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THE RESTORATIVE POWER OF NAPS: SLEEP-DEPENDENT CONSOLIDATION OF GENERALIZED PERCEPTUAL LEARNING

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ABSTRACT

Memory consolidation is a critical process by which labile learning is stabilized for long-term retention. While a growing body of research supports the role of sleep in consolidating memory, much of this work has focused on rote learning and overnight sleep. Less is known about whether generalized learning — where learners extract abstract patterns that can transfer across novel contexts — also benefits from shorter sleep opportunities, such as naps. Moreover, the mechanisms by which sleep supports such generalization remain unclear. In a series of behavioral and polysomnographic experiments, participants were trained to recognize synthetic speech stimuli in which no words were repeated. These tasks required learners to generalize beyond memorized items and instead acquire abstract acoustic-phonetic patterns. Chapter 2 asks whether a 90-minute nap is sufficient to stabilize generalized learning, compared to remaining awake. These findings suggest that a single nap can consolidate generalized learning, mimicking effects of overnight sleep. Chapter 3 replicates these behavioral findings using a revised design and confirms that participants show performance recovery after a nap. Importantly, this chapter introduces EEG measures and shows that the presence of the EEG cap does not disrupt consolidation. Chapter 4 investigates whether sleep history prior to the nap influences the effectiveness of sleep-based consolidation. Results reveal that recent and cumulative sleep restriction are associated with altered sleep architecture during the nap, and that waking out of slow wave sleep is associated with higher recovery post nap. This suggests that an individual's sleep history can modulate how effectively sleep consolidates learning. Chapter 5 uses polysomnography to examine which features of nap sleep predict individual differences in memory recovery. Contrary to predictions from some consolidation theories, sleep spindle density was not significantly associated with recovery. Instead, both slow wave sleep (SWS) and REM sleep duration were positively associated

with improved post-nap performance, as long as both stages were present during the nap. These findings highlight the importance of both sleep stages and suggest that intact sleep cycles may be necessary to support consolidation of generalized perceptual learning. Together, this work challenges the notion that multiple sleep cycles are required for consolidation, extends our understanding of generalized learning, and underscores the role of both sleep history and specific sleep stages — particularly SWS and REM — in supporting memory stabilization.

CHAPTER 1: INTRODUCTION

Learning depends on the formation of new memories, while forgetting describes the loss of use of those memories. Traditionally, there have been two broad theories of forgetting — decay and interference. Decay theories claim that information fades from memory with time which could arise because of the decay of representations or associations (Brown, 1958). Interference theories, on the other hand, argue that the retrieval (or storage or maintenance) of information is contested by other information, either proactive or retroactive (for review, see Wixted, 2004).

Consolidation is a process that works to stabilize memories against forgetting (Jenkins & Dallenbach, 1924), where memories are altered from a labile short-term form to a longer-lasting form (Dudai, Karni & Born, 2015). Consolidation is sometimes used to describe changes at the cellular level (such as with protein synthesis), where synaptic connections are stabilized shortly after learning (Kandel, Dudai, & Mayford, 2014). Consolidation is also sometimes considered at the systems level, where it is thought that brain systems reorganize themselves to support learned material and make them long-lasting memories (Squire, Genzel, Wixted, & Morris, 2015). A process by which consolidation may occur involves sleep, during which neural activity may help restructure and stabilize memories (Klinzing, Niethard & Born, 2019).

While there are several theories of sleep-dependent memory consolidation, theories that implicate various stages of sleep for being particularly important for consolidation (e.g., Ackermann & Rasch, 2014), and theories that implicate specific sleep microstructure in consolidation processes, it is unclear what mechanism, or mechanisms underlie the consolidation of generalized perceptual learning. Specifically, the dual process hypothesis, and variants of this hypothesis, associates different sleep stages with different types of memory. Research paradigms that are frequently used in support of this theory are half-night paradigms, where subjects are either

deprived of SWS or REM sleep. These studies investigate the dissociable effects of NREM and REM sleep on performance of different tasks — with deprivation of the first half of the night (heavy in NREM) negatively impacting performance on declarative tasks, and deprivation of the latter half of the night (heavy in REM) negatively impacting performance on non-declarative and procedural tasks (Plihal & Born, 1997).

However, this dissociation of NREM and REM sleep serving distinct memory systems is incongruent with findings, for example, that demonstrate that N2 sleep predicts performance on simple procedural memory tasks more than REM sleep (that dual process theory would predict would drive performance). As such, it may be that both sleep states support declarative and procedural memory. In fact, the sequential nature of sleep cycles and the integrity of those cycles may be important for memory consolidation. The Sequential Hypothesis (e.g., Giuditta et al., 1995) emphasizes the importance of intact NREM and REM cycles, suggesting that we weaken non-adaptive memories during NREM sleep and then store and integrate those surviving memories into pre-existing memories during REM sleep. However, this theory is not explicit about whether multiple cycles of intact NREM-REM are necessary for consolidation.

The Complementary Learning Systems Theory (McClelland, McNaughton, & O'Reilly, 1995; McClelland, 2013) takes an active system consolidation point of view and proposes that during sleep information is transferred from the hippocampus to the neocortex via a phenomenon called "neural replay" — which describes the repetition of specific neural activity that occurred during initial learning, again repeated later during sleep. These replay events are marked by spindles that occur during the up phase of slow waves during sleep. It is thought that the hippocampal system is initially more involved with encoding processes, whereas neocortical

networks provide a lasting basis for retention, signifying that these events facilitate long-lasting memory stability.

Finally, the Synaptic Homeostasis Model (Tononi & Cirelli, 2014) outlines the importance of spindles and slow waves in the memory consolidation process; however, it describes consolidation at the synaptic level. This model describes that during wakefulness, individuals undergo synaptic potentiation, increasing the number of neural connections that there are overall, and that during slow-wave sleep there is synaptic downscaling, where only the most pertinent connections are maintained to support consolidation, improving the signal-to-noise ratio of specific memories.

Much of the research on sleep consolidation has focused on rote learning specific pieces of information. For example, there is some research where the focus is on learning a particular motor pattern (Fischer, Hallschmid, Elsner, & Born, 2002). In other research it is learning declarative lists (Tucker, Hirota, Wamsley, Lau, Chaklader, & Fishbein, 2006) or a particular visual pattern (Karni, Tanne, Rubenstein, Askenasy, & Sagi, 1994). In these cases, participants learn items or associations that are specific and then must retrieve those same items later — in other words, study and test materials are the same. However, much of the learning that individuals perform naturally on a day-to-day basis does not rely on explicit memorization, or rote learning, but rather a more flexible process whereby one can generalize their initial learning to new situations.

Rote learning paradigms repeat information until it is committed to memory and have been thought to be most effective for the means of short-term memorization rather than long-term retention. Additionally, learners who utilize rote memorization may not understand the underlying architecture of the concepts that they are trying to understand, which is central to generalized learning. In other words, generalized learning allows individuals to understand the relationships

between and underlying rules that govern items that they are studying. This learning is thought to be retained long-term and can be applied in multiple contexts, not just the one in which they originally learned it.

Since generalized perceptual learning involves learning in one context and applying that learning to novel stimuli or in new contexts, it can be considered a type of skill learning. As such, it is distinguished from rote learning, which refers to repeated training on a fixed set of stimuli (Greenspan, Nusbaum, & Pisoni, 1988; Heald & Nusbaum, 2014; Heald, Van Hedger, Veillette, Reis, Snyder, & Nusbaum, 2022; Schwab, Nusbaum, & Pisoni, 1985). As with all learning, generalized perceptual learning is susceptible to interference; it can also be stabilized by sleep (i.e., Fenn, Nusbaum, & Margoliash, 2003; Brawn, Fenn, Nusbaum, Margoliash, 2008; Fenn, Margoliash, & Nusbaum, 2013). In other words, sleep has been shown to restore loss in performance seen after training and following a waking retention interval, as well as making learning robust against any future loss during subsequent retention intervals (i.e., Fenn, Nusbaum, & Margoliash, 2003; Fenn, Margoliash, & Nusbaum, 2013). Other research has suggested that a short daytime nap can produce consolidation for procedural memories, suggesting that successive sleep cycles may not be required to produce a consolidation effect (Backhaus & Junghanns, 2006). However, previous research has also demonstrated that there can be differences in sleep-dependent consolidation for generalized and rote learning — for example, that a night of sleep can restore performance of generalized perceptual learning but not rote learning (Fenn, Margoliash, & Nusbaum, 2013).

Although we know that a night of sleep consolidates generalized perceptual learning, the mechanisms of consolidation are still not well understood, even though there are different theories of consolidation that exist at varying levels of description (cellular, synaptic, systems, etc.). The

goal of this dissertation is to answer the overarching question: how does sleep support generalized auditory perceptual learning?

Specific Aim 1

Theories of memory consolidation either imply needing multiple cycles of sleep to produce a consolidation effect or do not explicitly address this as an issue. As a result, the first aim of the proposed research is to understand whether a full night of sleep is needed for consolidation of generalization learning. Does stabilization of generalized memories develop over consecutive sleep cycles? Or can we achieve stabilization of generalized perceptual learning with shorter bouts of sleep? The first approach to testing this is to determine whether a 90-minute nap opportunity is sufficient to restore and stabilize learning. We will then compare the effects of a 90-minute nap opportunity to the results of Fenn, Margoliash & Nusbaum (2013) to determine whether the magnitude of our effect is similar to that achieved by a full night of sleep.

Specific Aim 2

Part of the standard procedure in sleep research is to collect information about the sleep history of participants. This information is typically used to determine exclusion criteria to disqualify participants with poor sleep history from participation. While we disqualify individuals with psychiatric, neurological, and sleep conditions, we believe it is important to consider how the quality of a sleep intervention is impacted by sleep history. While there is substantial research on the impacts of partial sleep deprivation and irregular sleep patterns on cognitive performance, there is little research on how sleep history may impact a nap's ability to consolidate generalized perceptual learning performance. For example, immediate sleep history may contribute to sleep

inertia and homeostatic sleep pressure, both of which have been thought to impact cognitive performance (e.g., Hilditch & McHill, 2019). As such, the second aim is directed at understanding whether we can identify markers of partial sleep deprivation, such as evidence of increased homeostatic sleep pressure in our subjects and subsequently determine whether these markers are correlated with measures of consolidation.

Specific Aim 3

What are the neural mechanisms that mediate sleep consolidation? There is research that suggests that there are macrostructural components of sleep, or specific stages, that support consolidation of perceptual learning — with either N2, N3, and/or REM as being thought of as critical to consolidation (McDevitt, Duggan, & Mednick, 2015). Further, some theories of consolidation identify microstructural aspects of sleep as being crucial to the consolidation process. For example, spindles have been implicated as being a marker of the transfer of information from short-term memory to long-term memory stores (e.g., McClelland, 2013). Other research has demonstrated that through modulating the number of spindles in a bout of sleep via targeted memory reactivation or sleep cueing paradigms (e.g., Oudiette & Paller, 2013) the consolidation of various forms of learning can be enhanced. While perceptual learning research has implicated macrostructural (duration of N2, N3, and/or REM) and microstructural components (spindles), this research was largely conducted overnight, not with a daytime nap. Therefore, the third aim is directed at understanding which components of sleep are important to consolidating learning during a short daytime nap.

CHAPTER 2: IS A NAP SUFFICIENT TO PRODUCE A CONSOLIDATION EFFECT?

2.1 Introduction

A large body of research has shown that sleep consolidates memories by stabilizing and protecting them from forgetting (Fenn, Nusbaum, and Margoliash, 2003; Walker, Brakefield, Seidman, Morgan, Hobson, and Stickgold, 2003; Born and Wilhelm, 2012, etc.). Sleep-dependent memory consolidation has been widely demonstrated for both declarative and procedural learning (Plihal & Born, 1997; Backhaus & Junghanns, 2006; Alger, Lau, & Fishbein, 2010). Sleep aids in the ability to recall newly learned declarative memories (Plihal & Born, 1997; Gais, Lucas, & Born, 2006; Lahl, Wispel, Willigens, and Pietrowsky, 2008) and consolidates both motor (Walker, Brakefield, Seidman, Morgan, Hobson, and Stickgold, 2003; Brawn, Fenn, Nusbaum, and Margoliash, 2010) and perceptual learning (Stickgold, Whidbee, Schirmer, Patel, & Hobson, 2000).

It is important to note that there is a difference between rote learning — creating memories of specific experiences, stimuli, and actions — and generalized learning, which improves performance for stimuli that have not been specifically experienced previously (see Greenspan, Nusbaum, & Pisoni, 1988). Sleep does not simply consolidate rote learning — the repetition of a fixed set of items — but also generalized skills (e.g., Fenn, Nusbaum, and Margoliash, 2003; Brawn, Fenn, Nusbaum, & Margoliash, 2008). That is, sleep is beneficial not only for consolidating memories of particular stimuli or a particular action but also for consolidating procedural knowledge that leads to generalizable improvements in performance. Perceptual learning that is generalized in nature is thought to play a critical role in perception as we rarely experience the same stimuli twice. In the context of speech, rote-memorization of acoustic signals has been argued to be an untenable model to account for performance changes in recognition

performance, due to the lack of invariance that exists between the acoustic patterns of speech and the linguistic interpretation of those patterns. As such, it has been argued that listeners rely on generalized learning to uncover how best to attend to the acoustic properties of speech for a given context (Heald & Nusbaum, 2014), and there are differences in the neural mechanisms involved in rote and generalized learning (Heald, Van Hedger, Veillette, Reis, Snyder, & Nusbaum, 2022).

Recent research has demonstrated that when participants learn to recognize low-intelligibility synthetic speech in a context where no words repeat, the participants engage in a kind of procedural learning to guide how best to direct their attention to the speech, presumably by forming an abstract representation of the talker's vocal (acoustic-phonetic) space (Heald, Van Hedger, Veillette, Reis, Snyder, & Nusbaum, 2022). In these settings, participants who are trained and tested on all novel words, never hearing the same word twice, show significant improvements in performance, gaining on average about another 20 percentage points correct from pretest to posttest (Schwab, Nusbaum, & Pisoni, 1985; Fenn, Nusbaum, & Margoliash, 2003, 2013; Heald, Van Hedger, Veillette, Reis, Snyder, & Nusbaum, 2022). This is a robust form of learning that persists for 6 months without additional exposure (Schwab, Nusbaum, & Pisoni, 1985). Importantly, significant performance improvement is found immediately after training, but performance deteriorates over the course of a subsequent waking day. Sleep has two effects on the fate of this learning. First, after performance degradation over a waking day, sleep can restore performance to immediate post-training levels. Second, a night of sleep after training can protect against subsequent waking degradation (Fenn, Margoliash, & Nusbaum, 2013). This pattern of results has been replicated in a generalized sensorimotor task (Brawn, Fenn, Nusbaum, & Margoliash, 2008), and these studies, along with others (Sidaras, Alexander & Nygaard, 2009;

Pace-Schott, Verga, Bennett, & Spencer, 2012; Van Hedger, Hogstrom, Palmer, & Nusbaum, 2015; Batterink & Paller, 2017) suggest that sleep consolidates generalized learning.

The aforementioned studies evaluate performance after a full night of sleep, but due to physiological similarities between a nap and one sleep cycle in a full night's sleep, some memory consolidation may be possible from a short daytime nap. Sleep consists of four main stages that individuals cycle through over a full night of sleep (Berry, Brooks, Gamaldo, Harding, Marcus, & Vaughn, 2012). Notably, there is evidence to suggest that all these sleep stages can be potentially achieved in a 90-min period (Carskadon & Dement, 2005) and even as short as 45 min (Backhaus & Junghanns, 2006). Given that previous work has argued that processing that occurs during sleep stages N2, N3, and REM (McDevitt, Duggan, & Mednick, 2015) may be responsible for the consolidation of perceptual learning, this then raises the possibility that consolidation may be possible from an opportunity for a 90-min daytime nap. Does the minimal amount of sleep from a nap produce consolidation of learning, or is a full night's sleep necessary for consolidation to accrue over sleep cycles? If a nap presents sufficient sleep to produce some consolidation, this offers a potential explanation for why a daytime nap can prevent future daytime deterioration in a visual texture discrimination learning task and improve performance above baseline levels, similar to the improvement seen after a full night of sleep (Mednick, Nakayama, & Stickgold, 2003). Further, additional studies have demonstrated that a nap can generate a similar benefit for consolidating declarative memories compared to a full night of sleep (Mednick, Nakayama, & Stickgold, 2003; Tucker, Hirota, Wamsley, Lau, Chaklader, & Fishbein, 2006; Tucker & Fishbein, 2008; Alger, Lau, & Fishbein, 2010). While it has additionally been shown that a nap as short as 6 min can promote memory performance, it has been argued that the simple onset of sleep may be enough to kickstart processes associated with memory consolidation. Under this view, memory

consolidation processes once triggered by the onset of sleep are thought to continue upon waking, allowing for benefits to be realized despite only 6 min of putative sleep (Lahl, Wispel, Willigens, & Pietrowsky, 2008). Taken together, there is both behavioral and physiological evidence that the opportunity for a 90-min midday nap can consolidate generalized learning.

Despite this evidence, however, the efficacy of short-duration sleep on the consolidation of generalized learning remains an open question. Does sleep consolidation depend on aggregation of consolidation over sleep cycles, or does a shorter bout of sleep suffice for consolidation of memory? First, we must understand whether the accrual of multiple sleep cycles over the course of the night is necessary for memory consolidation or if a short daytime nap is sufficient for producing consolidation (Specific Aim 1). As such, the objective of my first proposed experiment is to demonstrate that a daytime nap provides a protective effect on learning against subsequent loss, similar to a full night of sleep (see Fenn, Margoliash, & Nusbaum, 2013).

To assess the consolidation effect of less than a full night's sleep with multiple cycles, we tested whether an opportunity for a 90-min afternoon nap would consolidate generalized perceptual learning of synthetic speech. The synthetic speech used in this paradigm is English speech generated by a computer program using orthographic-phonetic and phonetic-acoustic rules. The acoustic-phonetic patterns differ substantially from normally produced natural American English speech. Some acoustic cues are produced by errors in the synthetic speech model implementation, and thus the speech can be misleading or incorrect in comparison with English acoustic-phonetics, whereas some are consistent but show less acoustic cue covariation than found in natural speech (Nusbaum & Pisoni, 1985). For example, the word "bit," when pronounced by the synthesizer, might sound more like "bat." In this regard, the process of learning and understanding synthetic speech can be thought of as similar to the process of understanding

foreign-accented speech: individuals must learn new acoustic-phonetic patterns and map them onto pre-existing phonological categories. We hypothesized that an afternoon nap would consolidate this learning and reduce waking degradation of performance. Participants were trained on synthetic speech in the morning, then randomly assigned to take a short nap in the afternoon or to remain awake. All were subsequently tested in the evening after a waking retention interval.

2.2 Materials and Methods

2.2.1 Participants

We recruited 75 participants from the University of Chicago community. Participants had an average mean age of 20.83 (SD = 2.63, range: 18–32, 48 female, 27 male). All were right-handed and spoke English as a primary language with no history of speech or hearing disorders. Participants were randomly assigned to nap ($N = 39$) or to remain awake for a matched period of time ($N = 36$). There were no significant age ($t(73) = -0.46$, $p = 0.65$) or gender ($X^2_{(1, N=75)} = 0.97$, $p = 0.33$) differences between the groups.

2.2.2 Stimuli

We generated 700 monosyllabic words using *rsynth*, a text-to-speech synthesizer based on a formant-synthesis engine (Klatt, 1980). Three hundred words were used for training, and the 400 remaining words were divided into four 100-word tests matched in terms of overall recognition performance. In other words, these four tests are all balanced in terms of overall recognition performance at pretest, which indicates that each of the tests is matched in terms of initial difficulty. No word was repeated in any of the tests or during training. As a result, participants could not memorize the sound of any words to aid recognition (no rote memorization) but had to

learn general acoustic-phonetic characteristics of the speech in order to improve word identification. The tests were counterbalanced across participants within each condition. Stimuli were delivered through Sennheiser HD570 headphones with a mean RMS amplitude of 66.5 dB; sound level was measured using a sound level meter.

2.2.3 Procedure

All participants arrived at the lab at approximately 09:00 for a Pretest, Training, and a Posttest ("Posttest1"). During Pretest and Posttest1, participants listened to 100 unique monosyllabic synthetic speech words and transcribed what they heard by typing their responses on a computer keyboard. There was no feedback given during the tests.

During training, the 300 words were presented in 6 blocks (50 words per block). During each training block, participants first heard each of the 50 words paired with the orthographic form of the word displayed on the computer screen. There was a 1,000-ms inter-trial interval between words. After receiving this feedback on 50 words, participants were then played the words a second time and asked to identify them. During identification trials, participants had four seconds to type in a response. If they did not respond in that time, the next stimulus was presented. Words were presented randomly during both the feedback and identification phases. After finishing each block, participants were allowed to take a short break before proceeding with the rest of the training. This first session lasted ~45 min, including pretest, training, and first posttest. At the conclusion of the session, all participants were told to return at 15:00, prepared to nap, although not all participants did nap (wake controls). Participants were tested in groups of two.

At 15:00, both participant groups returned and were randomly assigned to either the nap or wake condition. The nap participants were given the opportunity to nap in a quiet, dark room for

90 min. Wake control participants remained awake and were either (1) allowed to leave the lab (out-of-lab control group) until 16:30 (N = 16) or (2) asked to remain in the lab (in-lab control group) and not perform any tasks with audio and/or use electronics (N = 20). In-lab control participants were permitted to do puzzles, read, or otherwise occupy themselves but were not permitted to speak or listen to speech or music (and were not permitted access to cell phones). We created two separate wake groups to ease future data collection. Our goal was to better understand whether the results vary depending on whether participants remain in the laboratory or leave during the 90-min control interval.

Given that the groups differ in the amount of exposure to speech, with the in-lab group restricted in exposure and the out-of-lab group freely interacting with other people, it is possible that natural speech use by the out-of-lab group could adversely affect learning retention more than the in-lab group that was simply awake during this time. To control for speech interference, the in-lab control participants were housed in a room adjacent to nap participants to match general environmental noise conditions. All wake control subjects were instructed to refrain from sleeping or consuming caffeine and/or alcohol.

At 16:30, the napping participant was awakened by the researcher, and verbal confirmation was obtained from the participant that they had slept. Control participants who did not sleep either returned to the lab (if they were assigned to the out-of-lab control) or were notified that their silent waking block was finished (if they were assigned to the in-lab control). It was confirmed that all participants assigned to the nap condition self-reported that they had napped for some portion of the nap block and that all control participants self-reported having remained awake for the entirety of the 15:00–16:30 block. All participants then completed a second posttest (Posttest2). After this, all participants were permitted to leave the lab and were instructed to refrain from napping until

after their final session. All participants returned at 21:00 for the final posttest (Posttest3). See Figure 2.1 for an outline of all study activities.

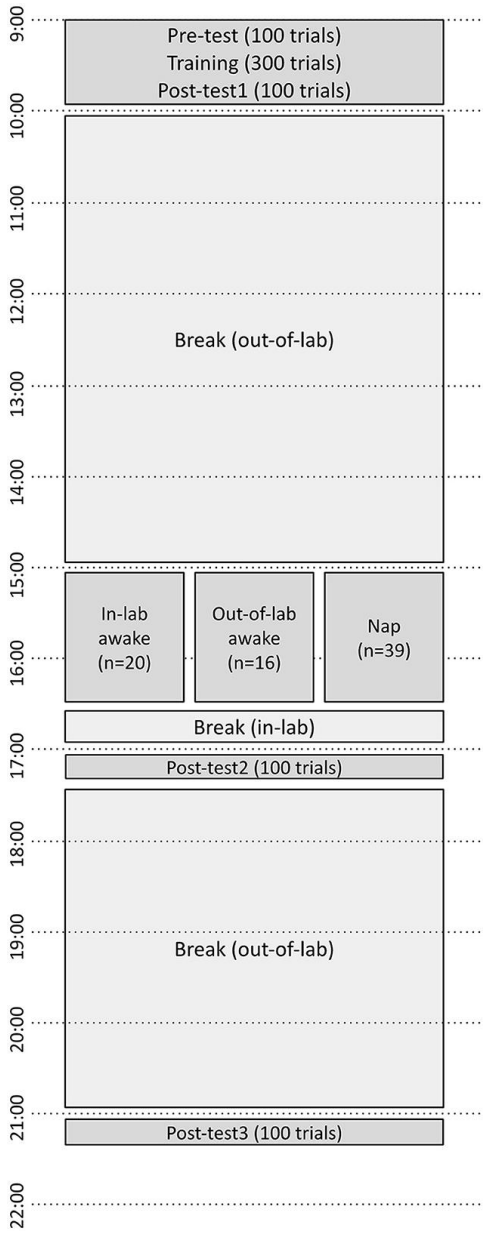

Figure 2.1. Timeline of perceptual learning tests and training over the course of the experiment. Pretest, Training, and Posttest1 began at 9:00. After an out-of-lab break, subjects either napped or remained awake (in the lab or outside of the lab) from 15:00 to 16:30, then performed Posttest2 at 17:00 and Posttest3 at 21:00 after another out-of-lab break.

