personality. If responses or scores on these questionnaires raise concerns about mental health, you will be contacted and given information about available resources for counselling at the Student Development Center.

For the sleep screening night you will be asked to sleep in the sleep laboratory with several electrodes and sensors applied to your scalp, face, and body including: 5 electrodes placed on the scalp to record brain activity, 2 electrodes placed beside the eyes to record eye movement, 2 electrodes placed under the chin to record muscle tension, 2 electrodes immediately below the clavicle bone to record heart rate, 2 electrodes on each leg to measure leg movement, and 2 respiration belts and a sensor under your nose to monitor your breathing. For the two nights of the experiment, the 4 leg electrodes and the breathing sensors will not be used, and an additional 8 scalp electrodes will be applied. Electrode application is a procedure which takes approximately 30 minutes to complete. Electrode sites will be quickly cleaned using an alcohol swab and a mildly abrasive conductive gel. Electrodes will then be applied to the skin with a conductive paste and tape. All electrodes will be removed after the 7AM wake-up time, and the gel and paste can be washed off easily in the laboratory with soap and warm water.

I understand that there will be no compensation for completion of the pre-study on-line screening questionnaire described above if I am deemed ineligible to participate or if I choose not to participate in the orientation and screening night (phase 2 above). If I withdraw or I am withdrawn by experimenters during the screening process, my information will be destroyed. I understand that $25 or course credit will be given as an honorarium for the completion of the overnight sleep screening regardless of whether or not I am found eligible for the remainder for the study.

I understand that on the day of my sleep screening and for the duration of the main study (phases 2 – 5), I must:

- be in bed between 11PM and 7am (getting out of bed at 07AM sharp)
- drink no alcohol
- drink no caffeine
- take no naps
- obtain no vigorous exercise

On the three evenings I am required to go to the Sleep Laboratory (phases 2, 3, and 4), I understand that I must arrive at 9PM sharp. I will be asked to remain in the lab until

morning (after 7AM) for the screening and first experimental sessions (phases 2 and 3) and until after the 9AM recall test for the second experimental session (phase 4)

During the main study day, I understand that I will perform multiple sessions of a computerized memory task. I will be asked to learn pairs of word syllables that I will later be asked to recall to the best of my ability. Women participants will be given a short questionnaire to verify phase of the menstrual cycle; this is needed to ensure there is not influence of menstrual cycle phase on the sleep variables studied.

PART B: INFORMATION ABOUT STUDY RISKS AND YOUR RIGHTS AS A PARTICIPANT

I understand that I may experience some skin irritation (redness and dry skin) as a result of having electrodes attached to my scalp, face, and body. This is temporary and may be reduced by applying moisturizing cream to the areas where electrodes were placed.

I understand that the Sleep Laboratory facilities are under 24-hour video surveillance. All activities in the main laboratory, bedrooms, and the kitchen/lounge areas are recorded and stored in the Sleep Laboratory until completion of the study. The videotaped data will not be used in public presentation or advertising.

I understand that I will receive a total honorarium payment of up to $75 or $50 and course credit for completion of the full study. This includes the $25 or course credit received for completion of the orientation and screening night, a $25 payment for completion of the first experimental overnight procedure (phase 3), and a $25 payment for completion of the full study. If I withdraw or do not meet study inclusion criteria at any point, I will receive the payment for the phases completed up to that point I understand that should I withdraw from the study, researchers will destroy any data that I have provided upon request.

I understand that my participation is voluntary and I may withdraw from the study at any time, for any reason, without penalty. I am under no obligation to answer any question or participate in any aspect of this project that I consider invasive, offensive, or inappropriate. I understand that I may ask further questions at any time.

I understand that all personal data will be kept strictly confidential and all information will be coded so that my name is not associated with my answers. Only the researchers named above, and research assistants working under supervision of these researchers, will have access to the data. Electronic copies of data will be kept in the Sleep Research laboratory indefinitely. I understand that I am not anonymous in this study because the nature of the study requires that research assistants interact with each participant in the laboratory on a one-to-one basis and have contact information to schedule appointments.

Your signature below indicates that, you are of the age of legal consent (i.e., 18 years or older), you have read and understood the procedures of the study, and you agree

to participate.

Participant's Signature_____ Date _____
(to be s***igned during your visit to the Sleep Laboratory for orientation and sleep screening)***

PART C: CONTACT INFORMATION

This study has been reviewed and cleared by the Bioscience Research Ethics Board (File # 12-265-COTE). For answers to questions about your rights as a research participant, contact the Research Ethics Officer, at (905) 688-5550 ext. 3035, or reb@brocku.ca.

If you have any questions or concerns about your participation in the study you may contact the Principle Student Investigator, Kevin MacDonald in the Sleep Laboratory at (905) 688-5550, extension 5743, or the Faculty Supervisor Dr. Kimberly Cote in the Psychology Department at (905) 688- 5550, extension 4806 or.

No individual feedback from the sleep study or performance data may be provided at any time. Feedback about the outcome of the study will be available by request after final publication of the data (email: kcote@brocku.ca).

Please take a copy of this form with you for future reference. IF YOU NEED TO CONTACT THE LABORATORY REGARDING YOUR APPOINTMENT OR STUDY PROCEDURES, PLEASE CALL US AT **905-688-5550, EXT. 3795.**

I have fully explained the procedures of this study to the above volunteer.