2.2.4 Data Analysis: Behavioral Task

For each test, we scored accuracy based on phonetic transcription, without penalizing spelling errors. For example, if the stimulus was “tune” the response “toon” would be scored as correct, but “tunes” would be incorrect because it included an additional phoneme. For all participant responses that did not exactly match the spelling of the stimulus, response accuracy was decided upon as a group using the International Phonetic Alphabet (IPA) using a Midwestern dialect. In the case that the spelling of a response matched a homophone found in the IPA, the spelling of the response was marked as correct. In other words, word recognition accuracy was standardized by the International Phonetic Alphabet. Additionally, scorers were blind to the experimental condition to which participants were assigned.

2.3 Results

To assess whether initial generalized learning did indeed occur, we compared Pretest and Posttest1 word recognition accuracy (proportion correct) using a 2 × 3 repeated measures ANOVA, with test (Pretest, Posttest1) as a within-subjects factor and condition (Nap, Out-of-Lab Wake, In-Lab Wake) as a between-subjects factor. Participants showed significant improvement as a result of training, as revealed by a significant main effect of test ($F(1, 72) = 239.89$, $p < 0.001$, $\eta 2 = 0.34$), with an average improvement of 17 percentage points (Pretest percent correct: $M = 33$, $SE = 1$; Posttest1 percent correct: $M = 50$, $SE = 2$). This demonstrates that participants did indeed demonstrate generalized learning, as no words were repeated across the tests or training. As conditions were treated identically through Posttest1, it is unsurprising that there was no significant main effect of condition ($F(2, 72) = 0.82$, $p = 0.45$, $\eta 2 = 0.01$), or significant interactions between the factors ($F(2, 72) = 2.11$, $p = 0.13$, $\eta 2 = 0.01$). For estimated marginal means for each cell of

the repeated measures ANOVA (see Table 2.1). For a plot of the proportion correct for all groups between pretest and Posttest1 (see Figure 2.2).

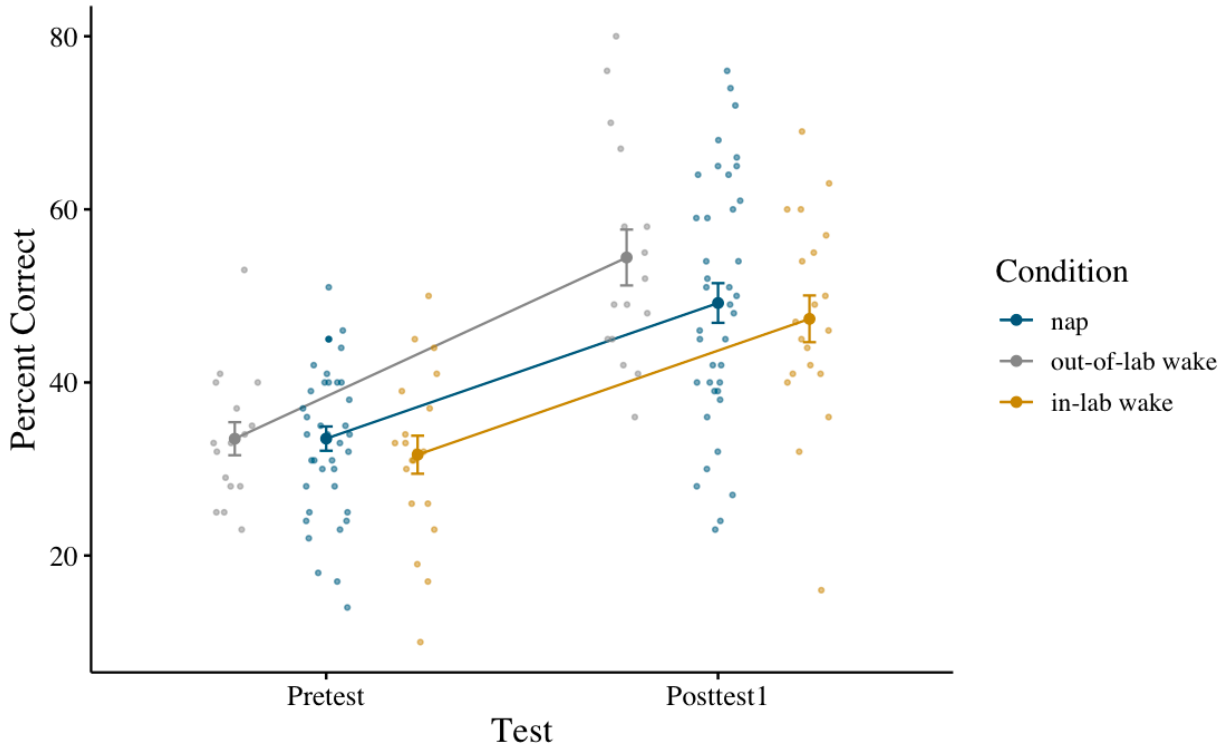

Figure 2.2. Pretest and Posttest1 performance on the perceptual learning task for all conditions. Error bars depict standard error.

Estimated Marginal Means Percent Correct – Test * Condition					
Condition	Test	Mean	SE	95% Confidence Interval	
				Lower	Upper
Nap	Pretest	34	1	31	36
	Posttest1	49	2	45	54
Out-of-lab wake	Pretest	34	2	29	38
	Posttest1	54	3	48	61
In-lab wake	Pretest	32	2	28	36
	Posttest1	47	3	41	53

Table 2.1. Estimated marginal percent correct means table for Pretest and Posttest1 performance for all conditions. Mean values represent the proportion of correct trials (for example, a mean percent correct value of 34 is equivalent to 34% correct trials).

To see if there was a difference between our two wake control conditions across any of our tests, we additionally performed a 2 (control type: in-lab vs. out-of-lab control groups) × 4 (test block: Pretest/Posttests 1–3) repeated measures ANOVA. Failure to find a significant main effect of control type and failure to find an interaction effect between control type and test block would allow us to collapse across the waking control conditions for further analyses. There was a main effect of test block ($F(3, 102) = 54.30$, $p < 0.001$, $\eta 2 = 0.25$), which is to be expected as this demonstrates that participants fluctuate in performance across the day; in other words, learning and loss occurred over the course of the day. While we failed to find a significant interaction effect ($F(3, 102) = 2.28$, $p = 0.08$, $\eta 2 = 0.01$), we did find a marginally significant effect of control type ($F(1, 34) = 3.44$, $p = 0.07$, $\eta 2 = 0.05$; see Figure 2.3 for a plot of the data associated with this analysis, as well as Table 2.2 for each cell of the repeated measures ANOVA). To better understand the marginally significant effect of condition we found, we conducted a post-hoc independent sample t-test to additionally assess how retained learning (Posttest3 – Posttest1) over the course of the experiment differed between the two wake conditions. For these groups, the retained learning measure captures individual differences in post-wake retention performance relativized by individual differences in initial post-training performance. Using an independent sample t-test, we fail to find a difference in retained learning between the two wake groups ($t(34) = -0.30$, $p = 0.76$).

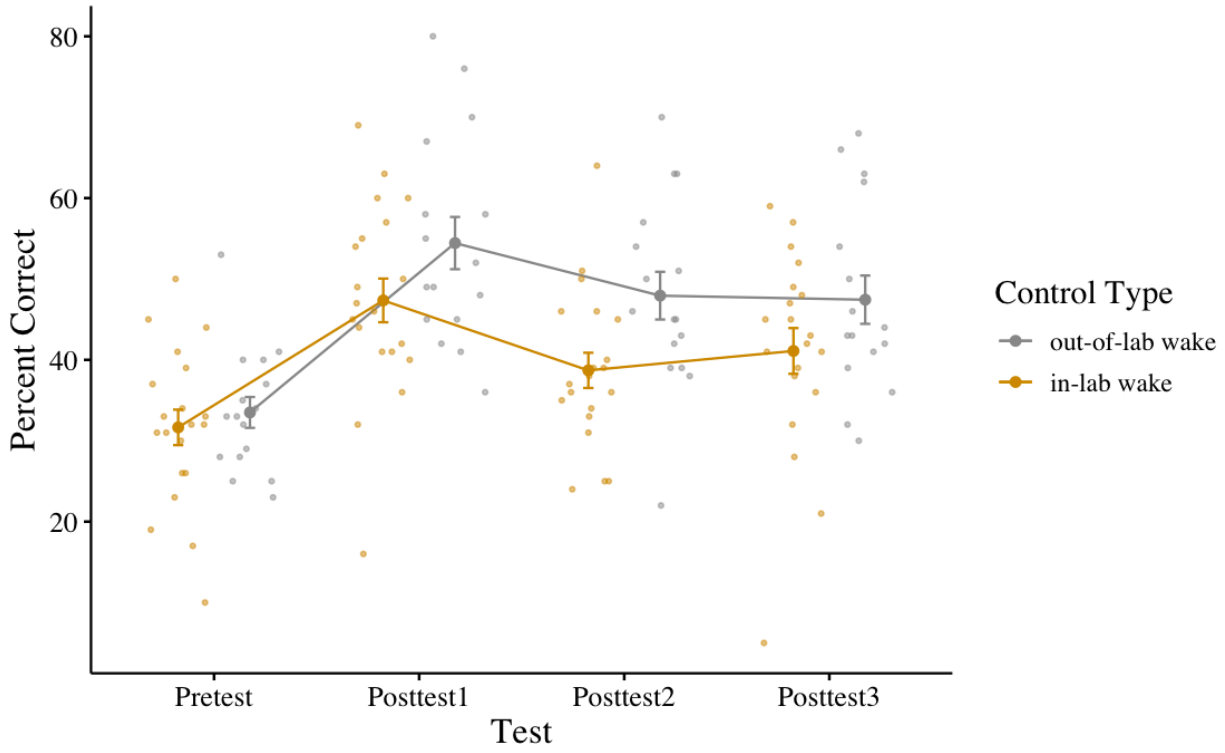

Figure 2.3. Performance of the two wake control groups across all perceptual learning tests. One control group remained in the lab during the 15:00–16:30 block (in-lab group), while another was allowed to leave the lab (out-of-lab group) during this time. Error bars depict standard error.

Estimated Marginal Percent Correct Word ID Means – test * control type					
Condition	Test	Mean	SE	95% Confidence Interval	
				Lower	Upper
Out-of-lab wake	Pretest	34	2	29	38
	Posttest1	54	3	48	61
	Posttest2	48	3	43	53
	Posttest3	47	3	41	54
In-lab wake	Pretest	32	2	28	36
	Posttest1	47	3	41	53
	Posttest2	39	2	34	44
	Posttest3	41	3	36	47

Table 2.2. Estimated marginal percent correct means table for Pretest, Posttest1, Posttest2, and Posttest3 performance for both wake conditions.

Given that we could only find evidence that the control conditions acted marginally differently across the various tests, any additional variance obtained by collapsing the control

conditions should only contribute to a type II error for later tests, and as such, we have chosen to collapse the control conditions for the remainder of the analyses.

To examine the effect of napping on word recognition ability, we examined test performance (proportion correct) directly after training (Posttest1), directly after the nap or wake manipulation depending on condition assignment (Posttest2), and later in the day (Posttest3) using a 3 × 2 repeated measure ANOVA with test (test block: Posttest1, Posttest2, and Posttest3) as a within-subjects factor and condition (condition type: Nap and Wake) as a between-subjects factor. We failed to find evidence for a main effect of condition type ($F(1, 73) = 0.23$, $p = 0.63$, $\eta^2 = 0.003$). However, a significant interaction effect between the factors ($F(2, 146) = 3.03$, $p = 0.05$, $\eta^2 = 0.01$) indicates that the two condition types (Nap and Wake) performed differently across the three posttests. Additionally, a significant main effect of test block was found ($F(2, 146) = 21.43$, $p < 0.001$, $\eta^2 = 0.04$). A post-hoc Tukey test reveals that there was a significant pairwise difference between Posttest1 ($M = 50$, $SE = 2$) and Posttest2 ($M = 44$, $SE = 1$; $p < 0.001$), and Posttest1 and Posttest3 ($M = 46$, $SE = 1$; $p < 0.001$). By using the post-hoc Tukey test, we failed to find evidence that Posttest2 and Posttest3 ($p = 0.13$) were significantly different from one another. See Figure 2.4 for a plot of the data associated with this analysis, as well as Table 2.3 for each cell of the repeated measures ANOVA.

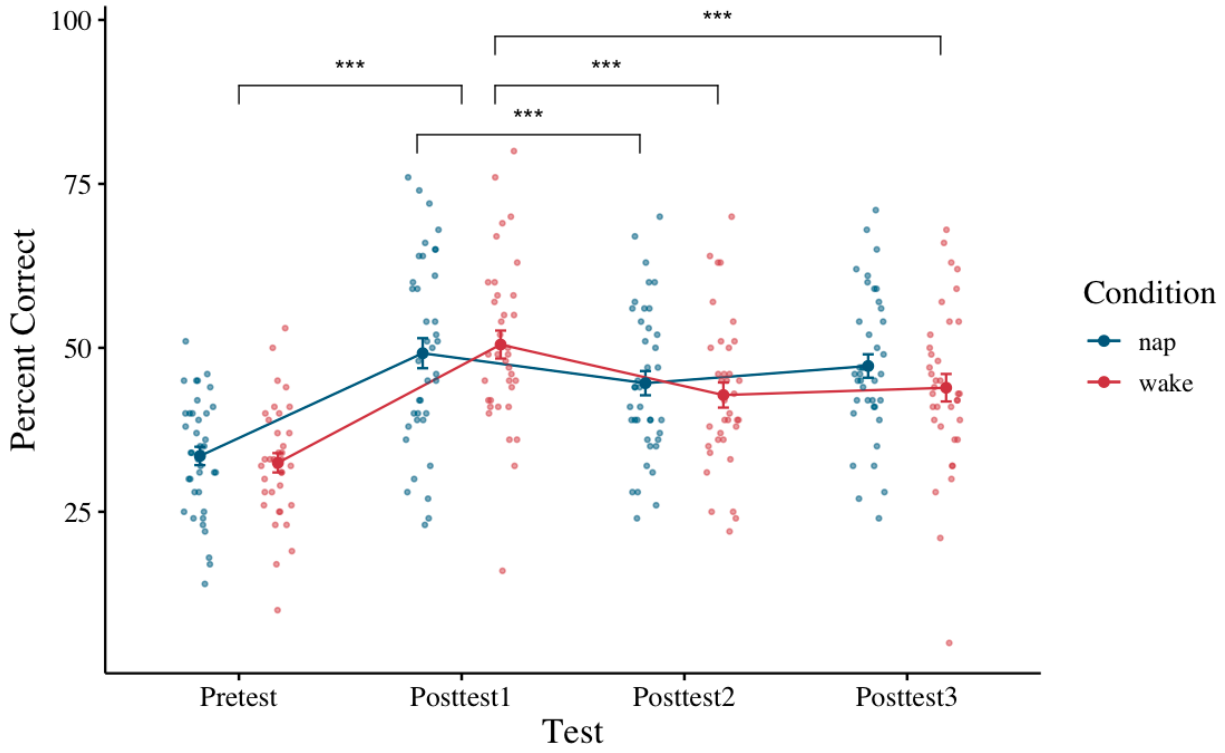

Figure 2.4. Performance patterns in the nap and wake groups across all tests (Pretest, Posttest1, Posttest2, Posttest3). Error bars depict standard error. *** $p < 0.001$

Estimated Marginal Means – test * condition					
Condition	Test	Mean	SE	95% Confidence Interval	
				Lower	Upper
Nap	Posttest1	49	2	45	54
	Posttest2	45	2	41	48
	Posttest3	47	2	43	51
Wake	Posttest1	51	2	46	55
	Posttest2	43	2	39	47
	Posttest3	44	2	40	48

Table 2.3. Estimated marginal means table for Pretest, Posttest1, Posttest2, and Posttest3 performance for both the condition types (nap and wake).

Given our a priori hypotheses and to better understand the significant interaction term in the previous model, we conducted two a priori planned one-way (test block: Posttest1, Posttest2, and Posttest3) repeated measure ANOVAs, one for the nap condition and one for the wake

condition on test performance (proportion correct) directly after training (Posttest1), directly after the nap (Posttest2), and later in the day (Posttest3).

For the nap condition, we found a significant main effect of test block ($F(2, 76) = 5.74$, $p = 0.01$, $\eta^2 = 0.02$). A post-hoc comparison using Tukey found evidence that Posttest1 ($M = 49$, $SE = 2$) and Posttest2 ($M = 45$, $SE = 2$) significantly differed from one another ($p = 0.01$), although we failed to find evidence that Posttest1 and Posttest3 ($M = 47$, $SE = 2$; $p = 0.31$) differed or that Posttest2 and Posttest3 ($p = 0.11$) differed.

While we would have expected sleep to restore any loss of learning, the decrease in proportion correct seen between Posttest1 and Posttest2 ($M = 5$, $SE = 1$) could be due to sleep inertia. However, we see that this loss in performance is ameliorated by Posttest3, as we fail to find evidence of a difference in performance between Posttest1 and Posttest3 ($p = 0.31$). Important to our research question, this return to initial post-training performance should only be possible if sleep consolidation has occurred.

For the wake condition, we found a significant main effect of test type ($F(2, 70) = 18.7$, $p < 0.001$, $\eta^2 = 0.07$). A post-hoc comparison using Tukey found evidence that Posttest1 ($M = 51$, SE = 2) and Posttest2 ($M = 43$, $SE = 2$) significantly differed from one another ($p < 0.001$), and Posttest1 and Posttest3 ($M = 44$, $SE = 2$) significantly differed from one another ($p < 0.001$), although we failed to find evidence that Posttest2 and Posttest3 ($p = 0.71$) differed.

Here, the decrease in proportion correct seen between Posttest1 and Posttest2 ($M = 8$, $SE = 1$) is consistent with the traditional loss usually seen as a consequence of a waking retention interval. However, we fail to find evidence that this loss in performance changes by Posttest3, as we fail to find evidence of a difference in performance between Posttest2 and Posttest3 ($p = 0.71$). Unlike in the nap condition, in the wake condition participants show a significant difference

between initial post-training performance and Posttest3 ($p < 0.001$), which is consistent with a pattern of performance where learning has yet to be consolidated.

While these results are consistent with the idea that the nap conditions demonstrated learning that has been consolidated, a direct statistical comparison between the groups in retained learning (Posttest3 - Posttest1 performance) is still needed. Posttest3 - Posttest1 performance would indicate how much of a performance decrement participants are experiencing over the course of the day. We would expect that if our nap subjects were consolidating their learning, then this difference score would be close to zero, while our wake subjects should be demonstrating steady performance over the course of the day, producing a negative Posttest3 - Posttest1 value. Indeed, we find that retained learning is not significantly different from zero for our nap subjects ($M = -2$, $SE = 1$; $t(38) = -1.48$, $p = 0.15$) and is significantly less than zero for our wake subjects ($M = -7$, $SE = 1$; $t(35) = -5.44$, $p < 0.001$), reflecting loss. Further, an independent sample t-test on retained learning between the nap group and the wake group is indeed significant ($t(73) = 2.58$, $p = 0.01$). See Figure 2.5 below for the distribution of retained learning for the nap and wake groups. The results from this test demonstrate that there is a direct difference in retained learning performance as a function of the nap. Notably, we find this significant result despite having collapsed against the two waking control groups, which may have added some additional variance in performance despite a lack of statistical evidence saying that the two groups performed differently.

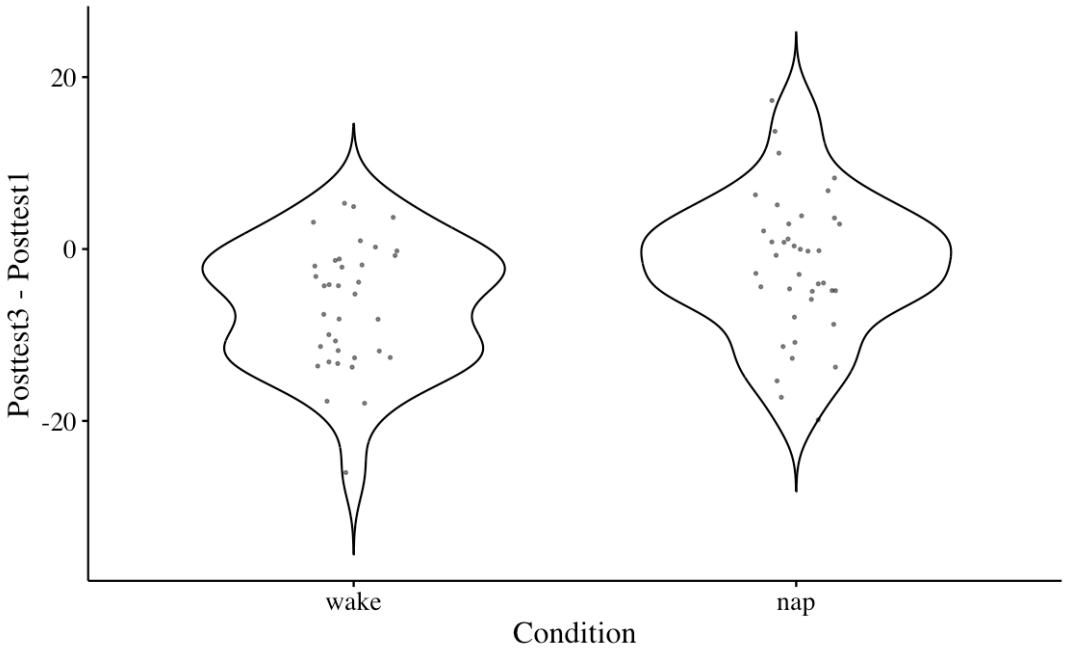

Figure 2.5. Violin plot for the distribution of retained learning (Posttest3–Posttest1) performance across nap and wake groups. The mean near zero here for nap subjects indicates an overall consolidation effect at the group level, and the dispersion around this demonstrates the observed variation found in this effect across subjects. Negative values indicate worse performance at Posttest3 than Posttest1 and positive values indicate better performance on Posttest3 than Posttest1.

Is learning, and the amount of learning retained in this study, similar to previous studies where subjects slept for an entire night? In Fenn, Margoliash, & Nusbaum (2013), a group of subjects received Pretest, Training, and Posttest1 at 9:00, Posttest2 at 21:00, and Posttest3 at 9:00 the next morning after a night of sleep. Learning in this study (M = 18, SE = 2) was similar to our nappers (M = 16, SE = 1; $t(60)$ = -1.08, p = 0.28), and the amount of learning retained (which can be calculated by the difference in Posttest3 − Pretest performance) is similar between Fenn, Margoliash, & Nusbaum (2013) (M = 15, SE = 2), and the present study (M = 14, SE = 1; $t(60)$ = -0.76, p = 0.45). As such, this suggests that a short bout of sleep in the middle of the day produces consolidation similar to a full night of sleep for this type of learning. Additionally, this is evidence

against hypotheses that indicate that multiple cycles of sleep are vital for consolidation, as it is highly unlikely that multiple full cycles of sleep will be seen during a 90-minute nap opportunity.