Researcher's Signature_____ Date _____

Appendix H

Analyses of Block P

One reason for the inclusion of Block P in the memory task was its use as a control set for the directed forgetting manipulation given that it would not be cued as either TBR or TBF. In this sense, forgetting of Block P could be considered a measure of the natural degradation of memories that need not be retained. The following analyses explore this idea in a reduced sample size to account for the overall worse performance in learning Block P compared to the TBF Block A and TBR Block B. To conduct these analyses, an additional four participants were removed from the sample because they did not meet the 60% criterion in the last Syllable Trial of Block P. Thus for the following analyses, the sample included a total of 27 participants with 15 participants (11 women) in the Stable group and 12 participants (6 women) in the Labile group.

Even once those performing below the criterion were removed, the remaining participants were found to make more errors in learning Block P (M = 10.93, SD = 5.68) compared to both the TBF Block A (M = 7.15, SD = 4.65; t (26) = 3.16, p = .004) and TBR Block B (M = 6.04, SD = 3.60; t (26) = 4.56, $p < .001$). However, most participants did reach ceiling-level performance over the block with 70% of participants making zero errors in the last Syllable Trial of Block P, compared to 82% for both Block A and Block B. Because of the heavily skewed distributions, the Wilcoxon signed-rank test was used to compare performance in these last Syllable Trials. It was found that participants did not make significantly more errors in last Syllable Trial of Block P compared to those in either the TBF Block A (z = -0.37, p = .713) or the TBR Block B (z = -1.17, p = .244). These results suggest that although participants showed a greater difficulty in learning

Block P, they eventually learned its syllable pairs to a similar strength as they learned those of Blocks A and B by the end of the presentation of each.

Forgetting scores were analyzed in this sample using a Group (Stable vs. Labile) by Block (P vs. TBR vs. TBF) mixed-model ANOVA to explore the fate of Block P in comparison to the effects of the directed forgetting and memory reactivation manipulations. A significant main effect of Block was found ($F(2, 50) = 14.35$, $p = .001$, $\eta^2 = .359$) while both the main effect of Group and the Group by Block interaction were found to be non-significant. Follow-up paired-samples *t*-tests were performed to compare the forgetting scores for the three blocks. While there was no significant difference between forgetting of the TBR ($M = 1.67$, $SD = 1.24$) and the TBF ($M = 1.82$, $SD = 1.18$) blocks, participants showed greater forgetting of Block P ($M = 3.00$, $SD = 1.24$) when compared to both the TBR block ($t(26) = 5.10$, $p < .001$) and the TBF block ($t(26) = 4.19$, $p < .001$). It may be the case that there was not enough statistical power to adequately test the interaction in this further reduced sample; however, these results do suggest that there is less forgetting of material learned for a test compared to material that was never intended to be remembered for a test.

In this vein, participants were also more likely to make relearning errors in Block P compared to the TBR and TBF blocks with 70% of participants making at least one error in relearning Block P while only 44% and 41% made errors in relearning the TBR and TBF blocks, respectively. Further, comparisons made using the Wilcoxon signed-rank test found that participants had a significant tendency to make more relearning errors in Block P compared to the TBR block ($z = -3.19$, $p = .001$), and a similar tendency to make more relearning errors in Block P compared to the TBF block reached marginal

significance (z = -1.92, p = .055).

Correlation coefficients were calculated to further explore the relationships between Block P forgetting scores and the other forgetting scores, the percentage of total sleep time spent in each stage on Night 2, and the power spectral variables of interest from Night 1 and Night 2 sleep. Block P forgetting did not significantly correlate with TBR forgetting, TBF forgetting, total forgetting, or directed forgetting scores in either the Stable or Labile groups. With the Stable and Labile groups combined, significant positive correlations were found between Block P forgetting and both TBR forgetting (r = .40, p = .039) and total forgetting (r = .41, p = .036). The correlation between Block P forgetting and TBF forgetting was not significant (r = .26, p = .185). No significant correlations were found between Block P forgetting and any of the power spectral variables of interest or any of the sleep stage percentages. However, a marginally significant positive correlation between the percentage of REM sleep on Night 2 and Block P forgetting was found (r = .34, p = .079). This is interesting as it is reminiscent of findings in the main analyzes that suggest REM sleep plays a role in the forgetting of stable or irrelevant memories. In addition, marginally significant negative correlations were found between Block P forgetting and both Night 1 Early Stage 2 delta power (r = -.35, p = .076) and Night 1 Early Stage 2 gamma power (r = -.35, p = .073), the same variables that, from Night 2, negatively correlated with the Labile group's TBR forgetting in the main analyses. Although one should be careful in interpreting such marginal findings, these correlations may suggest that the mechanisms that affect one's consolidation of material never intended to be remembered for a test (Block P) may be the same as those that affect the reconsolidation of TBR material following memory reactivation.

Appendix I

Comparisons of Stable and Labile Group Means for Night 2 EEG Power in Each Frequency Band Measured Globally in Each Sleep Stage Sampled

	Stable (n = 16)		Labile (n = 15)			
Measure	*M*	*SD*	*M*	*SD*	*t* (29)	*p*
Early Stage 2						
Delta	1.77	0.23	1.66	0.14	1.63	.119
Theta	1.08	0.19	1.01	0.16	1.06	.300
Alpha	0.58	0.22	0.52	0.26	0.73	.474
Slow sigma	0.57	0.28	0.38	0.24	2.06	**.048**
Fast sigma	0.42	0.26	0.31	0.32	1.05	.301
Beta	-0.54	0.21	-0.66	0.23	1.46	.154
Gamma	-1.53	0.18	-1.57	0.24	0.60	.557
SWS						.620
Delta	2.33	0.23	2.30	0.13	0.50	.337
Theta	1.22	0.22	1.16	0.16	0.98	.282
Alpha	0.68	0.28	0.58	0.26	1.10	.164
Slow sigma	0.45	0.25	0.32	0.29	1.40	.237
Fast sigma	0.23	0.28	0.11	0.30	1.21	.292
Beta	-0.77	0.21	-0.85	0.21	1.07	.347
Gamma	-1.70	0.22	-1.61	0.32	0.96	.131
REM						
Delta	1.30	0.22	1.19	0.16	1.55	**.026**
Theta	0.71	0.21	0.54	0.18	2.35	.080
Alpha	0.35	0.21	0.19	0.25	1.81	**.041**
Slow sigma	-0.06	0.27	-0.24	0.22	2.14	.078
Fast sigma	-0.20	0.29	-0.37	0.20	1.82	.390
Beta	-0.62	0.27	-0.69	0.15	0.87	.675
Gamma	-1.66	0.14	-1.68	0.15	0.42	**.003**
Late Stage 2						
Delta	1.80	0.20	1.55	0.22	3.23	.243
Theta	0.90	0.16	0.75	0.17	2.43	.142
Alpha	0.45	0.24	0.32	0.21	1.67	.134
Slow sigma	0.44	0.34	0.35	0.24	0.90	.095
Fast sigma	0.27	0.32	0.20	0.33	0.60	.071
Beta	-0.72	0.24	-0.79	0.21	0.90	.072
Gamma	-1.63	0.27	-1.65	0.22	0.20	**.018**

Note. Significance values of $p < .05$ are in boldface. EEG = electroencephalographic; SWS = slow-wave sleep; REM = rapid eye movement. EEG band power measures have been log10 transformed at each scalp site. Global power measurements averaged over F3, Fz, F4, C3, Cz, C4, P3, Pz, P4, O1, Oz, and O2.

Appendix J

Table J1

Correlations between TBR Forgetting and Night 2 EEG Power in Each Frequency Band at Each Scalp Site for Each Sleep Stage Sampled in the Stable Group

Band	F3	Fz	F4	C3	Cz	C4	P3	Pz	P4	O1	Oz	O2
Early Stage 2												
Delta	.31	.31	.21	.41	.43	.43	.29	.40	.38	.23	.36	.21
Theta	**.50**	**.50**	.36	**.53**	.38	**.50**	.39	.43	.44	.35	.35	.18
Alpha	.22	.17	.07	.38	.33	.32	.30	.39	.35	.23	.21	.18
Slow sigma	.06	-.05	-.06	-.04	-.03	-.03	-.20	-.13	-.09	-.19	.21	-.16
Fast sigma	-.17	-.17	-.25	-.15	-.10	-.13	-.14	-.06	-.06	-.07	.07	-.07
Beta	.16	.21	.09	.29	.27	.26	.25	.25	.30	.13	.23	.07
Gamma	.10	.30	.28	.26	.32	.33	.24	.28	.32	.29	.35	.10
SWS												
Delta	.08	.04	.04	.14	.17	.21	.15	.21	.21	.13	.35	.09
Theta	.06	.04	-.01	.17	.15	.19	.17	.26	.23	.25	.29	.16
Alpha	.10	.08	.03	.20	.19	.20	.14	.21	.22	.16	.33	.12
Slow sigma	.11	.02	.02	.03	.02	.10	-.15	-.08	-.02	-.08	.31	-.09
Fast sigma	-.07	-.09	-.11	-.11	-.10	.01	-.14	-.07	-.02	-.09	.19	-.03
Beta	.10	.18	.11	.18	.17	.24	.13	.17	.28	.06	.30	.07
Gamma	-.07	.08	.07	.07	.13	.14	.04	.08	.11	-.00	.21	-.07
REM												
Delta	.13	.08	-.30	.24	.15	.21	.15	.20	.18	.05	.10	.04
Theta	.42	.42	.19	.49	.40	**.51**	.34	.39	.43	.17	.32	.17
Alpha	.43	.41	.11	.46	.35	.44	.27	.31	.35	.08	.17	.07
Slow sigma	.41	.43	.07	.45	.36	.44	.32	.33	.38	.17	.29	.10
Fast sigma	.35	.35	.01	.40	.31	.40	.32	.38	.41	.23	.32	.17
Beta	.38	.40	.05	.45	.40	.43	.38	.45	.41	.24	.34	.18
Gamma	.16	.40	-.24	.33	.42	.22	.29	.39	.35	.18	.22	.15
Late Stage 2												
Delta	.21	.20	.24	.23	.17	.35	.18	.23	.43	.17	.37	.24
Theta	.34	.34	.28	.36	.28	.46	.26	.41	**.52**	.34	.32	.34
Alpha	.29	.26	.20	.37	.36	.41	.28	.38	.43	.25	.25	.29
Slow sigma	.18	.07	.07	.05	.03	.08	-.09	-.05	.02	-.11	.36	-.05
Fast sigma	.10	.14	.10	.10	.19	.21	.08	.20	.20	.17	.35	.18
Beta	.14	.19	.08	.24	.31	.32	.23	.30	.35	.13	.29	.21
Gamma	.17	.22	.11	.18	.27	.25	.17	.22	.21	.11	.24	.17

Note. Coefficients with significance values of $p < .05$ are in boldface; $p < .01$ are in boldface and italicized; and $p < .001$ are in boldface, italicized, and underscored. TBR = to-be-remembered; EEG = electroencephalographic; SWS = slow-wave sleep; REM = rapid eye movement. EEG band power measures have been log10 transformed.