2.4 Discussion

Is a 90minute nap sufficient to produce consolidation of generalized perceptual learning, and if so, is consolidation from a nap similar to consolidation from a night's sleep? Previous research (e.g., Tucker, Hirota, Wamsley, Lau, Chaklader, & Fishbein, 2006; Tucker & Fishbein, 2008; Alger, Lau, & Fishbein, 2010) has suggested that a nap can consolidate rote learning. However, differences between rote and generalized learning and the effect of a daytime nap on consolidation of these forms of learning suggest that napping may not have the same effect for consolidation of generalized learning as it does for rote learning. We predicted that participants who learn to better recognize synthetic speech after training but remain awake should show a reduction in recognition performance for novel words after the waking retention interval, even though this was a shorter retention interval than the 12 hours previously investigated in Fenn, Nusbaum, & Margoliash (2003). While both the nap and wake groups showed evidence of loss between Posttest1 and Posttest2 (see Figure 2.4), we believe that this loss in the two groups arises from different sources. In the case of the nap group, the observed decrement in performance may be explained by sleep inertia, while in the case of the wake group, the loss in performance may be best explained by a waking retention interval interfering with unconsolidated learning. Sleep inertia has been found to last from anywhere from 0 min (i.e., if a fire alarm goes off) to 4 h after waking and is known to impair cognitive function (Jewett, Wyatt, Riz-De Cecco, Khalsa, Dijk, & Czeisler, 1999). This variation is thought to arise due to differences across individuals in what sleep stage they were awoken from and their sleep propensity upon going to sleep (Trotti, 2017;

Hilditch & McHill, 2019). As such, it is reasonable that we may be observing effects stemming from sleep inertia at Posttest2, given that this test was given only 30 min after waking.

The interpretation that sleep inertia is responsible for the decrement in performance between Posttest1 and Posttest 2 for the nap group is further bolstered by a return to initial post-training performance at Posttest3 for those with the opportunity to nap as presumably effects of sleep inertia have dissipated by this time point. In the wake condition, however, the loss is maintained into Posttest3, which is consistent with the view that experience during waking retention has had an adverse effect on unconsolidated learning. The results for those in the nap group are similar to findings observed for a full night of sleep (Fenn, Nusbaum, & Margoliash, 2003; Fenn, Margoliash, & Nusbaum, 2013). As such we hold that the data presented here represent the first demonstration that a 90-min daytime nap opportunity — the length of an average NREM-REM sleep cycle (Carskadon & Dement, 2005) — can have the same basic effect as a full night of sleep on generalized perceptual learning. By comparison, participants who did not nap continued to show performance loss. Thus, these results demonstrate that napping does consolidate generalized perceptual learning once one takes into consideration the effects of sleep inertia. While we hold that sleep inertia is the most parsimonious source of the decrement in learning for those that napped that is found between Posttest1 and Posttest2, it is also possible that consolidation is not immediately made manifest during sleep but develops over waking time following sleep. In our previous human (Brawn, Fenn, Nusbaum, & Margoliash, 2008, 2010) and bird (Brawn, Nusbaum, & Margoliash, 2013) studies, performance testing following sleep was not proximate to when participants woke up. Thus, evidence of consolidation always occurred after an unspecified waking interval following waking. The present study demonstrates that consolidation may not immediately present and may need to develop over the subsequent waking period. Further work is

therefore needed to know if the decrement we see between Posttest1 and Posttest2 for the nap group is related to the slow accrual of sleep consolidation during waking retention or sleep inertia. To the degree that sleep consolidation does take time to accrue, it will be important to know whether this phenomenon is specific to a nap (fewer sleep cycles than in a night's sleep) or if it is a hallmark of consolidation more generally. These questions are relevant to understanding the time-course of consolidation and the nature of the mechanisms that support it.

While our experiments cannot distinguish between leading sleep consolidation hypotheses, they do suggest certain conclusions about the time-course of consolidation in models such as synaptic downscaling (Tononi & Cirelli, 2014) and Complementary Learning Systems (CLS, e.g., McClelland, McNaughton, & O'Reilly, 1995; Born & Wilhelm, 2012). For example, the synaptic homeostasis hypothesis proposes that (wakeful) learning potentiates synapses, while sleep preferentially downscales synapses irrelevant to the learned information (Tononi & Cirelli, 2014). Progressive synaptic downscaling occurs during NREM, and it is suggested (though not explicitly stated) that this takes place over the course of an entire night of sleep (Tononi & Cirelli, 2006; Bellesi, Riedner, Garcia-Molina, Cirelli, & Tononi, 2014). This prompts the question: how much synaptic downscaling is required to solidify learning? This suggests that consolidation occurs during sleep whereas the present evidence suggests that at the end of a sleep cycle (nap) consolidation has not yet occurred. Further, the present results demonstrate that for generalized synthetic speech learning, a full night of sleep is not necessary — given a typical average 90-minute sleep cycle (Carskadon & Dement, 2005), the present results suggest that one sleep cycle is sufficient for consolidation of learning. In contrast, in systems consolidation models like CLS, memories are solidified during transfer from fast-learning to slower-learning memory stores (e.g., from hippocampus to cortex, e.g., McClelland, McNaughton, & O'Reilly, 1995; Born & Wilhelm,

2012). Similarly, this model proposes that consolidation occurs during sleep cycles whereas the present results suggest that consolidation is not established until during the subsequent waking period.

The current results demonstrate that a daytime nap protects generalized learning of new acoustic-phonetic mappings, allowing participants to apply learning to new words. There is scant prior work on the impact of a nap on consolidating generalized perceptual learning. One study found that perceptual learning of motion direction detection in one direction generalized to a second direction (McDevitt, Rokem, Silver, & Mednick, 2014). Another study investigated the efficacy of exposure therapy in arachnophobic participants and found that learning (defined as a reduction of the fear response to a novel spider) generalizes better after sleep than wake (Pace-Schott, Verga, Bennett, & Spencer, 2012). Other generalization studies find that either a nap or a full night of sleep promotes insight and creativity (Wagner, Gais, Haider, Verleger, & Born, 2004; Cai, Mednick, Harrison, Kanady, & Mednick, 2009), enhances abstraction in a pattern sequence understanding task (Durrant, Taylor, Cairney, & Lewis, 2011), and assists performance of a visual categorization task governed by an implicit rule (Djonlagic, Rosenfeld, Shohamy, Myers, Gluck, & Stickgold, 2009). One difference between the present work and these other studies is that consolidation in the prior work could reflect generalization due to explicit inferences from declarative learning of specific episodes. In these prior cases, participants had explicit conscious access to the stimuli encountered during training and could have made inferences by generalizing consciously from them to the new situations. This cannot be true for the perceptual learning of synthetic speech. Due to categorical perception (e.g., Pisoni, 1978), listeners cannot access the auditory properties that underlie the linguistic categories that are perceived. Thus, this is a clear

case in which a nap consolidates generalized perceptual learning when learning and consolidation of the exemplars themselves cannot account for the learning.

Our results also demonstrate that the source of interference responsible for the loss seen in the wake groups (in-lab and out-of-lab), is likely not due to auditory experience, as the pattern of loss was similar for awake participants who were exposed to speech outside the lab and those without this exposure. One possibility is that the loss of learning seen in the wake condition between Posttest1 and Posttest2 is due to reengagement of cognitive resources to another task. Generalized perceptual learning has been argued to be an active cognitive process that requires working memory and attentional resources (Heald & Nusbaum, 2014). Under this view interference does not stem from exposure to new auditory experiences but a change in how cognitive resources are used to maintain labile generalized perceptual learning previous to consolidation. Another possibility is that the loss of learning that we see in the wake condition between Posttest1 and Posttest2 arises via a decay function as a consequence of time simply lapsing after the conclusion of learning (Arthur, Bennett, Stanush, & McNelly, 1998). Future work is needed to understand the source of the loss, as this will be important to understand the role that consolidation is playing in its recovery.

The lack of an objective (physiological) sleep measure precludes relating specific features of sleep and the sleep cycle to consolidation from a nap. Nap subjects self-reported that they slept for some time during the 90-minute sleep period, as we have done in past studies (e.g., Fenn, Nusbaum, & Margoliash, 2003; Fenn, Margoliash, & Nusbaum, 2013; Brawn, Fenn, Nusbaum, & Margoliash, 2008, 2010). Additionally, subjects did not immediately respond to a knock at the door and only woke up at the time of lights on. However, the differences in performance between wake and nap groups suggest that the intervention of a 90-minute nap opportunity on the whole is

what is driving the results. Further research is needed to connect this variability to potential sources or mechanisms (e.g., differences in time spent in various stages of sleep; Nusbaum, Uddin, Van Hedger, & Heald, 2018). In other words, future research using polysomnography to quantify the macro- (sleep staging) and micro-architecture (individual components of various stages of sleep, such as sleep spindles or K-complexes) of sleep, are imperative to understand how sleep specifically supports this type of memory consolidation.

In conclusion, the present study is the first to demonstrate that the opportunity for a 90-minute nap is sufficient to consolidate generalized perceptual learning of speech. While it has been demonstrated that a full night of sleep consolidates generalized perceptual learning (e.g., Fenn, Nusbaum, & Margoliash, 2003; Fenn, Margoliash, & Nusbaum, 2013), the potential of shorter naps to consolidate this type of learning was previously unexplored. As we found that the nap group retained almost the same amount of learning that sleep groups did in our previous research (Fenn, Margoliash, & Nusbaum, 2013), this suggests that, although the nap in our current study is much shorter than a night's sleep, there is very similar consolidation given comparable learning across the studies. This then leads to the question of whether certain micro- or macro-architecture overnight/during a nap is predictive of the amount of benefit toward auditory perceptual learning derived from a period of sleep. Prior work suggests that naps containing REM sleep preferentially aid rote procedural memory, while naps containing slow wave sleep assist rote declarative memory (Plihal & Born, 1997), however, this dichotomy is likely an oversimplification. Future experiments might examine the polysomnographic correlates of this type of sleep-dependent consolidation to better understand the mechanism underlying this effect, and to identify potential reasons that some individuals derive greater benefit during sleep.

CHAPTER 3: DO WE REPLICATE BEHAVIORAL EFFECTS?

3.1 Introduction

While the study outlined in Chapter 2 demonstrated that the opportunity for a 90-minute day-time nap is sufficient to produce a consolidation effect, we can still obtain more nuanced information regarding whether the exact length of the nap is predictive of performance. As a result, we collected another dataset similar to the Chapter 2 dataset with a few methodological changes.

The various changes that occurred were (1) the addition of a test right before napping, to determine how much subjects lost over the first retention period and to then subsequently quantify the amount recovered immediately after a nap opportunity, and (2) the presence of an EEG cap on their head during napping (we wanted to make sure that the presence of the cap would not prevent them from being able to consolidate their learning).

3.2 Materials and Methods

3.2.1 Participants

We recruited 63 participants from the University of Chicago community. Prior to recruitment, subjects were screened for the following inclusion criteria: (1) age 18-35, (2) native English speaker, (3) no head injuries that still cause cognitive symptoms, history of stroke, seizures, neurological disorders, (4) no history of speech or hearing problems, (5) no history of a language based learning disorder, (6) not an abuser or alcohol or caffeine, (7) regularly gets at least 6 hours of sleep, (8) not currently on any psychoactive or sleep-affecting medications. Of the 63 subjects successfully recruited, 4 were subsequently excluded because they did not complete both sessions in their entirety.

Participants had an average mean age of 22.3 (SD = 3.07, range = 18-33, 41 female, 18 male). 55 were right-handed, and 4 were left-handed. All participants spoke English as a primary language with no history of speech or hearing disorders. There were no significant age ($t(50.19)$ = -0.15, p = 0.88) or gender ($X^2_{(1, N=59)}$ = 1.65, p = 0.20) differences between the groups. Participants were randomly assigned to nap (N = 32) or to remain awake for a matched period of time (N = 27).

3.2.2 Procedure: Visits

Subjects came in for two sessions. Visit 1 preceded Visit 2 by a minimum of two days, with Visit 1 lasting approximately 45 minutes and Visit 2 lasting approximately 12 hours. The main purpose of Visit 1 was to prepare subjects for Visit 2, including explaining the procedure, conducting a hearing test, and giving them instructions on to-do items to execute in between Visit 1 and Visit 2.

3.2.3 Visit 1 Procedure

During Visit 1, subjects were consented to participate and completed a hearing test. We administered Etymotic ER120-HHT Home Hearing Test, which assesses hearing sensitivity across various frequencies which cover both high-frequency sounds and frequencies of human speech (500Hz − 8,000 Hz). It assists in detecting potential hearing loss by evaluating a subject's hearing at various intensity levels. Intact healthy hearing was operationalized as being able to identify frequencies of tones at 20dB or less. If a subject did not have audiometric curves which constituted having intact healthy hearing, we did not have the subject come back in for Visit 2.

Subjects were additionally given several instructions to follow in the days leading up to Visit 2: (1) to ensure that they get at least 6 hours of sleep, (2) to abstain from alcohol, drugs,

nicotine, and other psychoactive/sleep-affecting substances in the days leading up to Visit 2, (3) to maintain a sleep log, and (4) to wear a ActiGraph watch. These instructions were given to ensure that subjects were sleeping an adequate amount leading up to Visit 2, and that they were not taking any substances that could affect the quality of their sleep.

3.2.4 Visit 2 Procedure

Subjects arrived at the laboratory in the morning (9:00) to have a BrainVision 64-channel slim-electrode gel-based electroencephalography (EEG) net placed on their scalp (see Figure 3.1). We ensured that subjects were not wearing makeup nor other facial products that would interfere with electrode impedances and ensured that their hair was fully dry prior to placing the net. In the case that subjects were wearing makeup, subjects were instructed to wash their face prior to us additionally using an alcohol wipe and NuPrep solution to lightly exfoliate the areas of the face where the EEG/EOG electrodes would have contact. In the case that subjects did not come in with completely dry hair, they were given a hair dryer to fully dry their hair.

Electrodes FT9 and FT10 were removed from their original spots on the cap, as they were used as electrooculography (EOG) electrodes for this experiment. Prior to placement, the areas of the face that these eye electrodes would be placed were lightly abraded with an alcohol wipe and NuPrep solution. We then applied Ten20 conductive paste to these electrodes and secured them with tape to the participants' left and right upper cheek respectively, approximately two centimeters laterally, and below the outer edges of the subjects’ eyes. EEG preparations were considered finished once impedances were measured to be below 10kOhms using BrainVision Pycorder.

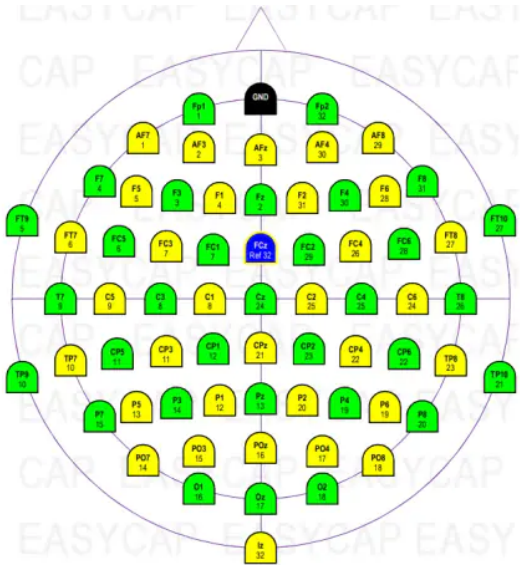

Figure 3.1. Electrode placement montage for BrainVision's actiCAP-slim 64-channel electrode cap. Electrodes FT9 and FT10 were removed from the cap and manually placed on the cheeks for EOG measurement.

At 10:00, subjects completed a Pretest, Training, and Posttest in our perceptual learning task. This testing and training period lasted approximately 45 minutes − 1 hour. Tests in this study possessed 80 unique monosyllabic words each, with the training containing 300 unique words. During tests, subjects were asked to listen to these words, one at a time, and then transcribe what they perceived the word to be on a computer keyboard. After they entered their response, the next word was presented to them; there was no feedback given during the perceptual learning tests.

During training, 300 words were presented in 6 blocks, with 50 words per block. During each training block, participants would hear a monosyllabic synthetic speech word, transcribe what they heard by typing their response, and then were subsequently given feedback in the form of hearing the same word a second time paired with the orthographic form of the word displayed on the computer screen. After finishing each 50-word block, participants were allowed to take a short break before proceeding with the rest of the training.

Subjects then had an in-lab break until 14:15, where they were allowed to perform whatever tasks they pleased (e.g., completing homework, talking on a phone call, watching television, eating, etc.), as long as they did not nap during the break. At approximately 14:15, we placed additional peripheral electrodes for the upcoming polysomnography (PSG) recording. PSG was collected to ensure that all subjects who were assigned to the wake condition indeed remained awake for the entirety of the 15:00 − 16:30 time block, while all subjects who were assigned to the nap condition indeed slept for some portion of the 15:00 − 16:30 time block. Placement followed AASM guidelines (Berry, Brooks, Gamaldo, Harding, Marcus, & Vaughn, 2012). We prepped the subject's chin (for the positive and negative electromyography (EMG) channels) and clavicle (for the ground EMG electrode) with an alcohol wipe and NuPrep gel. Ten20 paste was applied to the electrodes before they were secured to the skin using surgical tape. An illustration of placement of PSG electrodes can be found below in Figure 3.2.

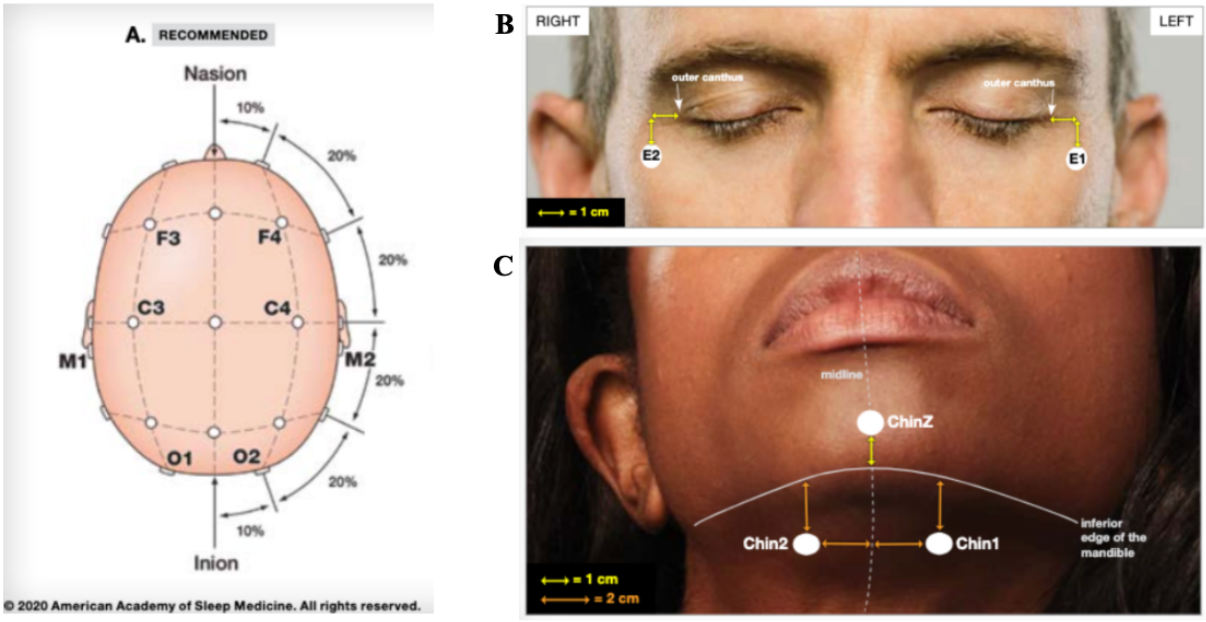

Figure 3.2. Placement of EEG, EOG, and EMG electrodes for polysomnography recording. A. An image illustrating the placement of EEG electrodes during polysomnography. This electrode placement follows the International 10-20 System. B. The placement of electrodes for EOG recording. C. The placement of electrodes on the chin for EMG recording.

At 14:30, subjects were retested on the perceptual learning task (Posttest2), in order to quantify the amount of performance lost after a period of time away from the task, as well as how much performance improved after a bout of sleep. At approximately 14:50, we prepped our subjects for the PSG recording which would be taking place from 15:00 − 16:30 (during the sleep/wake block). Details regarding PSG acquisition can be found below in the *Polysomnography Recording Procedure* section below.

After the PSG recording was terminated at 16:30, chin EMG electrodes were then removed and an alcohol wipe was used to wipe away any residual electrode paste. Subjects were then given about 30 minutes to allow nap subjects to recover from sleep inertia by sitting up prior to undergoing the next perceptual learning block.

Subjects were retested on the perceptual learning task at 17:00 (Posttest3), where they listened to and transcribed a new set of 80 unique monosyllabic synthetic speech words, and had another in-lab free period until 21:00. Again, subjects were allowed to perform any task that they wanted to in the lab, as long as they did not nap during this time. Subjects then performed their final Posttest at 21:00 (Posttest4), again listening to and transcribing 80 unique words. After finishing the experiment, the EEG net was removed, subjects were compensated for their time and were then allowed to leave the laboratory. The timeline of events can be visualized below with Figure 3.3.

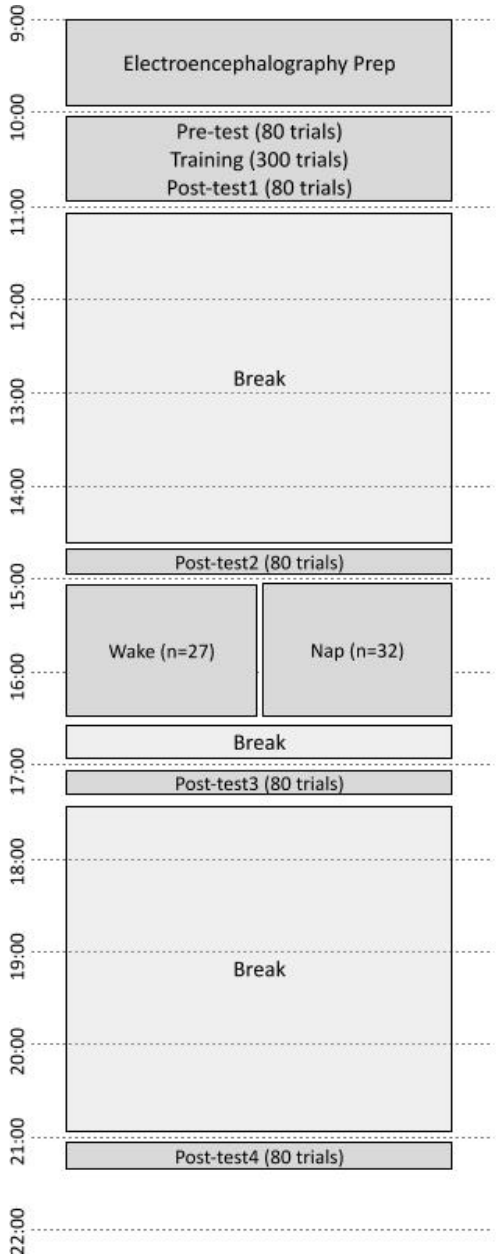

Figure 3.3. Timeline of perceptual learning tests and training over the course of the experiment. Pretest, Training, and Posttest1 began at 10:00. Subjects then performed Posttest2 at approximately 14:30 after an in-lab break. Subjects either napped or remained awake from 15:00 to 16:30, then performed Posttest3 at 17:00, and Posttest4 at 21:00 after another in-lab break.

3.2.5 Stimulus Presentation

Stimuli used during the perceptual learning task were all monosyllabic words, produced by the speech synthesizer *rsynth* (Klatt, 1980). A full list of words presented during test and training

blocks can be found at this GitHub repository: https://github.com/katie-reis/PLPSG/. Stimuli were delivered with a mean RMS amplitude of 66.5 dB via Etymotic ER-3C Insert Earphones. Stimuli were presented using a MATLAB script that made use of the *psychtoolbox* package, a package that is frequently used for stimulus presentation given its precise, low-latency sound playback capabilities. The requisite files for experiment presentation can be found in this GitHub repository: https://github.com/katie-reis/PLPSG/tree/main/Stimuli%20Presentation. After the subject pressed enter to lock in their typed response, the next stimulus was presented after 500 ms (with a -100 ms to +100 ms jitter).

3.2.6 Polysomnography Recording Procedure

At 14:50, we measured impedances using BrainVision Pycorder. Impedances for electrodes used in our PSG analysis (F3, F4, C3, C4, O1, O2, M1, M2, LOC, ROC) were below 10 kOhms, while impedances for all other electrodes were accepted to be below 20 kOhms. While we recorded from all 64 EEG electrodes during our PSG recording, we only used F3, F4, C3, C4, O1, O2, M1, M2, LOC, and ROC for sleep scoring.