Table J2

Correlations between TBR Forgetting and Night 2 EEG Power in Each Frequency Band at Each Scalp Site for Each Sleep Stage Sampled in the Labile Group

Band	F3	Fz	F4	C3	Cz	C4	P3	Pz	P4	O1	Oz	O2
Early Stage 2												
Delta	**-.62**	**-.54**	**-.64**	***-.69***	**-.64**	***-.72***	-.51	-.47	**-.63**	-.42	**-.53**	-.34
Theta	-.36	-.30	-.43	-.25	-.28	-.35	-.25	-.15	-.35	-.30	-.39	-.41
Alpha	-.12	-.10	-.16	-.11	-.14	-.21	-.17	-.03	-.19	-.25	-.14	-.22
Slow sigma	-.21	-.12	-.14	-.01	.07	-.00	-.09	.09	-.03	-.16	-.42	-.02
Fast sigma	-.41	-.40	-.47	-.29	-.28	-.35	-.25	-.23	-.33	-.26	-.47	-.21
Beta	***-.66***	**-.63**	***-.69***	**-.58**	-.51	**-.59**	-.50	-.44	**-.55**	**-.51**	***-.75***	-.48
Gamma	***-.66***	***-.66***	***-.71***	***-.67***	**-.63**	***-.67***	***-.66***	***-.65***	***-.70***	***-.66***	-.47	-.64
SWS												
Delta	-.44	-.39	-.39	**-.55**	**-.52**	**-.54**	-.51	-.46	-.45	-.24	-.27	.18
Theta	-.46	-.34	-.46	-.40	-.33	-.43	-.42	-.36	-.45	-.34	-.49	-.37
Alpha	-.33	-.21	-.31	-.28	-.17	-.29	-.36	-.27	-.35	-.36	-.35	-.38
Slow sigma	-.22	-.18	-.24	-.14	-.16	-.17	-.23	-.15	-.24	-.26	-.26	-.15
Fast sigma	-.36	-.35	-.42	-.36	-.38	-.40	-.30	-.30	-.40	-.24	-.30	-.20
Beta	**-.55**	**-.56**	-.62	**-.55**	-.50	**-.52**	**-.53**	-.47	**-.56**	-.49	**-.57**	-.47
Gamma	-.30	-.47	-.45	-.38	**-.52**	-.29	**-.54**	**-.53**	**-.54**	**-.57**	-.33	**-.55**
REM												
Delta	-.39	-.26	-.27	-.36	-.48	-.28	-.42	-.34	-.32	**-.54**	-.20	-.44
Theta	**-.58**	-.50	-.48	-.51	-.49	-.39	-.49	-.40	-.49	**-.54**	-.47	-.49
Alpha	-.38	-.26	-.27	-.38	-.30	-.30	-.40	-.30	-.37	-.31	-.22	-.15
Slow sigma	**-.59**	**-.52**	-.49	**-.57**	-.49	-.48	**-.52**	-.45	**-.53**	-.44	-.41	-.27
Fast sigma	**-.55**	-.47	-.50	**-.55**	-.43	-.42	-.51	-.42	-.46	-.47	-.48	-.30
Beta	-.51	-.26	-.29	-.45	-.32	-.26	-.44	-.40	-.45	-.41	**-.62**	-.22
Gamma	-.12	-.08	-.16	-.38	-.39	-.24	-.34	-.37	-.39	-.45	.27	-.26
Late Stage 2												
Delta	-.46	-.40	-.46	-.46	-.46	-.43	-.38	-.35	-.42	-.18	-.46	-.04
Theta	-.40	-.26	-.32	-.44	-.33	-.33	-.33	-.17	-.37	-.28	-.30	-.40
Alpha	-.32	-.24	-.29	-.32	-.26	-.30	-.34	-.18	-.33	-.37	-.34	-.43
Slow sigma	**-.53**	-.49	-.45	-.45	-.39	-.40	-.48	-.36	-.47	**-.55**	**-.57**	-.49
Fast sigma	-.41	-.35	-.42	-.34	-.28	-.30	-.32	-.28	-.38	-.30	**-.57**	-.27
Beta	**-.63**	**-.54**	**-.58**	**-.56**	-.44	-.50	**-.52**	-.42	**-.53**	**-.62**	***-.67***	**-.60**
Gamma	***-.64***	**-.58**	***-.66***	***-.68***	***-.68***	**-.62**	***-.65***	***-.66***	***-.64***	***-.75***	-.38	***-.69***

Note. Coefficients with significance values of $p < .05$ are in boldface; $p < .01$ are in boldface and italicized; and $p < .001$ are in boldface, italicized, and underscored. TBR = to-be-remembered; EEG = electroencephalographic; SWS = slow-wave sleep; REM = rapid eye movement. EEG band power measures have been log10 transformed.

Table J3

Correlations between TBF Forgetting and Night 2 EEG Power in Each Frequency Band at Each Scalp Site for Each Sleep Stage Sampled in the Stable Group

Band	F3	Fz	F4	C3	Cz	C4	P3	Pz	P4	O1	Oz	O2
Early Stage 2												
Delta	.06	.10	-.10	.10	.12	.10	-.04	.03	.01	-.21	-.03	-.22
Theta	.14	.22	-.08	.19	.11	.15	.10	.11	.10	.20	.31	.05
Alpha	.05	.09	-.09	.12	.09	.08	.00	.02	.02	-.02	.29	-.01
Slow sigma	-.02	-.07	-.15	.06	.07	.03	-.03	.03	.03	-.10	.08	-.06
Fast sigma	-.04	.01	-.14	.08	.01	.03	-.09	-.03	.02	-.22	.11	-.10
Beta	.34	.39	.24	.38	.24	.33	.24	.15	.29	.02	.27	.03
Gamma	.16	.28	.24	.36	.36	.31	.30	.25	.28	.18	.09	.05
SWS												
Delta	**.55**	.45	.41	.46	.41	**.50**	.32	.39	.44	.12	.24	.13
Theta	.23	.24	.09	.24	.17	.23	.15	.18	.21	.09	.33	.05
Alpha	.22	.21	.10	.20	.17	.16	-.01	.02	.05	-.07	.37	-.03
Slow sigma	.20	.18	.08	.27	.24	.24	.11	.17	.18	-.06	.15	-.02
Fast sigma	.15	.14	.04	.14	.00	.10	-.08	-.06	.01	-.24	.21	-.15
Beta	.22	.28	.12	.24	.13	.22	.09	.05	.18	-.18	.22	-.08
Gamma	-.02	.03	-.03	.17	.15	.06	.07	.06	.10	-.13	-.05	-.13
REM												
Delta	.20	.20	-.34	.24	.21	.28	.17	.20	.29	.04	.02	.02
Theta	.46	.44	.01	.39	.30	.47	.28	.30	.42	.09	.27	.07
Alpha	.28	.29	-.13	.28	.15	.28	.17	.15	.21	-.11	.34	-.13
Slow sigma	.25	.27	-.17	.31	.19	.27	.19	.16	.23	-.11	.34	-.13
Fast sigma	.30	.31	-.14	.36	.24	.33	.25	.22	.29	-.02	.36	-.01
Beta	.49	**.54**	.03	**.53**	.47	**.53**	.36	.37	.41	.06	.42	.08
Gamma	.09	**.52**	-.40	.48	**.55**	.44	.35	.39	.46	-.03	-.06	.02
Late Stage 2												
Delta	.12	.10	-.05	.08	-.00	.12	-.00	.03	.19	-.14	.07	-.13
Theta	.18	.23	-.04	.13	.08	.21	.12	.18	.28	.23	.34	.16
Alpha	.17	.16	.01	.05	.06	.09	-.11	-.08	-.01	-.17	.29	-.08
Slow sigma	.24	.17	.13	.23	.19	.25	.10	.13	.23	-.03	.26	.06
Fast sigma	.35	.28	.27	.31	.22	.35	.11	.20	.27	-.02	.40	.06
Beta	.36	.37	.20	.35	.27	.39	.20	.22	.35	-.01	.25	.15
Gamma	.16	.19	-.02	.21	.27	.27	.15	.17	.20	.00	-.06	.08

Note. Coefficients with significance values of $p < .05$ are in boldface; $p < .01$ are in boldface and italicized; and $p < .001$ are in boldface, italicized, and underscored. TBF = to-be-forgotten; EEG = electroencephalographic; SWS = slow-wave sleep; REM = rapid eye movement. EEG band power measures have been log10 transformed.

Table J4

Correlations between TBF Forgetting and Night 2 EEG Power in Each Frequency Band at Each Scalp Site for Each Sleep Stage Sampled in the Labile Group

Band	F3	Fz	F4	C3	Cz	C4	P3	Pz	P4	O1	Oz	O2
Early Stage 2												
Delta	-.13	-.16	-.19	-.14	-.11	-.13	-.13	-.13	-.17	.03	***-.70***	.05
Theta	.21	.30	.19	.24	.21	.28	.12	.17	.11	.30	.34	.21
Alpha	.26	.26	.25	.24	.23	.23	.19	.26	.17	.38	.42	.28
Slow sigma	.47	.39	.44	.50	.36	.44	.31	.30	.29	.36	.07	.36
Fast sigma	.36	.31	.29	.42	.41	.43	.43	.46	.40	.33	.49	.39
Beta	.10	.08	.03	.15	.16	.11	.21	.23	.16	.26	-.04	.20
Gamma	-.06	.01	-.06	.01	.07	-.17	-.03	.01	-.05	.08	-.23	.01
SWS												
Delta	-.02	-.09	-.09	-.13	-.15	-.11	-.12	.00	-.04	-.03	**-.59**	.26
Theta	.15	.19	.11	.15	.21	.18	.07	.20	.09	.26	.12	.26
Alpha	.16	.14	.16	.18	.17	.18	.20	.26	.21	.36	.18	.33
Slow sigma	.35	.27	.37	.35	.20	.33	.23	.23	.23	.35	.21	.33
Fast sigma	.33	.26	.26	.32	.23	.32	.36	.39	.33	.34	**.60**	.43
Beta	.18	.21	.13	.16	.20	.12	.19	.24	.15	.30	.05	.25
Gamma	-.09	.01	-.07	-.06	.07	-.07	-.01	.05	-.02	.10	-.12	.06
REM												
Delta	.25	.28	.33	.39	.41	**.53**	.35	.30	.30	.29	-.28	.30
Theta	.35	.41	.43	.40	.37	**.52**	.37	.38	.34	.28	.10	.31
Alpha	.07	.14	.14	.10	.15	.20	.08	.13	.11	.05	.39	.08
Slow sigma	.18	.26	.24	.17	.24	.25	.18	.23	.20	.21	.46	.28
Fast sigma	.01	.09	.09	.08	.18	.23	.20	.22	.28	.32	.36	.42
Beta	-.21	-.21	-.14	-.13	-.07	.08	.13	.10	.17	.32	-.21	.38
Gamma	.30	.21	.15	.09	.11	.33	.16	.14	.22	.20	-.30	.24
Late Stage 2												
Delta	.02	.02	.03	.10	.09	.21	.14	.17	.12	.21	-.48	.20
Theta	.26	.34	.43	.30	.25	.40	.19	.24	.14	.32	.48	.23
Alpha	.08	.15	.23	.10	.16	.19	.08	.16	.06	.22	.40	.11
Slow sigma	-.08	-.10	.09	-.08	-.13	.04	-.11	-.20	-.18	-.04	-.26	-.17
Fast sigma	.13	.14	.18	.16	.18	.26	.21	.21	.16	.16	.23	.16
Beta	.18	.24	.21	.27	.30	.29	.27	.35	.26	.25	.06	.21
Gamma	-.08	-.03	-.15	-.08	-.02	-.09	-.09	-.05	-.08	.01	-.22	-.14

Note. Coefficients with significance values of $p < .05$ are in boldface; $p < .01$ are in boldface and italicized; and $p < .001$ are in boldface, italicized, and underscored. TBF = to-be-forgotten; EEG = electroencephalographic; SWS = slow-wave sleep; REM = rapid eye movement. EEG band power measures have been log10 transformed.