After impedances were measured, a PSG recording was started using BrainVision Recorder. We then performed the standard AASM biocalibration procedure to ensure the accuracy and reliability of our PSG measurements, determining for each individual the electrophysiological responses that were evoked for all of the delineated physical maneuvers (Berry, Brooks, Gamaldo, Harding, Marcus, & Vaughn, 2012). For example, biocalibration allowed us to understand whether subjects were alpha producers (as approximately 10% of individuals do not produce alpha rhythms during resting state EEG due to individual variability in thalamocortical network activity or other benign factors; Niedermeyer & da Silva, 2005), the EOG signals for their vertical and horizontal

eye movements, the signals associated with teeth grinding, etc. A display montage that followed AASM filtering guidelines (Berry, Brooks, Gamaldo, Harding, Marcus, & Vaughn, 2012) was used in BrainVision Recorder so that the results of biocalibration could be read live. Below are the commands that were given to subjects:

1. "Keep your eyes open for 30 seconds, you may blink during this time."
2. "Close your eyes for 30 seconds."
3. "Look up and down 5 times."
4. "Look left and right 5 times."
5. "Blink 5 times."
6. "Clench your jaw for 5 seconds."
7. "Simulate a snore or hum for 10 seconds."

After biocalibration, subjects were then given earplugs and instructed to get comfortable on their pillow. When the clock struck 15:00, we turned off the lights (only for nap subjects), closed the door to the room (for all subjects), and a marker was added to the PSG recording that was labeled "lights off" (only for nap subjects) or "door closed" (only for wake subjects), indicating the beginning of the sleep/wake block. Approximately half of the subjects had the opportunity to take a daytime nap ($N = 32$), while half of the subjects remained awake ($N = 27$) and were only permitted to conduct silent activities (e.g., watching a silent movie with the captions on, reading, writing, etc.). During this period of time, we collected EEG and polysomnography (PSG) to (1) sleep score nap subjects using American Academy of Sleep Medicine staging criteria, and (2) to have an objective measure of wakefulness for our wake subjects, for the entirety of the 90-minute period.

Later, at 16:30, a marker was added to the PSG recording that was labeled "lights on" (for nap subjects) or "door opened" (for wake subjects). After the subject was successfully woken up/told that their wake block was over, the recording was then stopped.

3.2.7 Data Analysis: Behavioral Task

For the perceptual learning words, accuracy was based on phonetic transcription, using the International Phonetic Alphabet IPA using a Midwestern dialect, without penalizing for spelling errors. If a subject's response matched a homophone found in the IPA, their response would be marked as correct. For instance, if the stimulus being presented was "lean", and the subject typed in the response "lien", this would be scored as correct. However, if the subject instead typed in "leans", this would be scored as incorrect due to the added phoneme.

Afterwards, responses were checked for syntax errors. For example, including an extra space after a correctly spelled response would be automatically scored as incorrect; spelling "lean" as "lean " with a space after the letters would be marked as incorrect, thus requiring that that word be re-scored as correct. Additionally, scorers were blind to the experimental condition when scoring.

3.3 Results

One goal of the present study was to replicate our prior study and to better understand the effect of a nap on learning. That is, the question was focused on how a nap consolidates learning. In prior research using this perceptual learning task there is individual variability in whether and how much learning occurs (e.g., Fenn, Margoliash, & Nusbaum, 2013; Reis, Heald, Uddin, Fenn, & Nusbaum, 2023). To increase sensitivity to the effects of a nap, we excluded from analysis any subjects who did not show learning, or in other words, subjects who performed worse on Posttest1 than at pretest. As a result, we excluded 3 subjects (all of whom were assigned to the nap condition) from the dataset.

Afterwards, we confirmed that our subjects learned as a function of our training using a 2 × 2 repeated measures ANOVA, with test (Pretest, Posttest1) as a within-subjects factor and condition (Nap, Wake) as a between-subjects factor. We failed to find a main effect of condition ($F(1, 54) = 0.30$, $p = 0.59$), suggesting that there was not a significant difference in performance between the nap and wake groups. Importantly however, participants showed significant improvement as a result of training, as revealed by a significant main effect of test ($F(1, 54) = 269.3$, $p < 0.001$), with an average improvement of 15 percentage points (Pretest percent correct: $M = 29$, $SE = 1$; Posttest1 percent correct: $M = 43$, $SE = 1$).

Additionally, by adding in Posttest2, our 14:30 test, we were able to approximate the amount of performance lost after our first waking retention interval for our subjects. As we expected, our subjects demonstrated significant loss after this waking retention interval — we used a 2 x 2 repeated measures ANOVA, with test (Posttest1, Posttest2) as a within-subjects factor and condition (Nap, Wake) as a between-subjects factor. Participants showed a significant main effect of test ($F(1, 54) = 38.97$, $p < 0.001$), with average loss of 6 percentage points (Posttest1 percent correct: $M = 43$, $SE = 1$; Posttest2 percent correct: $M = 37$, $SE = 1$).

The trajectory of performance is consistent with what we see with prior research (Reis, Heald, Uddin, Fenn, & Nusbaum, 2023). Performance continues to decrease from Posttest1 over the course of the day for wake subjects (percent loss $M = 4$, $SE = 2$ from Posttest1 to Posttest2; percent loss $M = 2$, $SE = 1$ from Posttest2 to Posttest3; percent loss $M = 0.4$, $SE = 2$ from Posttest3 to Posttest4).

Nap subjects, on the other hand, experience initial performance loss after time spent away from the task (percent loss $M = 8$, $SE = 1$ from Posttest1 to Posttest2), followed by marginally significant recovery in performance after a period of napping (percent improvement $M = 2$, $SE =$

2 from Posttest2 to Posttest3; $t(28) = 1.49$, $p = 0.07$). Nap subjects then demonstrate non-significant loss from Posttest3 to Posttest4 after another period of time spent away from the task (percent loss $M = 1$, $SE = 1$; $t(28) = -1.07$, $p = 0.29$). Figure 3.4 includes the spread of performance across tests for both groups.

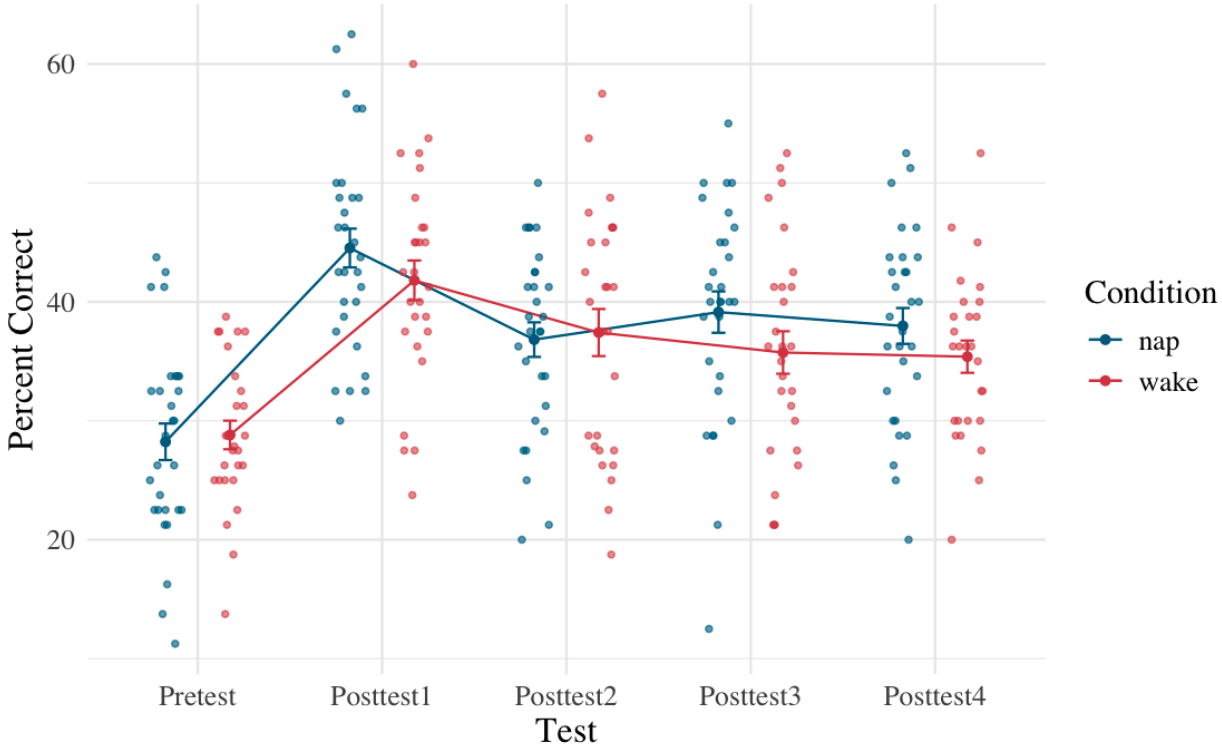

Figure 3.4. Performance of the nap and wake groups across all tests (Pretest, Posttest1, Posttest2, Posttest3, Posttest4). Total number of trials for each test = 80. Error bars depict standard error of the mean.

3.4 Discussion

In the Chapter 2 dataset, we asserted that there was no evidence of immediate recovery of performance in our nap subjects. However, it is hard to quantify recovery if there was no measurement of initial loss, and whether immediate post-nap performance has improved since immediate pre-nap performance. In other words, while performance change from Posttest1 (post

learning) to Posttest2 (post nap) was similar for both of our groups, it could be that if we had retested these subjects prior to the intervention, that a difference in the trajectory of performance would become apparent for our subjects (i.e., wake subjects showing continuous loss, nap subjects showing loss then recovery).

This analysis allowed us proof-of-concept that the changes that we made to our perceptual learning paradigm did not inhibit our subjects from being able to consolidate their learning after a bout of sleep. In other words, the presence of an EEG cap during napping did not prevent them from being able to consolidate their learning. Additionally, this PSG recording allowed us to have an objective sleep measure. To add, the addition of another test right before napping allowed us to confirm that subjects were performing significantly worse on this task after a waking retention interval and allowed us to quantify the amount of recovery that nap subjects experienced immediately after their nap.

As anticipated, there was no significant difference between our two groups in terms of how their performance changed from Pretest to Posttest1 (our measure of learning) and from Posttest1 to Posttest2 (our measure of loss). This was expected as these blocks occurred prior to our intervention (the sleep/wake interval), and since we were sampling from the same population, we would expect that our subjects would be matched on learning and on loss. In line with previous literature (Heald, Van Hedger, Veillette, Reis, Snyder, & Nusbaum, 2022; Reis, Heald, Uddin, Fenn, & Nusbaum, 2023), we saw significant learning as a function of our training paradigm, and then significant loss after a period of time spent away from the task.

After our intervention, performance changes then bifurcated between our two groups. In other words, the pattern of performance change from Posttest2 to Posttest3 (our measurement of

recovery) is significantly different between the two groups; on average, performance improved for the nap subjects, while performance declined for the wake subjects.

Importantly, we see that the initial loss that wake subjects experienced from Posttest1 to Posttest2 (over the first waking retention interval), then continues over the course of the day. There is no offline consolidation period for these subjects that protects them against interference, rather this group on average performs worse from Posttest1 to Posttest2 (over the first waking retention interval), Posttest2 to Posttest3 (over the sleep/wake block), and then Posttest3 to Posttest4 (over the second waking retention interval). On the other hand, in line with previous overnight sleep study experiments (Fenn, Margoliash & Nusbaum, 2013; Fenn, Nusbaum & Margoliash, 2003), sleep allowed subjects to recover performance and then stabilize performance against future decay.

We must consider the variance in performance across tests, however. While our nap subjects overall show recovery of performance, there are still subjects who demonstrate loss in performance from Posttest2 to Posttest3. It may be that factors such as sleep inertia and other sleep history/sleep related measurements may explain variation in performance pre-nap to post-nap.

CHAPTER 4: SLEEP HISTORY'S IMPACT ON PERFORMANCE

4.1 Introduction

After demonstrating that a short day-time nap is sufficient to produce a consolidation effect similar to a night of sleep in Chapter 2, it is important to understand how a short daytime nap may be different in some ways than a full night's sleep. On the one hand the conditions of the nap, such as it being a novel sleeping environment, may influence the quality of the nap. On the other hand, it may be that the need for a nap (homeostatic sleep pressure) and the effectiveness of a brief nap may depend on conditions outside the testing environment and may be measured by sleep history. As a result, we are interested in further understanding the impacts of sleep inertia and sleep pressure on performance, to see if these factors explain some of the variance in performance. Additionally, sleep history may impact the neural signature of one's daytime nap, thereby later influencing their behavioral performance.

Sleep inertia, which impairs cognitive function, can last from zero to four hours after waking, depending on factors like sleep stage at awakening and individual sleep propensity. A related process, homeostatic sleep pressure, builds throughout the day and is influenced by prior sleep duration and circadian rhythms. See Discussion, Chapter 2.

While homeostatic sleep pressure and circadian drive regulate the sleep-wake cycle, their relationship is grounded in the concept that the intensity of sleep inertia is influenced by the level of homeostatic sleep pressure. For example, individuals who experience higher levels of sleep pressure, through either attaining insufficient sleep the night before, or having irregular sleep patterns, will experience greater sleep inertia upon waking after their next bout of sleep.

A similar concept, sleep deprivation, arises via poor sleep quality, irregular sleep patterns, and/or insufficient sleep duration. As such, being sleep deprived results in sleep pressure not being

fully alleviated and therefore augments the accumulation of sleep pressure over the course of the day, impairing cognitive function and alertness.

Additionally, to compensate for this deficit, the sleep architecture of subsequent sleep periods is altered — in other words, individuals will experience "recovery sleep" or "slow wave sleep rebound". There is also research that demonstrates that naps containing slow wave sleep (SWS) increase sleepiness immediately after waking (Brooks & Lack, 2006), and that waking up out of SWS increases the effects of sleep inertia (Tassi & Muzet, 2000); however, SWS is important for reducing sleep pressure, as with increased time spent awake, SWS duration increases (Dinges, 1986).

As a result, we carried out several sets of analyses to determine: (1) how sleep history impacts performance on tests prior to our manipulation (prior to the nap/quiet wakefulness period) across *all* subjects, (2) whether sleep history is correlated with time spent in slow wave sleep, (3) whether there are PSG markers of sleep deprivation/sleep pressure during the nap period that predict performance (e.g., whether (a) nap subjects who spend more time in slow wave sleep demonstrate lower Posttest3 performance/ recovery than subjects who spend less, (b) whether subjects who are woken up during slow wave sleep experience performance decrements, etc.), and (4) whether sleep history can explain any variance in performance for wake subjects for the final two posttests.

4.2 Materials and Methods

The materials and general procedure for this experiment were identical to those described in Chapter 3. Please refer to Chapter 3: Materials and Methods for detailed descriptions of participant recruitment, screening criteria, experimental tasks, and polysomnography setup.

Additional methods relevant to this chapter (questionnaires) are described below. Please see Chapter 5 for details regarding polysomnography and sleep scoring methods.

4.2.1 Procedure

In addition to the procedure outlined in Chapter 3, after completing Posttest1, subjects filled out short questionnaires which can be found below in Appendices A − D. The Demographics Questionnaire was used to collect age and gender information from our subjects. The Language Experience Questionnaire was used to confirm that our subjects were all Native English Speakers. The Sleep Log was completed by subjects prior to coming in for Visit 2 and describes how they slept the two nights prior to Visit 2, and collects information such as when they went to bed, woke up, how many times they woke up during the night, how long they were awake during the night, and the total time they were asleep for. Finally, the Sleep Questionnaire collects information regarding how the subject *usually* sleeps. For example, it will collect information such as when they normally go to bed/wake up, how long they usually sleep for ("*Usual TST"*), etc. As such, the Sleep Questionnaire was used to have a baseline of the subject's usual sleep length, so that sleep deficit measurements could be calculated later by comparing a subject's usual sleep length to how much sleep they received during the nights leading up to Visit 2.

4.2.2 Data Analysis: Questionnaires

The information that we are interested in for this analysis is (1) how many hours did the subject sleep the night before coming in ("*Night Before TST"*), (2) how many hours the subject slept the two nights before coming in, averaged ("*Average TST"*), (3) how much the subject deviated from their normal total sleep time the night before coming in (*"Immediate Restriction"*), and the difference between the subject's normal total sleep time and their average sleep/night over

the prior two nights leading up to Visit 2 (*"Cumulative Restriction"*). Negative values of Immediate Restriction and Cumulative Restriction signify that the subject slept fewer hours than they normally do. Immediate Restriction and Cumulative Restriction are calculated as indicated below:

$$Immediate\ Restriction\ =\ Usual\ TST\ -\ Night\ Before\ TST$$
$$Cumulative\ Restriction\ =\ Usual\ TST\ -\ Average\ TST$$

4.3 Results

Nap and wake subjects were matched on the amount of sleep that they got the night before coming in for Visit 2 (Night Before TST; $t(45.77) = 0.33$, $p = 0.71$, sleep: $M = 7.31$, $SE = 0.14$, wake: $M = 7.22$, $SE = 0.22$) and the average TST over the two nights leading up to Visit 2 (Average TST; $t(44.75) = -0.91$, $p = 0.37$, sleep: $M = 7.43$, $SE = 0.14$, wake: $M = 7.66$, $SE = 0.22$). We also wanted to ensure that our two groups were matched on the amount of sleep restriction that they were coming in with. Using Welch's two-sample t-tests, we find that nap and wake subjects were matched on the amount of Immediate Restriction that they were under ($t(38.26) = 0.72$, $p = 0.48$, sleep: $M = -0.99$, $SE = 0.12$, wake: $M = -1.19$, $SE = 0.25$) and on the amount of Cumulative Restriction that they were under ($t(36.65) = -0.49$, $p = 0.63$, sleep: $M = -0.88$, $SE = 0.11$, wake: $M = -0.75$, $SE = 0.24$). See Figure 4.1 below.

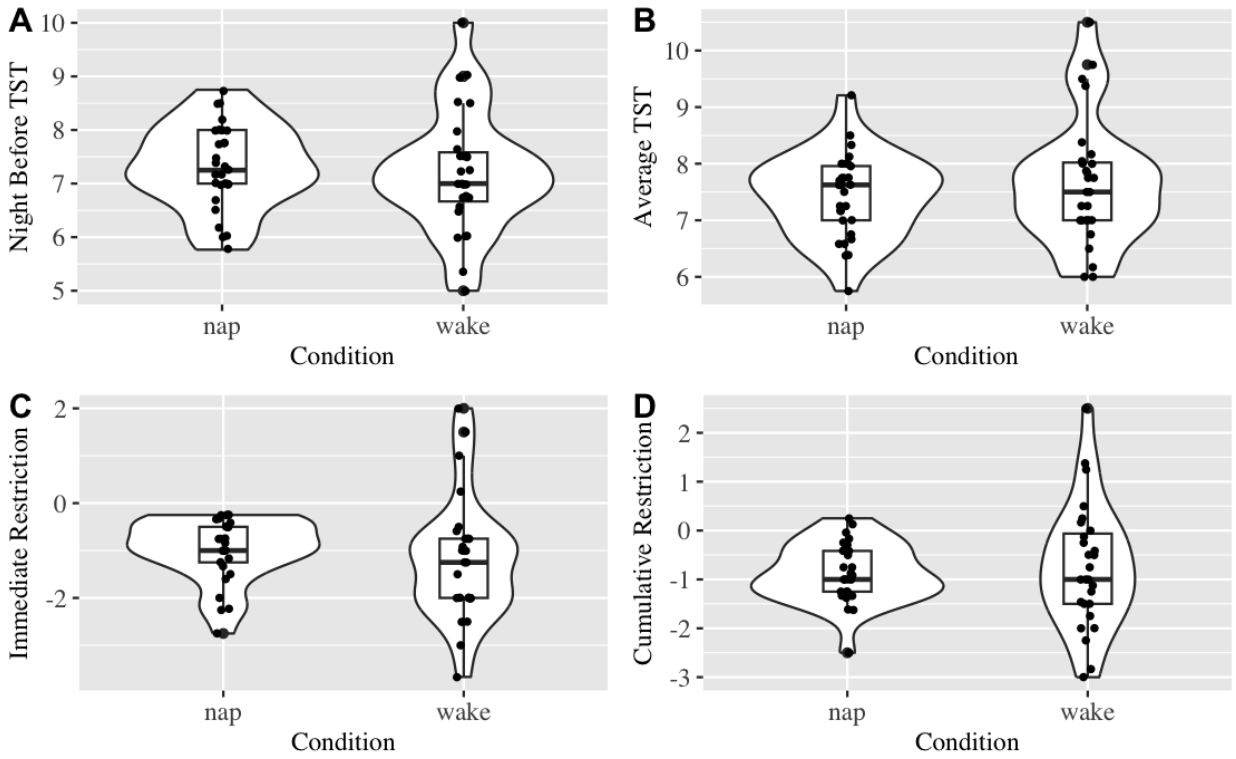

Figure 4.1. Distribution of various sleep history measures for nap and wake subjects. (A) Distribution of Night Before TST, or the hours of sleep subjects slept the night before Visit 2, (B) distribution of Average TST, or the average number of hours slept across the two nights prior to Visit 2, (C) distribution of Immediate Restriction, or the difference between what subjects usually sleep, and how much they slept the night before Visit 2 (Usual TST - Night Before TST), and (D) distribution of Cumulative Restriction, or the difference between what subjects usually sleep and how much they slept across the two nights leading up to Visit 2 (Usual TST - Average TST).

Additionally, we performed a series of Welch's two-sample t-tests between nap and wake subjects, demonstrated that there was no significant difference in Stanford Sleepiness (SS) Scale score collected immediately prior to Pretest ($t(53.95) = 0.79$, $p = 0.43$, nap $M = 3.03$, $SE = 0.23$, wake $M = 2.78$, $SE = 0.23$) and Posttest3 ($t(51.89) = 0.31$, $p = 0.76$, nap $M = 2.86$, $SE = 0.18$, wake $M = 2.78$, $SE = 0.21$), however there was a significant difference between groups on Stanford Sleepiness Scale score collected immediately prior to Posttest2 ($t(53.72) = 2.38$, $p = 0.02$, nap $M = 3.03$, $SE = 0.21$, wake $M = 2.37$, $SE = 0.19$) and Posttest4 ($t(53.74) = -2.42$, $p = 0.02$, nap $M = 2.52$, $SE = 0.20$, wake $M = 3.22$, $SE = 0.21$).

These results suggest that nap and wake subjects differ significantly on their subjective assessment of sleepiness prior to Posttest2 and Posttest4. The question was whether sleepiness ratings affect performance. As a result, we investigated the impact of subjective sleepiness prior to Posttest2 on performance loss, using a multiple linear regression predicting loss from Stanford Sleepiness prior to Posttest2 ("SS_Posttest2") and condition. This model was only marginally significant ($F(3, 52) = 2.49$, $p = 0.07$). The individual predictors were examined further and indicated that SS_PrePosttest2 ($\beta = -2.20$, $t(52) = -1.84$, $p = 0.07$) and the interaction between SS_PrePosttest2 and condition ($\beta = 3.65$, $t(52) = 1.95$, $p = 0.06$) just missed significance. However, we did find an effect of condition on loss ($\beta = -13.44$, $t(52) = -2.52$, $p = 0.01$), where individuals who were in the nap condition ($M = 8$, $SE = 1$) lost significantly more than those in the wake condition ($M = 4$, $SE = 2$) between Posttest1 and Posttest2. Given that our subjects knew whether they would be napping or staying awake after they completed Posttest2, this result suggests that subjects who are expecting to nap may be more likely to disengage from the task or relax cognitive effort, potentially leading to greater immediate performance loss. In contrast, wake participants may have remained more cognitively alert during Posttest2, knowing they would not have an opportunity to sleep. This anticipatory mindset could influence attentional state and motivation, contributing to the observed differences in loss.

We also further investigated the impact of SS prior to Posttest4 on performance, using a multiple linear regression predicting maintenance (Posttest4 − Posttest3) from SS prior to Posttest4 ("SS_PrePosttest4") and condition. The overall model was not near significant in fit ($F(3, 52) = 0.31$, $p = 0.82$). The individual predictors also did not reach significance: SS_PrePosttest4 ($t(52) = -0.68$, $p = 0.50$), condition ($t(52) = -0.64$, $p = 0.52$), SS_PrePosttest4 * Condition ($t(52) = 0.86$, $p = 0.40$).

Given our prediction that subjects who sleep less prior to Visit 2 and/or get sleep that deviates from their normal sleep habits will perform worse on pretest, Posttest1, and Posttest2 compared to subjects who got more sleep during the nights leading up to Visit 2/ got sleep which is more consistent with their normal sleep habits, we went on to perform a series of simple linear regressions. We found that the amount of sleep that subjects received the night before an experiment did not significantly predict performance on pretest ($\beta = 0.96$, $t(54) = 0.93$, $p = 0.36$), however, it did significantly predict performance on Posttest1 ($\beta = 2.51$, $t(54) = 2.09$, $p = 0.04$), and Posttest2 ($\beta = 2.92$, $t(54) = 2.39$, $p = 0.02$).

We were also interested in investigating the effect of prior sleep length on one's ability to learn as a function of training (learning), and the amount lost over the first waking retention interval (loss). We found that sleep duration the night before Visit 2 (Night Before TST) was not significantly predictive of the amount learned (Posttest1 − Pretest; $t(54) = 1.63$, $p = 0.11$), and of the amount lost (Posttest2 − Posttest1; $t(54) = -0.39$, $p = 0.70$).

Given research that shows that the negative effects of sleep restriction are compounding (e.g., Van Dongen, Maislin, Mullington, & Dinges, 2003), we examined how performance on these tests is affected by sleep across the prior nights leading up to the experimental session. By performing a series of simple linear regressions we found that the average amount of sleep that subjects received over the prior two nights before the experiment (Average TST) did not significantly affect performance on pretest ($t(54) = 0.75$, $p = 0.46$), Posttest1 ($t(54) = 1.49$, $p = 0.14$), and Posttest2 ($\beta = 2.39$, $t(54) = 1.89$, $p = 0.06$).

Additionally, we found that the average amount of sleep that subjects received over the prior two nights before the experiment did not significantly relate to the amount learned ($\beta = 1.07$,

$t(54) = 1.09$, $p = 0.28$) and lost over the waking retention interval ($\beta = -0.54$, $t(54) = -0.50$, $p = 0.62$).

Those with irregular sleep patterns and/or fewer hours of sleep will show evidence of increased homeostatic sleep pressure with more time spent in SWS during their daytime nap. However, there are several competing hypotheses for how recovery sleep may impact performance on our task. It may be that those who spend more time in SWS during their nap perform better on Posttest3 and Posttest4 as there are theories (such as the Synaptic Homeostasis Model, Tononi & Cirelli, 2014) that implicate SWS duration in memory consolidation. However, it may be that those who spend more time in SWS are more likely to experience stronger and longer effects of sleep inertia, leading to performance on our generalized perceptual learning task to be worse at Posttest3 (in line with Hilditch & McHill, 2019). It could also be that we will find that performance is negatively impacted by sleep restriction, regardless of SWS rebound/ time spent in SWS, therefore negatively impacting performance on Posttest3 and Posttest4 for nap subjects who were previously sleep deprived.

As such, we were first interested in seeing whether those who slept fewer hours the night(s) before the experiment indeed experienced more SWS during their daytime nap. Using linear regression, we found that total sleep time the night before Visit 2 (Night Before TST) did not predict SWS duration during the daytime nap ($t(27) = 0.07$, $p = 0.95$). Similarly, we found that the average sleep duration across the two nights prior to Visit 2 (Average TST) did not predict SWS duration during the daytime nap ($t(27) = -0.81$, $p = 0.43$).

We tested whether the percentage of time spent in SWS was influenced by these sleep history measures, as there was variability in the amount of time subjects spent asleep overall during their afternoon nap opportunity, and percentage of time spent in SWS is a more informative

measure of the density of SWS during this bout of sleep. Using linear regressions, we found that total sleep time the night before Visit 2 did not significantly predict percentage of time spent in SWS during the daytime nap ($t(27) = 0.40$, $p = 0.69$), and average sleep duration across the two nights leading up to Visit 2 did not significantly predict percentage of time spent in SWS during the daytime nap ($t(27) = -0.32$, $p = 0.75$).

However, it might not be that total sleep time for the night/prior two nights is the best predictor of sleep architecture. Rather, it may be that being in a sleep deficit is a predictor of sleep architecture. Using linear regressions, we find that Immediate Restriction was not predictive of the duration of N3 during the daytime nap ($t(27) = -0.92$, $p = 0.36$), nor was it predictive of the percentage of time spent in N3 during the daytime nap ($t(27) = -1.11$, $p = 0.28$). However, Cumulative Restriction was predictive of both the duration of N3 during the daytime nap ($\beta = -8.40$, $t(27) = -2.25$, $p = 0.03$), and of the percentage of time spent in N3 during the daytime nap ($\beta = -10.65$, $t(27) = -2.30$, $p = 0.03$). In other words, if subjects received less sleep than usual in the nights leading up to the experiment, then they spent more time in SWS. See Figure 4.2 below.

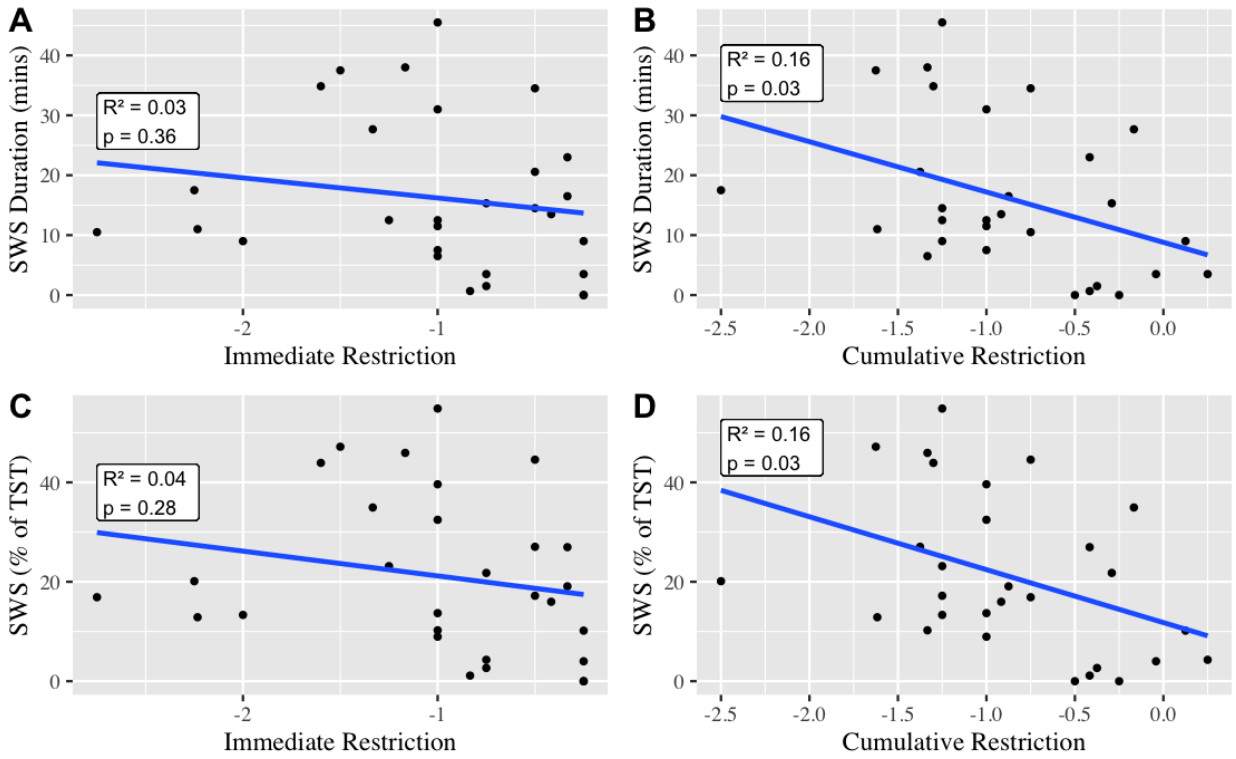

Figure 4.2. The relationship between Immediate and Cumulative Sleep Restriction and SWS Duration and SWS percentage of total sleep time.

In summary, individuals who have prior sleep restriction spend significantly more time in SWS. Given that prior sleep restriction is predictive of both SWS duration during the daytime nap, and the percentage of time spent in SWS, this suggests the amount of time that individuals proportionally spent in other stages of sleep should also be affected. As a result, we tested whether the time spent in N1, N2, and REM were affected by sleep restriction.

Immediate Restriction was significantly related to the duration of N1 during the daytime nap ($\beta = 4.56$, $t(27) = 2.34$, $p = 0.03$), as well as the percentage of time spent in N1 during the daytime nap ($\beta = 5.20$, $t(27) = 2.05$, $p = 0.05$). However, Cumulative Restriction was not related to either the duration of N1 during the daytime nap ($t(27) = 1.19$, $p = 0.24$), nor of the percentage of time spent in N1 during the daytime nap ($t(27) = 1.17$, $p = 0.25$). In other words, if nap subjects

experienced more sleep restriction the night before Visit 2, then they spent less time in N1. See Figure 4.3 below.

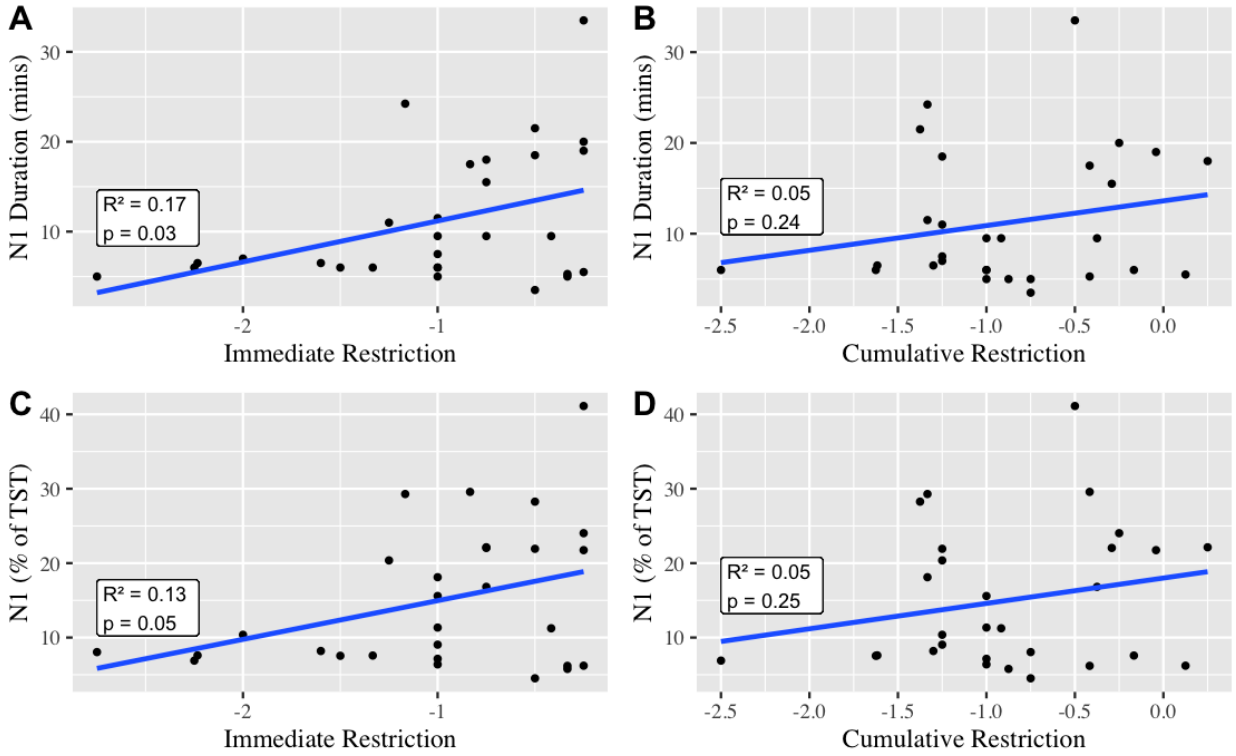

Figure 4.3. The relationship between Immediate and Cumulative Sleep Restriction and N1 Duration and N1 percentage of total sleep time.

In contrast, Immediate Restriction was not predictive of the duration of N2 during the daytime nap ($t(27) = 1.50$, $p = 0.15$), nor was it predictive of the percentage of time spent in N2 during the daytime nap ($t(27) = 0.47$, $p = 0.64$). However, Cumulative Restriction was marginally related to both the duration of N2 during the daytime nap ($\beta = 5.39$, $t(27) = 1.96$, $p = 0.06$), and of the percentage of time spent in N2 during the daytime nap ($\beta = 7.28$, $t(27) = 1.96$, $p = 0.06$). Nominally, if nap subjects experienced more cumulative sleep restriction, then they spent less time in N2 during their daytime nap. See Figure 4.4 below.

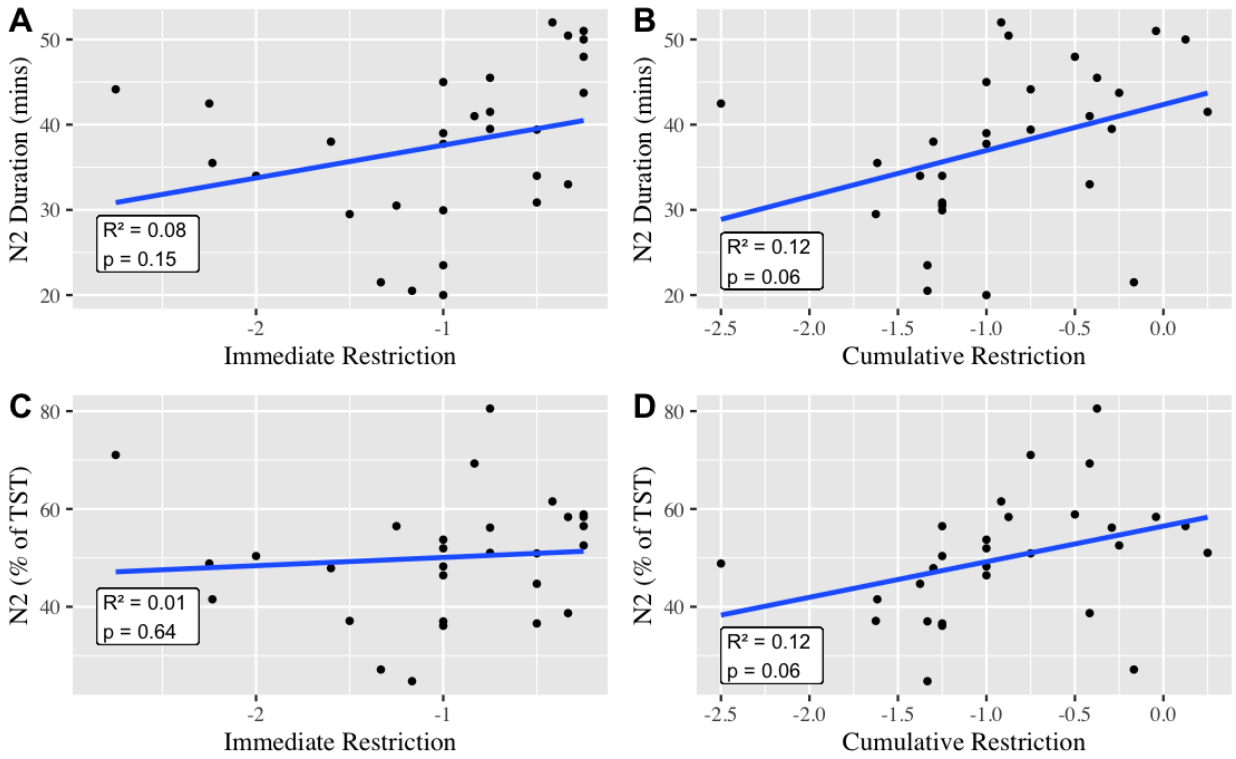

Figure 4.4. The relationship between Immediate and Cumulative Sleep Restriction and N2 Duration and N2 percentage of total sleep time.

However, Immediate Restriction was not predictive of the duration of REM during the daytime nap ($t(27) = -0.34$, $p = 0.74$), nor was it predictive of the percentage of time spent in REM during the daytime nap ($t(27) = -0.49$, $p = 0.63$), and Cumulative Restriction was also not predictive of both the duration of REM during the daytime nap ($t(27) = 0.09$, $p = 0.93$), nor of the percentage of time spent in REM during the daytime nap ($t(27) = -0.01$, $p = 0.99$). See Figure 4.5 below.

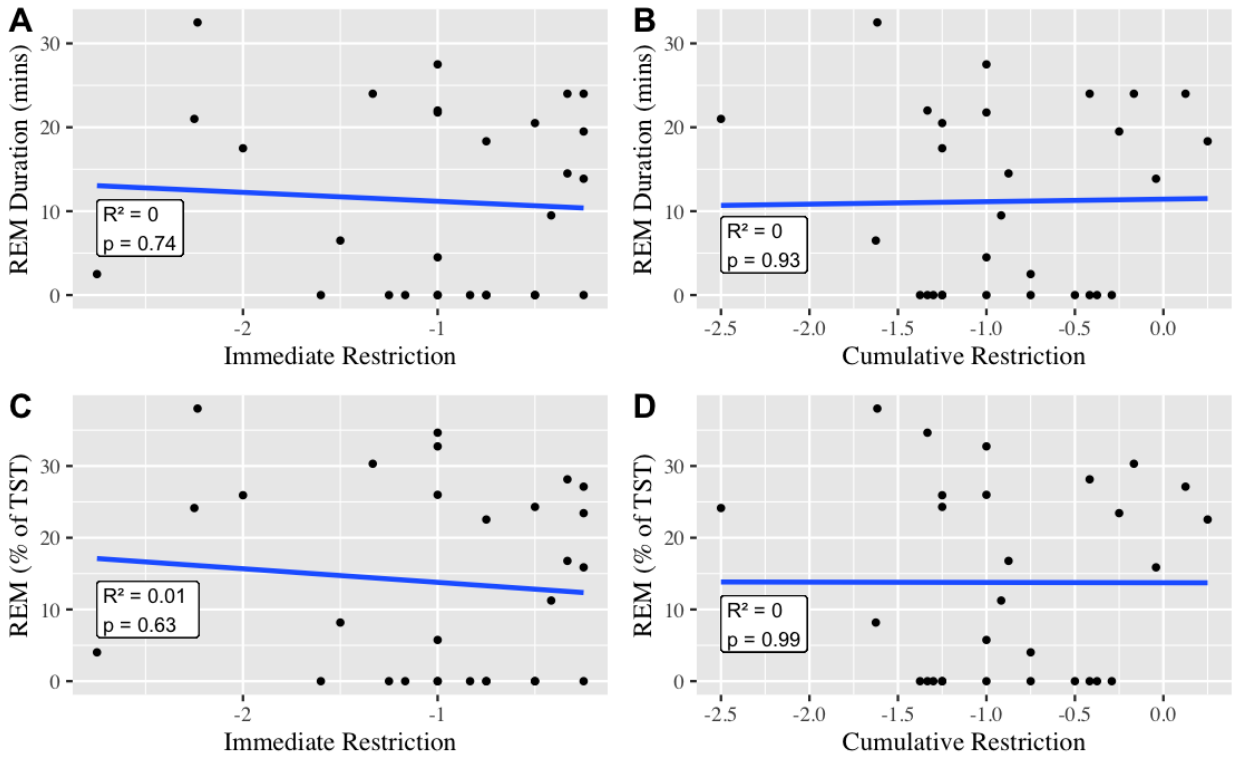

Figure 4.5. The relationship between Immediate and Cumulative Sleep Restriction and REM Duration and REM percentage of total sleep time.

Was recovery affected by the amount of restriction that subjects were under as well as their previous total sleep duration? A linear regression was conducted to examine the effects of prior night total sleep time (Night Before TST), condition (wake vs. control), and their interaction on recovery sleep. The overall model was not statistically significant, ($F(3, 52) = 1.43$, $p = 0.25$), the predictors were not significant (Night Before TST: $t(52) = -0.15$, $p = 0.88$, Condition: $t(52) = -0.76$, $p = 0.45$), and the interaction was also not significant (Night Before TST x Condition: $t(52) = 0.52$, $p = 0.60$). We then ran a linear regression model instead with Average TST, and find that the overall model was also not significant ($F(3, 52) = 1.34$, $p = 0.27$), the predictors were not significant (Average TST: $t(52) = 0.18$, $p = 0.86$, Condition: $t(52) = -0.36$, $p = 0.72$), and the interaction was not significant (Average TST x Condition: $t(52) = 0.13$, $p = 0.90$). We then ran a

linear regression model instead with Immediate Restriction, where the overall model was also not significant ($F(3, 52) = 1.94$, $p = 0.13$), and the predictors were not significant (Immediate Restriction: $t(52) = -0.47$, $p = 0.64$, Condition: $t(52) = -0.37$, $p = 0.72$, Immediate Restriction x Condition: $t(52) = 1.03$, $p = 0.31$). We lastly ran a linear regression model with Cumulative Restriction as a predictor variable, and the overall model again was not significant ($F(3, 52) = 1.72$, $p = 0.17$), and the predictors were not significant (Cumulative Restriction: $t(52) = -0.09$, $p = 0.93$, Condition: $t(52) = -0.91$, $p = 0.37$, Cumulative Restriction x Condition: $t(52) = 0.59$, $p = 0.56$). See Figure 4.6 below.

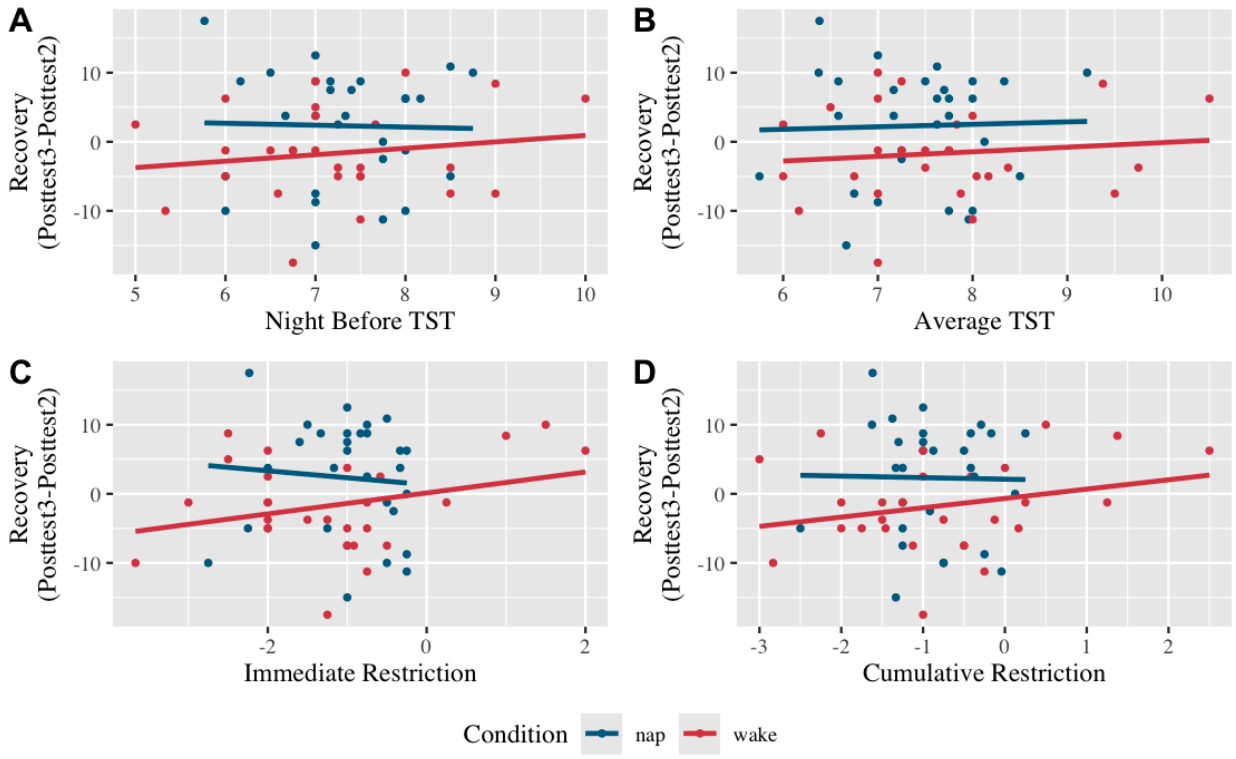

Figure 4.6: The relationship of various sleep history measures with recovery of performance (Posttest3 − Posttest2 score), including: (A) total sleep time the night before Visit 2, (B) the average sleep duration of the two nights leading up to Visit 2, (C) how much the previous night of sleep deviated from their normal sleep length, (D) how much the average of their prior two nights deviated from their normal sleep length.