Table J5

Correlations between Total Forgetting and Night 2 EEG Power in Each Frequency Band at Each Scalp Site for Each Sleep Stage Sampled in the Stable Group

Band	F3	Fz	F4	C3	Cz	C4	P3	Pz	P4	O1	Oz	O2
Early Stage 2												
Delta	.21	.23	.06	.29	.32	.30	.14	.25	.22	.01	.19	-.00
Theta	.37	.41	.16	.41	.28	.37	.28	.30	.31	.32	.37	.13
Alpha	.16	.15	-.01	.29	.24	.23	.17	.24	.21	.12	.29	.09
Slow sigma	.03	-.06	-.12	.02	.02	-.00	-.13	-.06	-.04	-.16	.17	-.13
Fast sigma	-.12	-.09	-.22	-.04	-.05	-.06	-.13	-.05	-.03	-.16	.11	-.10
Beta	.29	.34	.19	.38	.30	.33	2.78	.23	.33	.09	.29	.06
Gamma	.15	.33	.30	.35	.39	.37	.31	.30	.35	.27	.25	.09
SWS												
Delta	.36	.28	.25	.34	.33	.40	.27	.34	.37	.14	.34	.12
Theta	.16	.16	.04	.23	.18	.24	.18	.25	.25	.20	.35	.12
Alpha	.18	.17	.08	.23	.21	.21	.08	.13	.15	.05	.40	.05
Slow sigma	.18	.11	.06	.17	.15	.19	-.03	.05	.09	-.08	.26	-.06
Fast sigma	.04	.03	-.04	.02	-.06	.06	-.13	-.08	-.01	-.19	.23	-.10
Beta	.18	.26	.13	.24	.17	.26	.13	.12	.26	-.07	.29	-.00
Gamma	-.05	.06	.02	.14	.16	.12	.06	.08	.12	-.07	.09	-.11
REM												
Delta	.19	.16	-.37	.28	.20	.28	.19	.23	.27	.05	.07	.03
Theta	**.50**	.49	.12	**.50**	.40	**.56**	.36	.40	.48	.15	.33	.13
Alpha	.40	.40	-.01	.42	.29	.41	.25	.27	.32	-.02	.29	-.03
Slow sigma	.37	.40	-.05	.44	.32	.41	.29	.28	.35	.04	.36	-.02
Fast sigma	.37	.38	-.08	.43	.31	.42	.33	.34	.40	.12	.39	.09
Beta	.49	**.53**	.04	**.56**	.50	**.54**	.42	.47	.47	.17	.44	.15
Gamma	.14	**.52**	-.36	.46	**.55**	.38	.37	.44	.46	.09	.09	.10
Late Stage 2												
Delta	.19	.17	.11	.18	.09	.27	.11	.15	.35	.02	.25	.07
Theta	.30	.32	.14	.28	.21	.39	.22	.34	.46	.32	.38	.28
Alpha	.26	.24	.12	.24	.24	.29	.10	.17	.24	.05	.31	.12
Slow sigma	.24	.14	.11	.15	.13	.19	.00	.05	.14	-.08	.35	.01
Fast sigma	.26	.24	.21	.23	.23	.32	.11	.23	.27	.09	.43	.14
Beta	.29	.32	.16	.34	.33	.40	.24	.30	.40	.07	.31	.20
Gamma	.18	.24	.05	.23	.31	.29	.18	.22	.23	.06	.10	.14

Note. Coefficients with significance values of $p < .05$ are in boldface; $p < .01$ are in boldface and italicized; and $p < .001$ are in boldface, italicized, and underscored. EEG = electroencephalographic; SWS = slow-wave sleep; REM = Rapid eye movement. Band power measures have been log10 transformed.

Table J6

Correlations between Total Forgetting and Night 2 EEG Power in Each Frequency Band at Each Scalp Site for Each Sleep Stage Sampled in the Labile Group