While previous sleep was not related to recovery, does the stage that subjects woke up from affect performance? Based on prior literature, those who wake up out of SWS should be more likely to experience lingering sleep inertia (Tassi & Muzet, 2000), which can negatively affect performance on cognitive tasks (Jewett, Wyatt, Ritz-De Cecco, Khalsa, Dijk, & Czeisler, 1999). A Welch's two-sample t-test was conducted to compare recovery sleep between participants whose last sleep epoch was SWS and those whose last sleep epoch was not SWS, within the nap condition. There was a significant difference in recovery sleep between the two groups ($t(26.38) = 4.19$, $p < 0.001$). Specifically, participants who ended their nap in SWS showed greater recovery ($M = 9$, $SE = 1$) compared to those who did not end their nap in SWS ($M = 1$, $SE = 2$). See Figure 4.7 below.

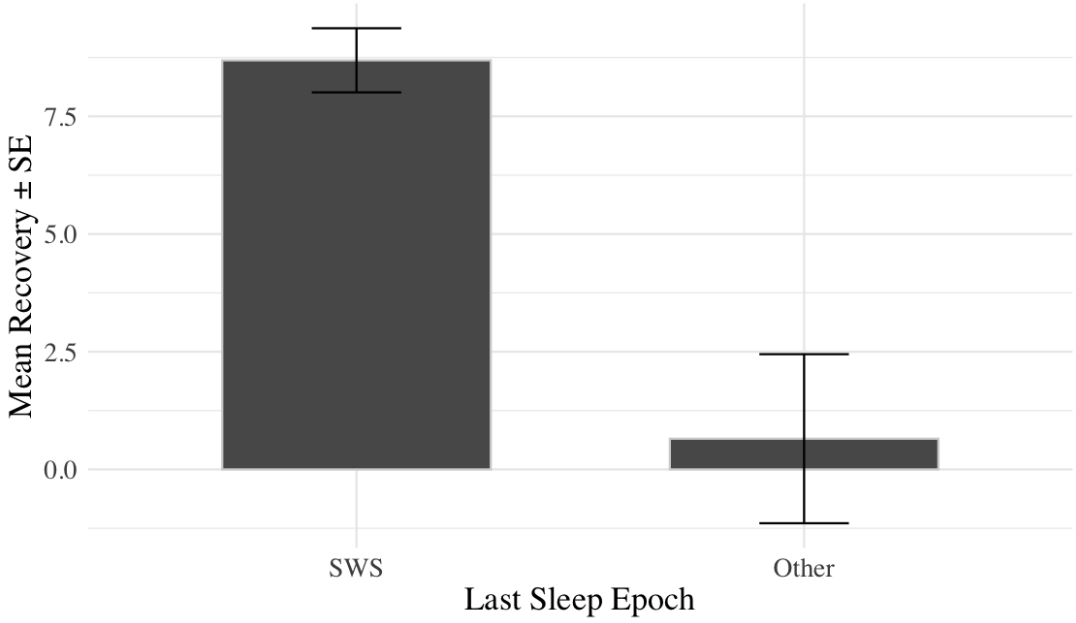

Figure 4.7: Waking up out of slow wave sleep in relationship to recovery of perceptual learning performance.

4.4 Discussion

This chapter examined how prior sleep behavior impacts learning, memory consolidation, and recovery sleep architecture. Our initial hypothesis — that sleep restriction or deviation from

normal sleep habits would be associated with poorer performance across cognitive tasks — was partially supported. Specifically, total sleep time the night before the experimental session did not significantly predict performance on the pretest, but it did significantly predict performance on both Posttest1 and Posttest2. This suggests that while immediate sleep may not influence baseline performance, it may play a role in the ability to retain or consolidate new information after training.

Interestingly, there was not a significant relationship between prior night sleep duration and either learning (Posttest1 − Pretest) or loss (Posttest2 − Posttest1). Nor did sleep across the two nights before significantly predict test performance or learning/loss metrics, though the relationship between average sleep across the prior two nights and Posttest2 performance approached significance ($p = 0.06$). These null effects challenge the idea that short-term variations in sleep quantity alone robustly shape learning dynamics, and suggest a more complex interaction between sleep quality, timing, and individual differences in baseline sleep need.

When investigating how prior sleep restriction influences sleep architecture during a daytime nap, we found that cumulative — but not immediate — deviations from a subject's typical sleep pattern (i.e., "Cumulative Restriction") significantly predicted both the duration and percentage of time spent in slow-wave sleep (SWS). This finding supports models of homeostatic sleep regulation (e.g., Borbély, 1982) suggesting that sleep pressure accumulates over time and is discharged through deeper sleep stages such as SWS. The lack of a relationship between absolute sleep duration and SWS metrics further underscores the importance of considering individual baselines when studying sleep behavior.

Investigation of other sleep stages revealed more nuanced results. Immediate Restriction is predictive of the amount of time spent in N1 and the percentage of time spent in N1 during a daytime nap, whereas Cumulative Restriction is marginally predictive of the amount of time spent

in N2 and the percentage of time spent in N2 during a daytime nap. Both Immediate Restriction and Cumulative Restriction are not predictive of REM duration nor percentage of time spent in REM during a subsequent daytime nap. This points toward evidence of a homeostatic response: the brain is prioritizing deeper, restorative sleep (SWS) faster and more efficiently when sleep debt is higher. In other words, in states of sleep restriction, the brain compresses lighter stages and dives faster into SWS to recover. N1 and N2 are essentially bypassed or shortened to prioritize the high-recovery stage of SWS. Additionally, while there have been previous accounts of REM rebound (Dement & Kleitman, 1957), these results suggest that REM sleep doesn't show compensation in a nap setting. Indeed, this supports the idea that REM is more circadian-regulated than homeostatically regulated (Achermann, Borbély, Kryger, Roth, & Dement, 2017), and REM rebounding may require nighttime circadian timing given that REM has a preferred timing window of early morning hours (Carskadon & Dement, 2005); in a daytime nap, it's less likely to appear robustly especially when sleep pressure is high and SWS takes precedence.

These results are also consistent with the idea that N1 is more reactive to immediate sleep pressure, where higher sleep pressure from one bad night of sleep may push the brain to move more rapidly out of N1 into deeper stages, whereas N2 is more stable and restorative than N1, but less so than SWS, meaning that if a person accumulates sleep debt over multiple nights then the body may begin to reallocate time towards SWS and away from N2. Indeed, there is research that has demonstrated that with a single night of sleep deprivation leads to reductions in the amount of time spent in N1 sleep (Curcio, Ferrara, Pellicciari, Cristiani, & Gennaro, 2003), and when sleep is restricted to 4 hours per night over multiple consecutive nights, there is a significant decrease in N2 sleep duration (Banks & Dinges, 2007). This reduction in N2 sleep suggests that cumulative sleep debt leads to alterations in sleep architecture, with the body potentially sacrificing N2 sleep

in an attempt to preserve or enhance deeper sleep stages, such as slow-wave sleep (SWS), which are crucial for recovery and cognitive function.

Perhaps most intriguingly, we found that the sleep stage at awakening was a strong predictor of performance, with participants who awoke from SWS showing significantly greater recovery effects than those who did not. This result is somewhat counterintuitive, given literature suggesting that waking from SWS can be associated with sleep inertia and impaired performance (e.g., Tassi & Muzet, 2000). One possibility is that the restorative benefits of SWS outweighed the transient deficits associated with inertia, particularly in the context of our perceptual learning task. Additionally, it could be that for this sample, 30 minutes to recover from the effects of sleep inertia was sufficient, as we failed to find a difference in subjective sleepiness (Stanford Sleepiness Scale) results between groups prior to Posttest3.

Together, our findings support a growing body of work suggesting that individual differences in sleep behavior and sleep regulation significantly shape cognitive function. The fact that Cumulative Restriction had stronger effects than Immediate Restriction may have practical implications for individuals with chronically insufficient sleep. Moreover, the relationship between SWS rebound and task performance highlights the complex, sometimes paradoxical, role that sleep plays in memory and learning.

CHAPTER 5: NEURAL FEATURES OF CONSOLIDATION

5.1 Introduction

Much of the learning that individuals perform naturally on a day-to-day basis does not rely on explicit memorization but rather on a more flexible process of attention reorganization whereby one can generalize their initial learning to new situations. This type of learning can be considered a type of skill learning, as individuals are tasked with learning features of a talker and taking those learned features and applying it repeatedly to new stimuli. This type of learning, generalized learning, distinguishes itself from rote learning which describes successful memorization over one singular set of stimuli (Greenspan, Nusbaum, & Pisoni, 1988; Heald & Nusbaum, 2014; Heald, Van Hedger, Veillette, Reis, Snyder, & Nusbaum, 2022; Schwab, Nusbaum, & Pisoni, 1985).

Further, previous research has also demonstrated that there can be differences in sleep-dependent consolidation for generalized and rote learning — for example, that a night of sleep can restore performance of generalized perceptual learning but not rote learning (Fenn, Margoliash, & Nusbaum, 2013) and sleep also aids in stabilizing generalized perceptual learning (i.e., Fenn, Nusbaum, & Margoliash, 2003; Brawn, Fenn, Nusbaum, & Margoliash, 2008, Fenn, Margoliash, & Nusbaum, 2013). In other words, behaviorally it has been shown that after initially learning the task as a function of training, subjects are then prone to interference during time spent away from the task. However, sleep provides two consolidation effects: (1) it can restore any loss in performance seen after training, and (2) it allows that learning to be robust against future interference (i.e., Fenn, Nusbaum, & Margoliash, 2003; Fenn, Margoliash, & Nusbaum, 2013). Although we know that a daytime nap is sufficient for providing a consolidation effect for generalized auditory perceptual learning, it is still not understood what neural mechanisms might underlie the consolidation of generalized perceptual learning.

One approach to answering this question is to investigate whether the macrostructure of sleep (the stage progression of sleep) facilitates consolidation — specifically, whether time spent in certain sleep stages during a daytime nap influences the degree of performance improvement following sleep. Previous work suggests that sleep stages N2, SWS, and REM (McDevitt, Duggan, & Mednick, 2015) may contribute to the consolidation of perceptual learning, potentially supporting not just task improvement but also the ability to generalize learning to novel contexts. While brief daytime naps often consist primarily of lighter sleep stages, a 90-minute nap provides an opportunity for some individuals to experience a full sleep cycle, as typical cycles range from 70 to 120 minutes (Carskadon & Dement, 2005). Moreover, variations in sleep history (e.g., the amount of homeostatic sleep pressure they are under as a function of the quantity and quality of previous sleep) may influence an individual's likelihood of entering deeper sleep stages during a daytime nap. Notably, recent work has suggested that a daytime nap can offer consolidation benefits equivalent to a full night of sleep (Reis, Heald, Uddin, Fenn, & Nusbaum, 2023), reinforcing the importance of studying naps as a window into sleep-dependent learning. Thus, a key question is to what extent traditional sleep features — such as the duration of N2, SWS, or REM sleep — are predictive of post-nap performance improvements.

Prior research suggests that the microstructure (the dynamic events that occur during individual stages of sleep) of sleep, rather than/in addition to the macrostructure of sleep, may be correlated with consolidation. Theories of sleep consolidation have linked a particular feature of sleep called spindles (bursts of brain activity during non-REM sleep) to the process of consolidation because spindles are hypothesized to reflect transfer from early, fast encoding into memory in the hippocampus to regions in neocortex (Antony, Schönauer, Staresina, & Cairney, 2019; Antony & Paller, 2017). Specifically, spindles have been used to predict the amount of

improvement in memory or learning tasks after a bout of sleep — meaning that individuals who have more pronounced spindle activity may show greater gains in their ability to generalize perceptual skills (Antony & Paller, 2017). Additionally, spindles are hypothesized to assist in the integration of new information with existing cognitive frameworks (Tamminen, Payne, Stickgold, Wamsley, & Gaskell, 2010) and promote synaptic plasticity, a vital component of the memory consolidation process. One may argue that generalized perceptual learning relies on the brain extracting common patterns across experiences. If sleep spindles contribute to this process, they could be a mechanism for strengthening and restructuring memories in a way that promotes generalization. As a result, we aim to explore whether this microstructural component of the EEG during a nap can predict subsequent performance on our speech recognition task.

This investigation will test between multiple competing theories of memory consolidation and can point to the features of sleep that are particularly important for supporting generalized perceptual learning consolidation. For example, if we find that consolidation is correlated with spindle count, this would be consistent with models such as the Complementary Learning Systems Model (McClelland, 2013) and the Active Systems Consolidation Model (Rasch & Born, 2013), suggesting that spindles are acting as replay events, and allowing for our subjects' learning during our task to be integrated with existing cognitive structures. However, if we find that slow wave activity, or the duration of slow wave sleep is correlated with recovery, this would point towards the Synaptic Homeostasis Model (Tononi & Cirelli, 2014), which postulates that slow wave activity dependent synaptic downscaling needs to occur to prioritize only the relevant neural connections for learning. If the duration of REM correlates with performance, this would be evidence for the Dual Process Hypothesis (Plihal & Born, 1997) which correlates REM sleep duration with consolidation of procedural learning (as opposed to declarative memories). Finally,

if we find that both SWS and REM are important for this consolidation process, this would be in alignment with theories such as the Sequential Hypothesis (Giuditta et al., 1995) and demonstrate that both stages play an important role in the consolidation process of generalized perceptual learning, but that consolidation does not require repetition of NREM-REM cycles, as our subjects demonstrate consolidation during a short daytime nap. As such, each above theory lends itself to different predictions about what neural markers of sleep may be associated with consolidation of generalized auditory perceptual learning, and this investigation allows us to elucidate which of these theories offers the best account for consolidation of this type of learning.

5.2 Materials and Methods

The materials and general procedure for this experiment were identical to those described in Chapter 3. Please refer to Chapter 3: Materials and Methods for detailed descriptions of participant recruitment, screening criteria, experimental tasks, and polysomnography setup. Additional methods relevant to this chapter (EEG preprocessing procedure, and sleep scoring procedure) are described below.

5.2.1 Data Analysis: Electroencephalography Preprocessing Procedure

The polysomnographic data was first preprocessed during BrainVision Analyzer 2.1. Raw BrainVision files were loaded into BrainVision Analyzer 2.1, and the standard electrodes used for sleep scoring were selected (O1, O2, F3, F4, C3, C4, Fp1 (a left EOG channel placed on the bottom outer edge of the left eye), Fp2 (a right EOG channel placed on the bottom outer edge of the right eye), M1, M2, and EMG) as they make up the typical channel set for sleep scoring. O1, F3, and C3 were referenced to M2 (opposite mastoid), and O2, F4, and C4 were referenced to M1 (opposite

mastoid). Fp1 was referenced to the left EOG channel that was placed on the bottom outer edge of the left eye (FT9 was used), and Fp2 was referenced to the right EOG channel that was placed on the bottom outer edge of the right eye (FT10 was used).

After re-referencing, bandpass filters were applied, as dictated by the AASM. Specifically, a 0.3 − 35 Hz bandpass filter was applied to all EEG and EOG electrodes, and a 10 − 100 Hz bandpass filter was applied to the EMG electrodes (Berry, Brooks, Gamaldo, Harding, Marcus, & Vaughn, 2012).

Files were then exported to .edf format, to allow for easy importing to Sleep, a python-based software for visualizing, analyzing, and scoring polysomnographic recordings (Combrisson et al., 2017). After these .edf files were imported into Sleep, each 30-second epoch was sleep scored following AASM guidelines (Berry, Brooks, Gamaldo, Harding, Marcus, & Vaughn, 2012). .txt files with epoch-by-epoch scoring information were exported to provide sleep statistics. Details on how epochs are scored are provided in the section *Data Analysis: Sleep scoring.*

5.2.2 Data Analysis: Sleep Scoring

The following guidelines for sleep scoring are outlined by the Academy of Sleep Medicine (Berry, Brooks, Gamaldo, Harding, Marcus, & Vaughn, 2012). Sleep consists of the following stages: (1) W or wakefulness, (2) N1 (NREM 1) sleep, (3) N2 (NREM 2) sleep, (4) N3 (NREM 3) sleep or slow wave sleep (SWS), and (5) REM (rapid eye movement) sleep. Stages are scored in 30-second sequential epochs for the entirety of the recording, where each epoch is assigned a single stage. In the case that an epoch contains wakefulness as well as sleep, the stage is scored as wakefulness (W) if more than 50% of the epoch can be scored as W. In the case that two or more

stages of sleep exist in a given epoch, and the majority of the epoch is in a sleep stage, the sleep stage assigned to the epoch is the sleep stage that represents the largest portion of the epoch.

The stage of sleep assigned is dependent on the existence of various EEG frequencies. These frequencies include: (1) slow wave activity, which is a frequency of 0.5 − 2.0 Hz with a peak-to-peak amplitude of >75 microVolts, predominant in frontal electrodes, (2) delta wave activity, which is a frequency of 0 − 3.99 Hz, (3) theta wave activity, which is a frequency of 4 − 7.99 Hz, (4) alpha wave activity, which is a frequency of 8 − 13 Hz, and (5) beta wave activity, which is a frequency greater than 13 Hz.

An epoch is scored as W if there are (a) alpha over occipital channels, (b) eye blinks, (c) rapid eye movements while muscle tone is normal or high, and/or (d) horizontal eye movements. An epoch is scored as N1 if there are (a) slow eye movements, (b) vertex sharp waves, and/or (c) low-amplitude mixed-frequency (theta) EEG activity. An epoch is scored as N2 if there are (a) K-complexes and/or (b) sleep-spindles. An epoch is scored as N3 if there is slow wave activity for more than 20% of the epoch. An epoch is scored as REM if there are (a) rapid eye movements, (b) low chin EMG tone, (c) sawtooth waves (a pattern of trains of "serrated" 2 − 6 Hz waves maximal in amplitude over central channels and often appear before bursts of rapid eye movements), and/or (d) transient muscle activity. Once the first epoch of REM is identified, following epochs are also scored as REM until (1) a K complex or sleep spindle is identified in the absence of rapid eye movements, (2) an arousal followed by LAMF EEG and slow eye movements, or (3) a transition to stage W or N3. Also, once definite stage REM is identified, epochs prior to this epoch are *re-scored* as stage REM until (1) a K complex or sleep spindle is identified, or (2) an arousal.

After sleep scoring each 30 second epoch between lights out (15:00) and lights on (16:30), text representations of the resulting hypnograms were exported, and provided a single line for each

epoch with a code representing what stage the subject was in during each epoch of the recording. These text files were then imported into REM to calculate various sleep statistics, such as the number of minutes spent in each stage of sleep (N1 duration, N2 duration, SWS duration, REM duration), along with the percentage of time spent in each of these stages, as calculated below:

$$Percentage\ of\ sleep\ in\ N1\ =\ perN1\ =\ (\frac{N1}{TST})\times\ 100$$
$$Percentage\ of\ sleep\ in\ N2\ =\ perN2\ =\ (\frac{N2}{TST})\ \times\ 100$$
$$Percentage\ of\ sleep\ in\ SWS\ =\ perSWS\ =\ (\frac{SWS}{TST})\ \times\ 100$$
$$Percentage\ of\ sleep\ in\ REM\ =\ perREM\ =\ (\frac{REM}{TST})\ \times\ 100$$

To detect sleep spindles, we employed the open-source Python library YASA (Yet Another Spindle Algorithm; Vallat & Walker, 2021), which provides a fast, reproducible, and well-validated approach to spindle detection based on established neurophysiological criteria. YASA is widely used in sleep research and is designed to work with both high- and low-density EEG recordings.

YASA's detection pipeline begins by selecting NREM epochs, which were previously scored and labeled as either Stage N2 or Stage N3 (SWS). Sleep spindles are then identified within these epochs through a multi-step signal processing routine. First, the EEG data are band-pass filtered within the sigma frequency range, typically between 11–16 Hz, although these bounds can be adjusted depending on the research question or subject characteristics. This filtering isolates activity that falls within the frequency domain characteristic of sleep spindles.

After filtering, YASA applies a Hilbert transform to compute the analytic signal envelope of the filtered EEG. This step captures instantaneous amplitude fluctuations and helps reveal periods of elevated sigma-band activity. The algorithm then defines adaptive amplitude thresholds, usually based on the mean or median absolute amplitude of the entire recording or a reference

baseline. A typical detection threshold requires that the signal exceed two standard deviations above the mean envelope amplitude for a minimum duration of 0.5 seconds, and remain below a maximum duration of 2 seconds, consistent with the temporal definition of spindles in human sleep literature.

Each candidate spindle is subjected to a cycle-by-cycle analysis to verify the presence of genuine oscillatory structure. This means that the signal must complete a minimum of 3 full cycles within the spindle duration, ensuring that the event reflects rhythmic neural activity rather than transient artifacts or noise. YASA also uses zero-crossing methods and optional peak detection to validate these oscillatory properties and define precise onset and offset times.

In addition to binary detection (i.e., whether a spindle is present or not), YASA provides rich metadata for each event, including:

1. Start and end times
2. Duration (in seconds)
3. Peak frequency (within the sigma band)
4. Amplitude metrics such as peak-to-peak amplitude and RMS amplitude
5. Number of oscillatory cycles
6. Globality score, if multichannel data are available, indicating whether the spindle is localized or widespread across electrodes
7. Relative sigma power, often normalized by background EEG power

YASA's flexibility allows researchers to tune detection parameters, such as threshold levels, duration bounds, and the frequency range of interest. For this study, we limited detection of fast spindles to central EEG channels (C3, C4), where these spindles tend to be most prominent and reliably detected, and limited detection of slow spindles to frontal EEG channels (F3, F4) as these spindles are most prominent there (Mölle, Bergmann, Marshall, & Born, 2011).

Importantly, because theoretical frameworks suggest that spindles in different NREM stages may serve distinct roles in memory consolidation (e.g., Antony, Schönauer, Staresina, & Cairney, 2019; Antony & Paller, 2017), we quantified spindles separately for N2 and N3 sleep.

This approach allowed us to examine whether spindle activity during lighter sleep (N2) or deeper, slow-wave sleep (SWS) more strongly predicted memory performance after the nap. By parsing spindles by stage, we aimed to more precisely evaluate the contributions of each sleep process to post-nap memory recovery.

5.3 Results

5.3.1 Role of Spindles in Memory Consolidation

To test models that implicate spindles in memory consolidation, we performed a linear regression to test whether recovery is related to the number of spindles detected during sleep. We chose to individually test whether the number of spindles detected during N2, SWS, and N2 as well as SWS sleep were correlated with recovery.

Spindles during N2 sleep are often linked to the processing of declarative memories (facts, events). N2 sleep plays a role in the stabilization and consolidation of newly encoded information. Since spindles are thought to orchestrate communication between the hippocampus (which is active in learning and memory encoding) and the neocortex (which is involved in long-term memory storage), the number or density of spindles in N2 sleep could be an important predictor of how well newly learned information is consolidated into long-term storage (e.g., Antony, Schönauer, Staresina, & Cairney, 2019; Antony & Paller, 2017). However, we failed to find an effect of the number of spindles detected in N2 sleep on recovery ($t(27) = -0.19$, $p = 0.85$). See Figure 5.1 below.

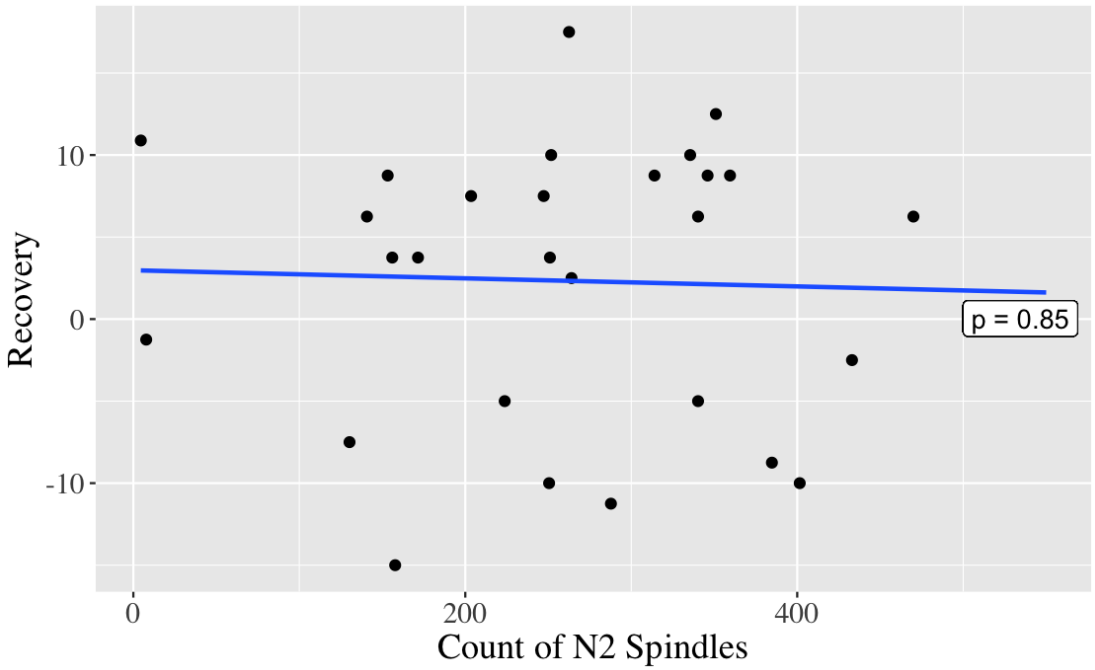

Figure 5.1. The relationship between the number of spindles detected during N2 sleep (fast spindles) and recovery of perceptual learning performance.

SWS sleep, particularly deep sleep (slow-wave sleep), is associated with synaptic downscaling and the redistribution of memory traces. Spindles in SWS have been shown to have a role in reorganizing memory traces that were previously processed during N2. In addition, SWS sleep might support the integration of these memories into existing cortical networks. Spindles here may promote the transfer of memories from the hippocampus to the neocortex, facilitating the long-term stabilization of memories. We, however, failed to find an effect of the number of spindles detected in SWS sleep on recovery ($t(27) = -0.13$, $p = 0.90$). See Figure 5.2 below.

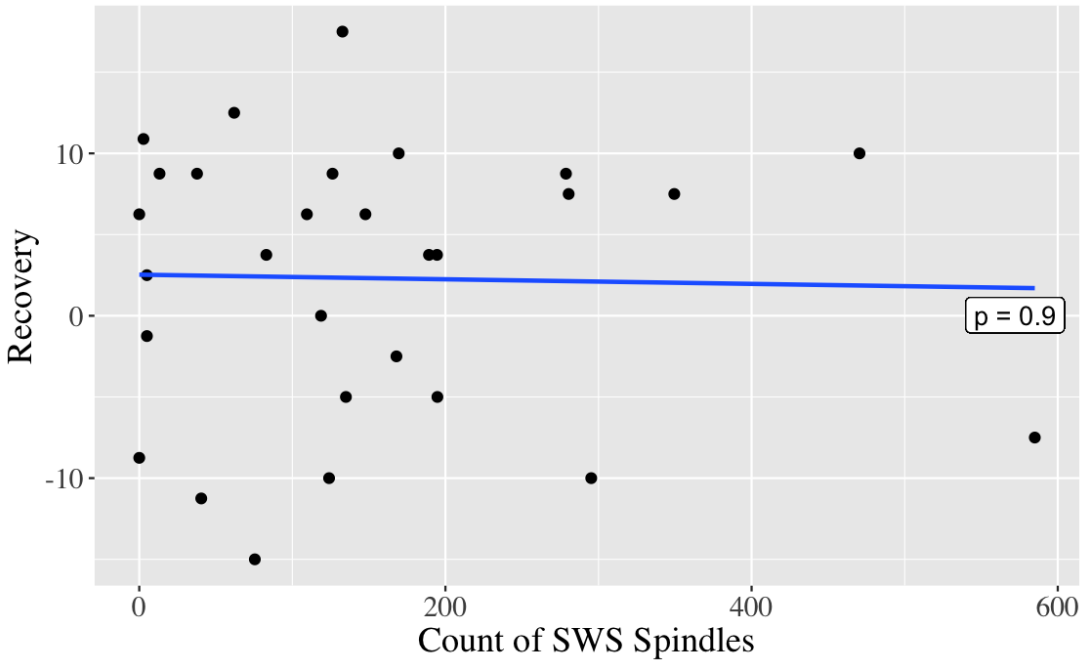

Figure 5.2: The relationship between the number of spindles detected during slow wave sleep (slow spindles) and recovery of perceptual learning performance.

By examining spindles in both N2 and SWS (see Figure 5.3), it is possible to assess how these spindles contribute to different aspects of the consolidation process — whether one stage is more involved in the transfer of information, while the other is more involved in memory stabilization, or whether they work in tandem to optimize memory consolidation. Additionally, it is possible that those who do not cycle through all stages of sleep (such as those who do not have the opportunity to experience any SWS spindles during their nap), behave differently from those who do. As a result, we created a model predicting recovery from the number of N2 spindles, the number of SWS spindles, whether the subject was a full cycler, and all two-way interactions. In addition to the overall model failing to reach significance ($F(6, 22) = 0.61$, $p = 0.72$), we failed to find any main effects (N2 spindles: $t(22) = 0.20$, $p = 0.85$, SWS spindles: $t(22) = 0.89$, $p = 0.38$, Full Cycler: $t(22) = 1.15$, $p = 0.26$) nor interaction effects (N2 spindles x SWS spindles: $t(22) =$ -

0.34, p = 0.74, N2 spindles x Full Cycler: $t(22)$ = -0.64, p = 0.53, SWS spindles x Full Cycler: $t(22)$ = -1.83, p = 0.08).

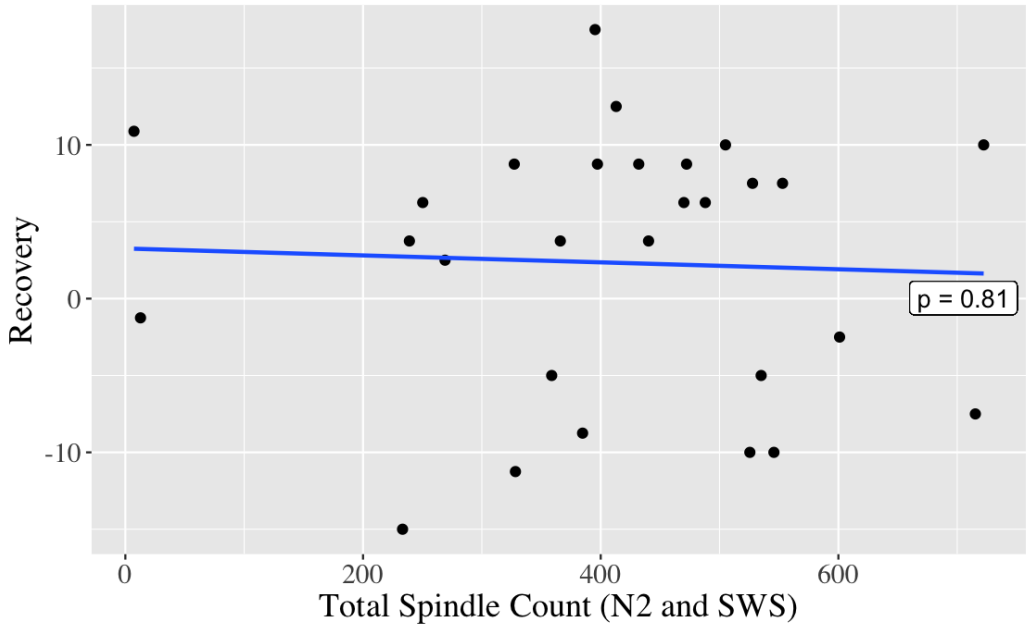

Figure 5.3. The relationship between the number of spindles detected during both N2 sleep (fast spindles) and slow wave sleep (slow spindles) and recovery of perceptual learning performance.

5.3.2 Role of Slow Wave Sleep in Memory Consolidation

We then tested whether the amount of time spent in slow wave sleep was predictive of recovery. Since synaptic homeostasis theory posits that slow wave sleep serves to downscale synaptic weights that are redundant and/or weak to bolster the signal-to-noise ratio for the most informative connections (Tononi & Cirelli, 2014), a longer duration of SWS sleep could potentially be associated with more significant synaptic pruning and, by extension, memory consolidation. However, using linear regression, we failed to find an effect of SWS duration on recovery scores in our nap subjects ($t(27)$ = 0.25, p = 0.81).

We examined the percentage of time spent in SWS sleep during a bout of sleep may be more sensitive to the distribution of sleep stages during a given sleep episode. Since the synaptic

homeostasis theory suggests that the intensity of deep sleep during certain periods might be critical for consolidating memories, if a particular bout of sleep (e.g., the first or last sleep cycle) has a high percentage of SWS sleep, it could reflect a higher likelihood of synaptic scaling and memory consolidation during that phase. Additionally, the percentage of time spent in SWS sleep might be a more precise indicator of the relative effectiveness of the synaptic downscaling process during different phases of the sleep cycle. Regardless, we failed to find an effect of the percentage of time spent in SWS on recovery as well ($t(27) = 0.21$, $p = 0.83$).

5.3.3 Role of Rapid Eye Movement Sleep in Memory Consolidation

Given that our task is a procedural learning task, it is important to test theories that implicate REM sleep in the consolidation of procedural learning, such as the Dual Process Hypothesis (Plihal & Born, 1997; Plihal & Born, 1999). The total time spent in REM sleep could predict how much consolidation happens for these specific types of memories, even during a short nap. If the nap includes a substantial amount of REM sleep (even though it may be less than during a full night), it might indicate that the nap is still an effective consolidation period, at least for certain memory types. We failed to find an effect of REM sleep duration on recovery scores in our nap subjects ($t(27) = 0.43$, $p = 0.67$).

Examining the percentage of REM sleep allows you to understand whether the proportion of the nap spent in REM sleep affects the consolidation process. If a larger portion of the nap is spent in REM, it might indicate that a more efficient or effective consolidation of procedural or emotional memories occurs within that short timeframe. We also failed to find an effect of the percentage of time spent in REM sleep on recovery ($t(27) = 0.21$, $p = 0.84$).

However, not all subjects experienced REM sleep, as a result, we tested whether being a full cycler had any impact on the relationship of REM sleep on recovery. In addition to the overall model failing to reach significance ($F(3, 25) = 1.24$, $p = 0.32$), we failed to find a main effect of REM duration ($t(25) = 1.35$, $p = 0.19$), a main effect of being a Full Cycler ($t(25) = 1.19$, $p = 0.24$), as well as an interaction effect ($t(25) = -1.85$, $p = 0.08$).

5.3.4 Combined Role of Slow Wave Sleep and Rapid Eye Movement Sleep in Memory Consolidation

In order to test hypotheses regarding the combined effects of SWS and REM sleep, or two-process models of sleep dependent memory consolidation (e.g., the Sequential Hypothesis; Giuditta et al., 1995), it is important to differentiate subjects who do cycle through all stages of sleep from those who do not, as we would expect that if SWS lays the groundwork for REM to finish integrating information in neocortex after transfer from hippocampus, that both stages would need to be present in order for the full effects of memory consolidation to be seen. It is important to note that not all subjects spent time in all stages of sleep; 12 subjects did not cycle through all sleep stages during their nap, while 17 subjects cycled through all stages.

A linear regression model was constructed predicting recovery (Posttest3 − Posttest2) from SWS duration, REM duration, and whether they were a "full cycler". The overall model was marginally significant ($F(6, 22) = 2.31$, $p = 0.07$), and SWS duration ($t(22) = 2.42$, $p = 0.02$), REM duration ($t(22) = 2.33$, $p = 0.03$), and completing a full cycle ($t(22) = 3.10$, $p = 0.005$) were all significant predictors of recovery. In other words, (1) SWS duration was positively correlated with recovery, (2) REM duration was positively correlated with recovery, and (3) those who cycle

through all stages of sleep (M = 2.50, SE = 2.18) have better recovery scores than those who do not (M = 2.05, SE = 2.23). See Figures 5.4, 5.5 and 5.6 below.

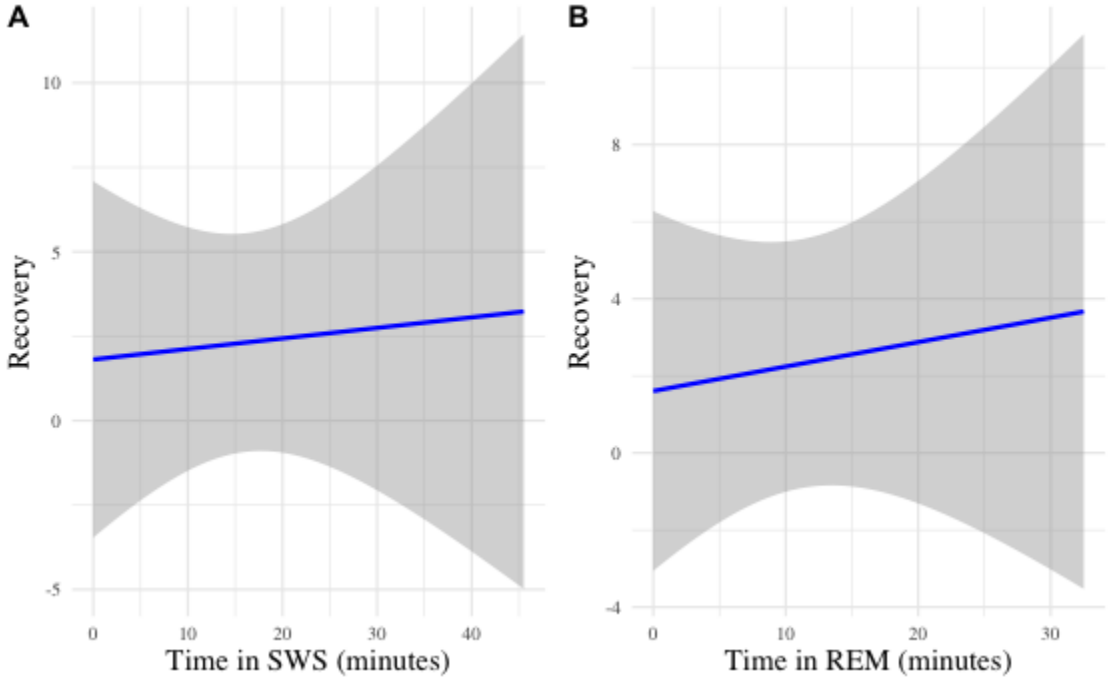

Figure 5.4. Relationship between SWS duration and recovery of perceptual learning performance, and REM duration and recovery of perceptual learning performance, for nap subjects. (A) Relationship of SWS Duration and (B) REM duration to recovery scores for nap participants.

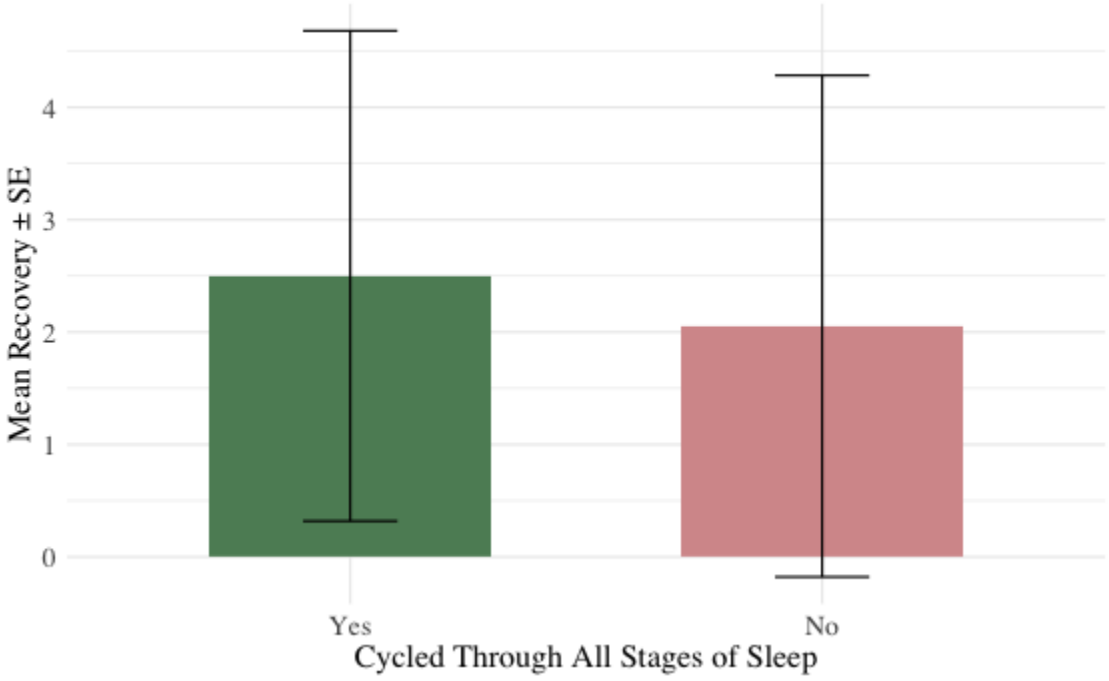

Figure 5.5. Full Sleep Cycle status related to recovery scores for nap subjects. Subjects who cycled through all stages of sleep spent some amount of time in stages N1, N2, SWS, and REM during their afternoon nap ($N = 17$), while those who did not cycle through all stages of sleep spent time in N1 and N2 sleep, but did not spend time in SWS and/or REM sleep. For a full breakdown of the amount of time that each sleep subject spends in each stage of sleep see Figure 5.6.

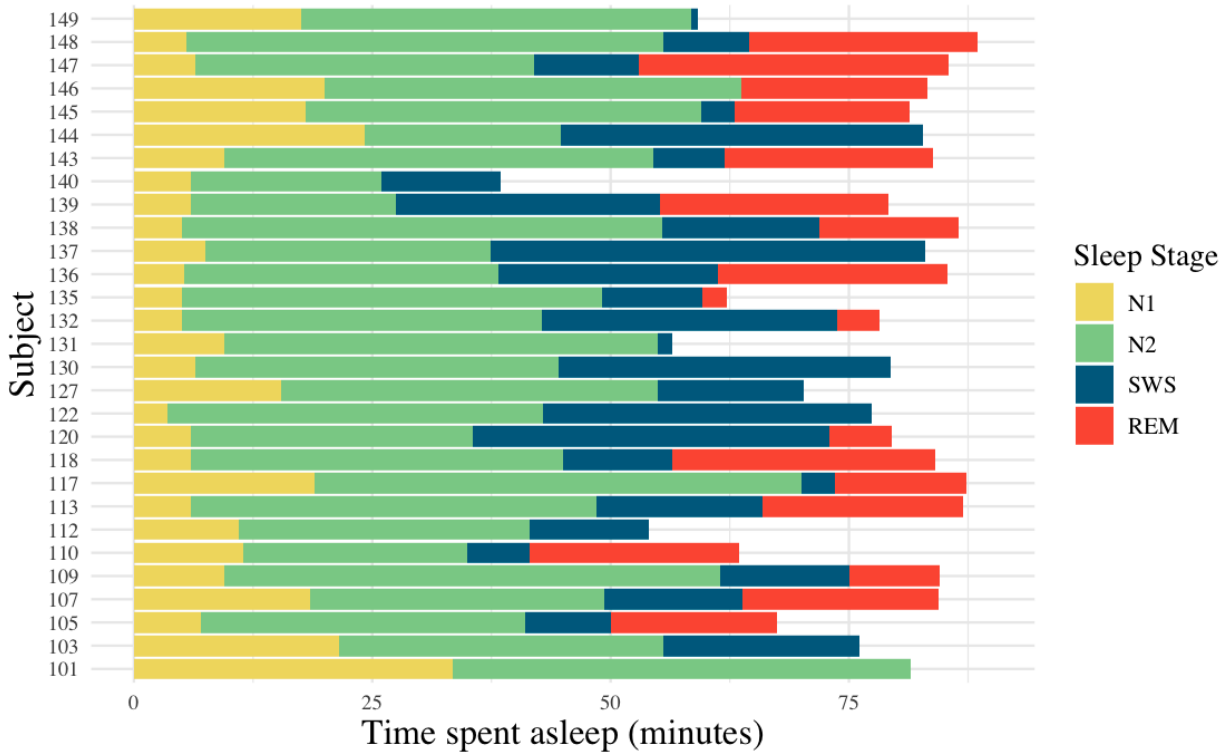

Figure 5.6. A full breakdown of the amount of time that each sleep subject spent in each stage of sleep during their daytime nap.

We did not find a significant interaction between SWS duration and REM duration on recovery ($t(22) = -1.47$, $p = 0.16$). However, we found a significant interaction effect between SWS duration and full cycle status that was negatively associated with recovery ($t(22) = -2.75$, $p = 0.01$). Additionally, we found a significant interaction effect between REM duration and full cycle status that was negatively associated with recovery ($t(22) = -3.02$, $p = 0.006$). See Figures 5.7 and 5.8 below.

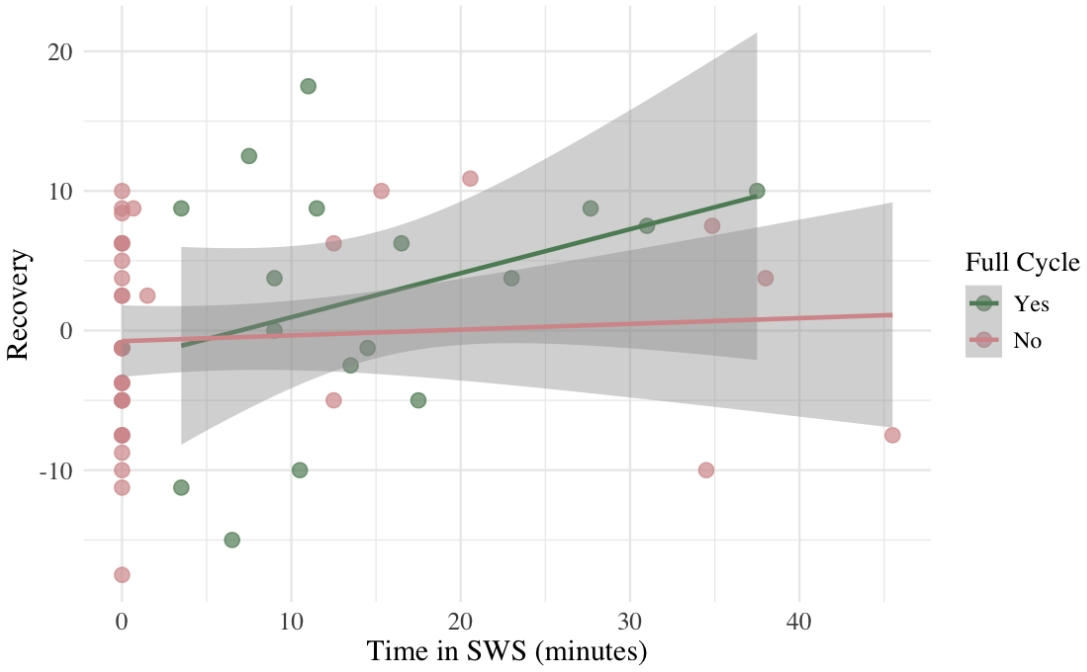

Figure 5.7. The interaction between time spent in slow wave sleep and being a "Full Cycler" on recovery.