Band	F3	Fz	F4	C3	Cz	C4	P3	Pz	P4	O1	Oz	O2
Early Stage 2												
Delta	-.46	-.43	-.51	-.51	-.46	**-.52**	-.39	-.37	-.49	-.24	***-.76***	-.18
Theta	-.10	-.01	-.15	-.01	-.04	-.04	-.08	.01	-.15	-.01	-.04	-.12
Alpha	.08	.09	.05	.08	.06	.01	.01	.14	-.02	.07	.17	.03
Slow sigma	.16	.17	.18	.30	.27	.26	.13	.24	.16	.12	-.22	.23
Fast sigma	-.03	-.06	-.12	.08	.07	.04	.11	.14	.04	.03	.00	.11
Beta	-.35	-.34	-.41	-.27	-.22	-.29	-.18	-.13	-.24	-.16	-.49	-.18
Gamma	-.44	-.40	-.47	-.41	-.35	-.43	-.43	-.39	-.46	-.36	-.43	-.39
SWS												
Delta	-.28	-.29	-.30	-.42	-.41	-.40	-.39	-.28	-.30	-.16	**-.53**	.27
Theta	-.19	-.10	-.13	-.16	-.08	-.16	-.22	-.10	-.22	-.05	-.23	-.07
Alpha	-.11	-.05	-.09	-.06	-.01	-.07	-.10	-.01	-.09	-.00	-.11	-.04
Slow sigma	.08	.05	.08	.13	.02	.10	-.00	.05	-.01	.05	-.04	.11
Fast sigma	-.02	-.06	-.10	-.03	-.09	-.05	.03	.05	-.05	.06	.18	.14
Beta	-.23	-.21	-.31	-.24	-.19	-.25	-.21	-.15	-.26	-.12	-.33	-.14
Gamma	-.24	-.29	-.32	-.27	-.28	-.22	-.34	-.30	-.35	-.29	-.27	-.31
REM												
Delta	-.09	.01	.03	.01	-.05	.15	-.05	-.03	-.02	-.16	-.29	-.09
Theta	-.15	-.06	-.03	-.07	-.08	.07	-.08	-.02	-.10	-.16	-.24	-.11
Alpha	-.19	-.08	-.08	-.18	-.09	-.06	-.20	-.10	-.16	-.16	.10	-.05
Slow sigma	-.26	-.16	-.16	-.25	-.15	-.15	-.21	-.14	-.21	-.15	.03	-.00
Fast sigma	-.34	-.24	-.26	-.29	-.16	-.12	-.20	-.13	-.12	-.10	-.08	.07
Beta	-.44	-.29	-.26	-.36	-.24	-.11	-.19	-.19	-.18	-.06	**-.51**	.09
Gamma	.10	.08	-.01	-.18	-.17	.05	-.11	-.14	-.11	-.16	-.02	-.02
Late Stage 2												
Delta	-.28	-.24	-.26	-.23	-.23	-.14	-.15	-.11	-.19	.02	**-.57**	.09
Theta	-.09	.05	.06	-.09	-.05	.04	-.09	.04	-.14	.02	.10	-.11
Alpha	-.15	-.06	-.04	-.14	-.07	-.07	-.16	-.02	-.17	-.10	.03	-.20
Slow sigma	-.37	-.36	-.23	-.33	-.32	-.22	-.37	-.35	-.40	-.36	-.51	-.41
Fast sigma	-.18	-.13	-.15	-.11	-.06	-.03	-.07	-.05	-.14	-.09	-.22	-.07
Beta	-.28	-.19	-.23	-.18	-.09	-.13	-.16	-.05	-.17	-.23	-.38	-.24
Gamma	-.45	-.38	-.50	-.47	-.43	-.44	-.46	-.44	-.45	-.46	-.37	-.51

Note. Coefficients with significance values of $p < .05$ are in boldface; $p < .01$ are in boldface and italicized; and $p < .001$ are in boldface, italicized, and underscored. EEG = electroencephalographic; SWS = slow-wave sleep.; REM = rapid eye movement. EEG band power measures have been log10 transformed.

Table J7

Correlations between Directed Forgetting and Night 2 EEG Power in Each Frequency Band at Each Scalp Site for Each Sleep Stage Sampled in the Stable Group

Band	F3	Fz	F4	C3	Cz	C4	P3	Pz	P4	O1	Oz	O2
Early Stage 2												
Delta	-.27	-.22	-.32	-.32	-.33	-.35	-.34	-.39	-.39	-.47	-.40	-.45
Theta	-.38	-.30	-.47	-.36	-.29	-.37	-.30	-.34	-.35	-.16	-.05	-.14
Alpha	-.18	-.08	-.16	-.28	-.25	-.25	-.31	-.38	-.34	-.26	.09	-.20
Slow sigma	-.08	-.02	-.09	.10	.10	.06	.18	.16	.12	.10	-.14	.11
Fast sigma	.13	.19	.12	.23	.11	.16	.06	.03	.08	-.16	.04	-.03
Beta	.18	.18	.15	.10	-.03	.07	-.01	-.10	-.01	-.12	.04	-.05
Gamma	.07	-.02	-.04	.11	.04	-.03	.06	-.04	-.04	-.11	-.27	-.05
SWS												
Delta	.49	.43	.38	.34	.25	.30	.18	.18	.24	-.01	-.11	.05
Theta	.18	.21	.11	.07	.01	.05	-.02	-.08	-.02	-.16	.03	-.12
Alpha	.12	.14	.07	-.00	-.03	-.04	-.15	-.20	-.17	-.23	.04	-.15
Slow sigma	.10	.17	.06	.26	.23	.14	.27	.27	.21	.02	-.18	.07
Fast sigma	.23	.24	.15	.26	.11	.10	.07	.01	.03	-.16	.02	-.12
Beta	.13	.10	.01	.07	-.05	-.03	-.04	-.12	-.10	-.24	-.08	-.16
Gamma	.06	-.05	-.11	.10	.02	-.09	.03	-.03	-.00	-.13	-.27	-.06
REM												
Delta	.07	.13	-.03	.00	.06	.07	.02	-.001	.11	-.01	-.09	-.02
Theta	.04	.02	-.19	-.11	-.10	-.04	-.06	-.10	-.01	-.09	-.06	-.10
Alpha	-.15	-.13	-.24	-.18	-.21	-.17	-.10	-.17	-.14	-.19	.17	-.22
Slow sigma	-.17	-.16	-.25	-.15	-.18	-.18	-.14	-.19	-.16	-.29	.05	-.24
Fast sigma	-.05	-.04	-.15	-.05	-.08	-.08	-.08	-.17	-.12	-.25	.05	-.19
Beta	.12	.14	-.02	.08	.07	.10	-.02	-.09	-.00	-.19	.08	-.11
Gamma	-.07	.12	-.16	.16	.13	.22	.06	-.01	.12	-.21	-.29	-.14
Late Stage 2												
Delta	-.10	-.10	-.30	-.15	-.18	-.24	-.19	-.21	-.25	-.32	-.31	-.39
Theta	-.16	-.11	-.34	-.24	-.20	-.26	-.16	-.24	-.25	-.12	.02	-.19
Alpha	-.13	-.11	-.20	-.33	-.31	-.34	-.41	-.48	-.46	-.43	.03	-.38
Slow sigma	.07	.11	.07	.19	.16	.17	.20	.19	.22	.08	-.11	.11
Fast sigma	.26	.15	.18	.22	.04	.15	.03	-.00	.08	-.20	.05	-.12
Beta	.22	.19	.12	.11	-.05	.06	-.03	-.09	-.00	-.15	-.04	-.06
Gamma	-.01	-.04	-.14	.03	.01	.02	-.03	-.05	-.00	-.11	-.32	-.09

Note. Coefficients with significance values of $p < .05$ are in boldface; $p < .01$ are in boldface and italicized; and $p < .001$ are in boldface, italicized, and underscored. EEG = electroencephalographic; SWS = slow-wave sleep; REM = rapid eye movement. EEG band power measures have been log10 transformed.

Table J8

Correlations between Directed Forgetting and Night 2 EEG Power in Each Frequency Band at Each Scalp Site for Each Sleep Stage Sampled in the Labile Group

Band	F3	Fz	F4	C3	Cz	C4	P3	Pz	P4	O1	Oz	O2
Early Stage 2												
Delta	.43	.34	.40	.49	.46	**.53**	.33	.30	.41	.39	-.14	.35
Theta	.50	**.52**	**.55**	.42	.43	**.54**	.33	.28	.40	**.52**	**.62**	.53
Alpha	.33	.31	.36	.31	.32	.38	.31	.25	.32	**.54**	.49	**.44**
Slow sigma	**.58**	.44	.50	.44	.25	.38	.35	.18	.27	.45	.43	.29
Fast sigma	***.67***	**.62**	***.67***	**.62**	**.60**	***.66***	**.59**	**.60**	**.63**	.51	***.83***	**.52**
Beta	***.67***	**.63**	**.62**	**.64**	**.58**	**.61**	**.62**	**.58**	**.61**	***.67***	**.62**	**.59**
Gamma	**.52**	**.59**	**.57**	**.59**	**.62**	**.58**	**.55**	**.58**	**.57**	***.65***	.22	**.57**
SWS												
Delta	.37	.26	.27	.37	.33	.38	.34	.40	.36	.18	-.27	.07
Theta	**.53**	.46	.49	.48	.47	**.53**	.43	.48	.47	**.53**	**.53**	**.55**
Alpha	.42	.30	.41	.40	.30	.41	.48	.46	.49	**.63**	.46	**.62**
Slow sigma	.50	.39	**.52**	.42	.31	.43	.41	.32	.40	**.53**	.41	.42
Fast sigma	**.59**	**.52**	**.59**	**.59**	**.53**	**.63**	**.57**	**.60**	**.63**	.51	***.78***	**.54**
Beta	**.63**	***.67***	***.66***	**.61**	**.61**	**.56**	**.63**	**.61**	**.62**	***.69***	**.54**	**.63**
Gamma	.19	.42	.34	.28	.51	.19	.47	.50	.46	**.58**	.18	**.53**
REM												
Delta	**.55**	.47	**.52**	***.65***	***.78***	***.70***	***.66***	**.56**	**.54**	***.72***	-.07	***.64***
Theta	***.81***	***.79***	***.79***	***.79***	***.75***	***.79***	***.75***	***.67***	***.72***	***.71***	.50	***.70***
Alpha	.39	.35	.36	.42	.39	.43	.41	.37	.42	.31	**.52**	.20
Slow sigma	***.67***	***.67***	***.64***	***.65***	**.63**	**.63**	**.61**	**.59**	**.64**	**.56**	***.76***	.47
Fast sigma	.49	.49	**.52**	**.56**	**.52**	**.57**	**.62**	**.56**	***.65***	***.68***	***.73***	**.62**
Beta	.57	.05	.14	.28	.23	.29	.50	.43	**.54**	**.63**	.36	**.52**
Gamma	.36	.25	.28	.41	.43	.49	.43	.45	**.53**	**.57**	-.49	.43
Late Stage 2												
Delta	.42	.37	.42	.49	.48	**.55**	.45	.45	.47	.34	-.00	.21
Theta	**.57**	**.52**	***.65***	**.64**	.51	**.63**	.45	.35	.45	**.52**	***.68***	**.54**
Alpha	.34	.34	.45	.36	.36	.43	.37	.29	.34	**.52**	***.64***	.47
Slow sigma	.39	.35	.47	.32	.23	.38	.33	.15	.26	.44	.28	.29
Fast sigma	.47	.43	**.52**	.44	.40	.48	.46	.42	.47	.39	***.69***	.38
Beta	***.71***	***.68***	***.69***	***.72***	***.64***	***.69***	***.69***	***.66***	***.69***	***.75***	**.63**	***.71***
Gamma	.49	.49	.44	**.52**	**.58**	.47	.49	**.53**	.49	***.67***	.14	.48

Note. Coefficients with significance values of $p < .05$ are in boldface; $p < .01$ are in boldface and italicized; and $p < .001$ are in boldface, italicized, and underscored. EEG = electroencephalographic; SWS = slow-wave sleep; REM = rapid eye movement. EEG band power measures have been log10 transformed.