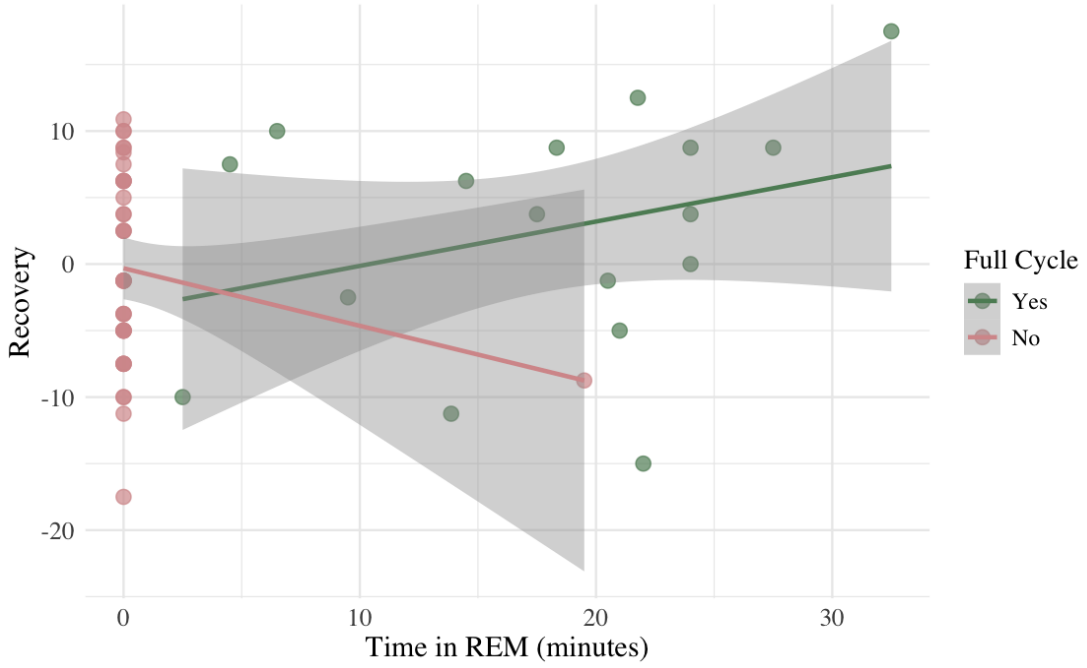

Figure 5.8. The interaction between time spent in rapid eye movement sleep and being a "Full Cycler" on recovery.

5.4 Discussion

The present chapter investigated the neural mechanisms that underlie consolidation of generalized auditory perceptual learning. More specifically, we examined the microstructural and/or macrostructural features of sleep that are critical for the consolidation of this type of learning. Previous research has put emphasis on N2, SWS, and REM sleep stages (McDevitt, Duggan, & Mednick, 2015) or sleep spindles (Antony, Schönauer, Staresina, & Cairney, 2019; Antony & Paller, 2017; Tamminen, Payne, Stickgold, Wamsley, & Gaskell, 2010) as potentially driving consolidation of other types of perceptual or skill based tasks, however, studying consolidation of this type of learning with a daytime nap lets us understand what features of sleep are important in the absence of multiple sleep cycles.

As anticipated, there was no significant difference between our two groups in terms of how their performance changed from Pretest to Posttest1 (our measure of learning) and from Posttest1 to Posttest2 (our measure of loss). This was expected as these blocks occurred prior to our intervention (the sleep/wake interval), and since we were sampling from the same population, we would expect that our subjects would be matched on learning and on loss. In line with previous literature (Heald, Van Hedger, Veillette, Reis, Snyder, & Nusbaum, 2022; Reis, Heald, Uddin, Fenn, & Nusbaum, 2023), we saw significant learning as a function of our training paradigm, and then significant loss after a period of time spent away from the task.

After our intervention, performance changes then bifurcated between our two groups. In other words, the pattern of performance change from Posttest2 to Posttest3 (our measurement of recovery) is significantly different between the two groups; on average, performance improved for the nap subjects, while performance declined for the wake subjects.

Importantly, we see that the initial loss that wake subjects experienced from Posttest1 to Posttest2 (over the first waking retention interval), then continues over the course of the day. There is no offline consolidation period for these subjects that protects them against interference, rather this group on average performs worse from Posttest1 to Posttest2 (over the first waking retention interval), Posttest2 to Posttest3 (over the sleep/wake block), and then Posttest3 to Posttest4 (over the second waking retention interval). On the other hand, in line with previous overnight sleep study experiments (Fenn, Margoliash & Nusbaum, 2013; Fenn, Nusbaum & Margoliash, 2003), sleep allowed subjects to recover performance and then stabilize performance against future decay.

There are various semi-competing theories of which sleep-related neural features may be driving the consolidation process of this type of learning. First, the Dual Process Hypothesis posits that different types of learning rely on different stages of sleep, where procedural and skill-based learning is supported by REM sleep, whereas declarative memories/tasks are supported by SWS. Given that this task trains a perceptual skill, the Dual Process Hypothesis would suggest that we should see a positive correlation between time spent in REM sleep and the amount of recovery that our sleep subjects experienced (Plihal & Born, 1997; Plihal & Born, 1999). However, our results suggest that REM sleep on its own is not sufficient to drive a consolidation effect (through our recovery measure).

While the Dual Process Hypothesis is somewhat rigid in its separation of the role of SWS and REM sleep, other research suggests that SWS is a primary player in procedural/skill-based memory consolidation (i.e., Tononi & Cirelli, 2014). The Synaptic Homeostasis Hypothesis (SHY) reflects on how synaptic strengths may change across periods of wakefulness and sleep — in other words, it suggests there is a bolstering of synapses during initial learning, and then synaptic downscaling, or a removal of unnecessary synaptic connections, that occurs during slow wave

sleep in an effort to improve signal-to-noise ratios (Tononi & Cirelli, 2014). Our results suggest that SWS on its own is not sufficient to produce recovery of generalized auditory perception task performance. However, Tononi & Cirelli (2020) later acknowledge that REM sleep may play a role in fine-tuning neural connections, however, they still hold that SWS is *the* crucial stage for skill learning, as it allows for the refinement and stabilization of synaptic connections.

Tononi & Cirelli's (2014; 2016) work also shows that sleep spindles (which occur during both N2 and SWS) may be linked to procedural memory consolidation. The Complementary Learning Systems (CLS) Model suggests that consolidation occurs via gradual a shift from hippocampal to neocortical storage through the aid of repeated sleep replay events. In this model, spindles that occur during N2 and SWS act as a mechanism of replay, where hippocampal patterns are reactivated and integrated into neocortical networks during these events (McClelland, McNaughton, & O'Reilly, 1995; McClelland, 2013). However, the total number of spindles that appeared during both N2 and SWS were not correlated with performance improvements from Posttest2 to Posttest3.

While the CLS model does not differentiate between sleep spindles that occur during N2 sleep versus sleep spindles that occur during SWS (McClelland, McNaughton, & O'Reilly, 1995; McClelland, 2013), the Active Systems Consolidation (ASC) Model does parse out how spindles that occur during SWS may serve a different function from spindles that occur during N2 sleep. More broadly, ASC, similar to CLS, suggests that memories are first encoded in the hippocampus during wakefulness and later during sleep there is transfer of memory traces from hippocampus to neocortex through the facilitation of sleep spindles. The ASC Model goes a step further to differentiate N2 spindles from SWS spindles, suggesting that N2 spindles are of importance for motor skill and perceptual learning, whereas SWS spindles are more tied to declarative memory

consolidation. However, our results suggest that the number of N2 spindles is not correlated with improvements in task performance from Posttest2 to Posttest3 for our sleep subjects. Similarly to SHY (Tononi & Cirelli, 2014), ASC then goes another step further to suggest that REM sleep may assist in the refinement and stabilization of memories, particularly in procedural memory tasks (Rasch & Born, 2013).

Our results suggest that this recovery of performance for our sleep subjects can be predicted by time spent in SWS as well as REM sleep, but only for those that spend some amount of time in both stages. This is in accordance with two-stage sleep-dependent memory consolidation models which put emphasis on the additive effects of both SWS and REM sleep. In other words, SWS and REM sleep each have a unique and vital role in memory consolidation.

The Sequential Processing Model suggests that SWS plays a role in laying the groundwork for memory consolidation prior to REM later refining, stabilizing, and integrating these memories with existing memory traces. This hypothesis proposes that one needs to have both SWS and REM to occur in sequence — where during SWS relevant synaptic connections are strengthened and then in REM that these connections are finely tuned and integrated in existing networks. Importantly, this model does not necessitate that REM appear immediately after SWS, in other words, it does not suggest consolidation is reliant on the phase transition of SWS to R, but rather the general ordered role of sleep stages in consolidation. It just states that REM must follow SWS at some point in order for consolidation to occur, meaning other stages of sleep, or arousals, can happen in between a bout of SWS and a bout of REM (Giuditta et al., 1995).

Along these lines, since the Sequential Processing Model requires that SWS come before REM at some point, REM cannot come before SWS without another bout of REM afterwards, because SWS plays a prerequisite role in setting up memory traces that are later optimized in REM

(Giuditta et al., 1995). Indeed, our data is in accordance with this requirement. We had three subjects that entered REM prior to SWS during their afternoon nap. One of these subjects had a bout of REM prior to their bout of SWS but then had a second bout of REM afterwards. For this subject, their recovery score was positive (they improved from Posttest2 to Posttest3). On the other hand, for the remaining two subjects who had a bout of REM sleep before SWS but did not later have another bout of REM, their recovery score was negative (meaning that their performance got worse from Posttest2 to Posttest3). While this is not many subjects, this demonstration is in alignment with the idea that SWS needs to provide the scaffolding for REM so that REM can stabilize memories and "finalize" the consolidation process.

While our study provides important insights into the role of sleep in consolidating generalized auditory perceptual learning, several limitations should be acknowledged. First, our sample size, particularly the subset of subjects who experienced REM before SWS, was small. Although their performance trends align with the Sequential Processing Model (Giuditta et al., 1995), a larger sample would be necessary to confirm the robustness of these findings. Additionally, while we examined macrostructural sleep stages (SWS and REM) and sleep spindles, we did not assess other neural features that may contribute to learning consolidation, such as slow oscillations or phase coupling between spindles and slow waves. Future work incorporating high-density EEG or neuroimaging techniques could provide a more detailed mechanistic understanding of how different sleep features interact to support learning recovery. Finally, our study focused specifically on auditory perceptual learning, and it is uncertain whether these findings generalize to other forms of skill acquisition, such as motor learning or visual perceptual tasks. Future research should explore whether the observed sleep-dependent recovery effects extend to different

learning domains and whether the same sequential sleep-stage dependencies hold across modalities.

The findings of this study have several practical implications, particularly in educational and skill-training contexts. These results suggest that incorporating structured nap opportunities into learning environments — such as in music training, language acquisition, or auditory processing therapies — could enhance skill retention and performance recovery. This may be especially relevant for individuals with demanding schedules, such as students, musicians, or individuals undergoing auditory rehabilitation, who may benefit from short sleep episodes rather than relying solely on overnight sleep for memory consolidation. Furthermore, our findings underscore the importance of sleep architecture in learning recovery. Strategies aimed at optimizing sleep quality — such as minimizing disruptions to SWS and REM, improving sleep hygiene, or using targeted interventions to enhance specific sleep stages — could improve learning outcomes.

In sum, our results align with prior research emphasizing the sequential role of SWS and REM in memory consolidation. Notably, previous work by Reis, Heald, Uddin, Fenn, & Nusbaum (2023) demonstrated that a daytime nap provides equivalent benefits to a full night of sleep in terms of learning consolidation. Our findings further support this by showing that even within the limited timeframe of a nap, the sequential occurrence of SWS followed by REM is sufficient to drive performance recovery. This reinforces the idea that the critical processes underlying memory stabilization do not necessarily require an entire night of sleep but can be achieved within a shorter sleep episode, provided that the necessary sleep architecture is present. However, our findings also diverge from some studies that have identified spindles as a primary driver of skill learning (e.g., Antony & Paller, 2017). The lack of a significant relationship between spindle density and

performance recovery suggests that, at least for generalized auditory perceptual learning, spindle activity alone may not be a sufficient indicator of consolidation. Instead, our data support a more holistic model in which both SWS and REM work in conjunction, consistent with sequential models of sleep-dependent memory processing. Further work is needed to determine whether this pattern is task-specific or if it applies broadly across other types of procedural learning.

CHAPTER 6: GENERAL DISCUSSION

6.1 Overview of Findings

This dissertation advances our understanding of how sleep — particularly naps — facilitates the consolidation of generalized auditory perceptual learning. Across two experiments, several critical insights emerged that refine existing models of memory consolidation and challenge traditional assumptions about the role and structure of sleep in learning. While previous research has established that sleep influences rote memory performance, less is known about how it mechanistically supports the abstraction and generalization processes that underlie skill-based learning, particularly in perceptual domains like speech recognition. Notably, Fenn and colleagues (2013) demonstrated that sleep not only restores performance losses incurred during the day — effectively reversing daytime deterioration in perception learning — but it also protects against further losses following subsequent wakefulness. These findings suggest that sleep serves both a restorative and protective function in the consolidation of perceptual learning. This dissertation builds on this research by examining the neural mechanisms that support this process — particularly in the context of shorter sleep opportunities like naps.

Experiment 1 demonstrated that a 90-minute nap can produce consolidation effects comparable to a full night of sleep for generalized auditory perceptual learning (as in Fenn et al., 2013). This is a significant contribution to the understanding of how brief sleep opportunities can influence memory, as it indicates that one does not need to cycle through multiple full sleep cycles in order to produce a consolidation effect. Additionally, given that a 90-minute nap produces effects comparable to a full night's sleep, Experiment 1 data rejects a strict dose-dependent consolidation model (Diekelmann, Wilhelm, & Born, 2009). Instead, the data is more consistent with the idea that specific sleep events (e.g. spindles, slow-wave ripples) or sleep stage transitions

— rather than their duration or number are the critical mechanisms. The results from Experiment 1 also reject the notion that consolidation effects are gated by circadian rhythms as they can occur outside of the typical nocturnal sleep period (Xia & Storm, 2017). This indicates that the timing of sleep is less critical than the presence of specific sleep stages or neural events (e.g. spindles, slow-wave ripples) in driving consolidation.

While the results of Experiment 1 advance our understanding of the conditions under which sleep-dependent consolidations can be achieved — demonstrating that even brief sleep opportunities can support generalized auditory perceptual learning — a key limitation of this experiment was the absence of an objective sleep measure, such as polysomnography. Because sleep was assessed via self-report, the specific physiological characteristics of the nap — such as sleep stage composition, the presence or density of spindles or the timing of slow-wave activity, remain unknown. This limits our ability to pinpoint the mechanisms most responsible for the observed consolidation effects. This is particularly important given that consolidation was not homogeneous across participants, as some participants showed stronger post-nap gains than others. Such variability suggests that certain neural features of sleep may be more critical than others in giving learning-related changes. Further, given that different sleep stages and associated neural events have been linked to distinct roles in memory processing (Diekelmann & Born, 2010), it remains an open and important question as to what specific features of sleep are necessary or sufficient for the consolidation of generalized auditory perceptual learning under these brief sleep conditions.

Experiment 2 addressed this limitation directly. It incorporated both self-reported and objective measures of sleep (PSG) to validate the occurrence and nature of the nap. The addition of the 14:30 test (Posttest2) allowed for a more precise quantification of loss during wakefulness

and recovery following sleep. As reflected in Chapter 3, not only did this experiment replicate the findings of Experiment 1, but it also confirmed that wearing an EEG net and peripheral electrodes did not interfere with the ability to consolidate learning. Importantly, it revealed clear sleep dependent restoration of daytime loss, indicating that sleep consolidation occurred.

Building on the variability in consolidation observed in Experiment 1 — where not all participants benefited equally from the nap — Experiment 2 examined whether individual differences in sleep history and sleep architecture could account for this variance. Rather than examining consolidation outcomes directly in Experiment 2, the focus shifted to slow wave sleep (SWS) as a candidate mechanism underlying the variability observed in consolidation performance. As such, effort was taken to identify the factors that predict SWS expression during the nap. Chapter 4 investigated whether there was evidence that sleep restriction influenced the sleep architecture observed in daytime naps. Previous work has shown that those who are more sleep restricted demonstrate more slow wave sleep ("recovery sleep") during their next sleep opportunity. In this study, we demonstrated that neither the amount of sleep obtained the night before Visit 2 (Night Before TST) nor the average total sleep time across the two nights leading up to Visit 2 (Average TST) predicted the amount of slow wave sleep seen during the daytime nap. The amount of immediate sleep restriction that subjects were under (Immediate Restriction) also did not predict the amount of slow wave sleep seen during the daytime nap. In contrast, Cumulative Restriction was a significant predictor of SWS duration and percentage of SWS. This finding is in line with research that suggests that individuals who are in a sleep restrictive state will engage in "rebound SWS" during their next sleep opportunity (Borbély & Achermann, 1999). This also outlines that in order to accommodate the amount of rebound SWS, that the amount of time spent in N1 and N2 sleep will thus become truncated (Ferrara & De Gennaro, 2001). Indeed, this is what

we see in our study, where those who experienced more immediate sleep restriction experienced significantly less N1 sleep, and subjects who experienced more cumulative sleep restriction experienced marginally less N2 sleep.

The differentiation between Immediate and Cumulative Restriction here is also of interest, as these results suggest that N1 may be more labile, while N2 and SWS changes may only be seen after some amount of sleep debt is accrued. In other words, these results suggest a differential sensitivity or hierarchical reactivity of NREM stages to sleep pressure, where lighter sleep stages are impacted earlier and deeper NREM stages show delayed or cumulative effects.

The reduction in N1 after immediate sleep restriction likely reflects a homeostatic prioritization of deeper sleep stages, particularly SWS, as N1 duration tends to decrease under high sleep pressure as the brain rapidly progresses into deeper sleep stages to begin restorative processes more efficiently (Carskadon & Dement, 2005). N1 has been characterized as a labile, transitional stage between wakefulness and stable sleep (Silber et al., 2007), and its flexibility may explain its immediate reduction under acute deprivation. Thus, N1 may act as a flexible "buffer" stage that is easily sacrificed when sleep is time-limited, or recovery is more urgent.

In contrast, a decrease in N2 duration and increase in SWS duration following multiple days of sleep restriction may reflect a more complex or delayed homeostatic adaptation. N2 sleep, while not as deep as SWS, plays a critical role in memory consolidation (De Gennaro & Ferrara, 2003). Some evidence suggests that N2 is more stable in the short term but may decrease as SWS demand increases with accumulated sleep debt (Van Dongen et al., 2003), as SWS is considered the most critical stage for physiological and cognitive recovery (Borbély & Achermann, 1999). This could explain why N2 reductions were more apparent after multiple nights of curtailed sleep:

as sleep pressure builds, the system reallocates time from N2 to SWS, maintaining the latter's restorative priority.

Together, these results support the idea that sleep architecture rebalances in a stage-specific and temporally dynamic way, with N1 acting as an immediately labile stage, and N2 showing compensatory shifts only after sustained sleep loss. These findings extend previous work on sleep stage reallocation under homeostatic pressure and underscore the need to consider both acute and cumulative sleep history when interpreting changes in sleep architecture.

While sleep restriction measures were not found to directly predict the amount of recovery seen immediately after waking (Posttest3 − Posttest 2), we did find that those who woke up out of SWS had a significantly higher recovery score (more consolidation) than those who woke up out of other stages of sleep. Prior research suggests that individuals who wake up immediately out of SWS should be experiencing more marked cognitive impairments due to sleep inertia (Tassi & Muzet, 2000), however, our results are not in accordance with this. We predicted that it could be that the restorative benefits of SWS outweighed the transient deficits associated with inertia (i.e. through significantly reducing homeostatic sleep pressure), at least in the context of this perceptual learning task, or that for this sample that 30 minutes to recover from the effects of sleep inertia was sufficient.

Finally, Chapter 5 addressed what microstructural/macrostructural features of sleep are critical for the consolidation of generalized auditory perceptual learning. The results of this chapter provided results in accordance with models of memory consolidation that delineate the separate, but additive effects of SWS and REM sleep on consolidation. For example, we review the Sequential Processing Model which posits that first SWS further strengthens relevant synaptic connections, and then later REM fine tunes and integrates these connections in existing neural

networks (Giuditta et al., 1995). In Experiment 2, we found that both the time spent in SWS and REM sleep positively impacted recovery, but only in the presence of the other stage during the bout of sleep. Additionally, while the sample size is limited in the number of subjects who cycled through REM prior to SWS did not improve from Posttest2 to Posttest3 (they had a negative recovery score). These findings support the idea that memory consolidation depends not only on the presence but also the sequence of sleep stages.

The results of Experiment 2 challenge models of sleep consolidation that emphasize the universal importance of spindles across memory domains. In particular, spindle-based models like the Complementary learning Systems (CLS) model and the Synaptic Homeostasis Hypothesis assume spindles either reflect or actively facilitate consolidation through reactivation and system-level transfer. However, our data suggest that spindles may not play a central role in consolidating generalized perceptual learning, at least during brief naps.

This raises the question: are spindles more relevant to episodic or declarative memory consolidation than to perceptual or generalized learning? Much of the work implicating spindles focuses on hippocampus-dependent tasks (e.g., word pairs, spatial navigation; Schabus et al., 2004, Fogel, Nader, Cote & Smith, 2007), where spindles during SWS are thought to promote system-level memory transformation.

Further, there are fewer studies which examine spindles in the context of perceptual learning or generalization, and the findings are more mixed. For example, Mednick et al. (2003) demonstrated that spending some amount of time in both SWS and REM during a daytime nap was critical to visual perceptual learning performance (a texture discrimination task), however, this study did not implicate spindles directly. As such, generalized perceptual learning may rely

more heavily on cortical reorganization, attentional tuning, or long-term changes in sensory representations — processes that might not require spindle-related reactivation.

Notably, our behavioral findings mirror those of procedural learning studies (e.g., motor sequence tasks), which sometimes implicate N2 or REM sleep (e.g., Walker, Brakefield, Morgan, Hobson, & Stickgold, 2002) — but not always spindles — as important for consolidation. Our work adds to this by showing that generalization itself — a core feature of skill learning — can be consolidated by sleep, but without clear electrophysiological correlates like spindle density or power. This suggests that generalized learning may reflect a distinct consolidation pathway that is sleep-dependent but spindle-independent.

Many prior studies implicating spindles used rote or episodic tasks (e.g., memory for word pairs, spatial maps, cued reactivation paradigms). Few, if any, directly address generalized procedural learning using non-repetitive stimuli. To our knowledge, no study demonstrating a spindle-behavior relationship did so with a generalization task devoid of item repetition, like the synthetic speech paradigm used here. This methodological distinction is critical, as in our task, participants cannot rely on specific item memory; instead, they must extract abstract patterns from complex distorted speech. The absence of a spindle x learning relationship in this context suggests that not all memory consolidation benefits from sleep rely on spindles, and their relevance may be domain- and/or task-specific.

6.2 Future Directions and Concluding Remarks

Based on the findings in Chapter 4, future work should further explore how individual differences in sleep history and sleep pressure influence the consolidation of generalized perceptual learning. One avenue involves understanding if chronic versus acute sleep deprivation

differentially impacts consolidation of generalized learning during a daytime nap by tracking sleep history past just 2 days. In fact, Van Dongen, Maislin, Mullington, & Dinges (2003) demonstrated that cumulative sleep debt over 7 − 14 days of restricted sleep (e.g., 4 − 6 hours/night) leads to progressive declines in cognitive performance, even when participants report feeling "used to it". Additionally, and importantly, performance did not rebound with just one night of recovery sleep. These results suggest that longer-term sleep history impacts both baseline cognitive function and the capacity for recovery, and that future studies should collect sleep history well beyond the 2-day mark.

Additionally, to establish causal relationships, studies could experimentally manipulate sleep pressure (e.g., through partial deprivation, extended wakefulness, or irregular sleep patterns, in both habitual and non-habitual nappers) to causally test its effect on nap-based consolidation outcomes.

In these studies, we also did not collect information about our subjects' nap history. In other words, we did not collect information about whether our subjects were habitual nappers. There is research to suggest that individuals who are habitual nappers demonstrate shorter sleep onset latencies, greater sleep efficiency, and altered stage distributions during naps — including more REM and N2 sleep, especially in longer naps (Milner & Cote, 2009). Importantly, there is research that has demonstrated that habitual nappers exhibit faster cognitive recovery after naps than non-habitual nappers, and this difference remains even when groups were well-rested, suggesting reduced vulnerability to sleep inertia among habitual nappers (Tietzel & Lack, 2001).

Additionally, research has suggested that habitual nappers with short nocturnal sleep often display greater SWS and REM during naps. In other words, naps in these individuals may be compensatory and structurally adapted to maximize recovery — showing faster sleep onset, more

SWS, and fewer lighter stages (N1 and N2). Importantly, this adaptation may blunt cognitive impairments and reduce sleep inertia (Faraut, Andrillon, Vecchierini, & Leger, 2015).

This previous literature (Tietzel & Lack, 2001; Faraut, Andrillon, Vecchierini, & Leger, 2015) also gives a potential explanation for our observation that individuals who wake directly from slow-wave sleep (SWS) did not appear to impair post-nap performance. This attenuation may reflect an adaptive mechanism from repeated exposure to waking up mid-sleep, which improves arousal regulation and post-nap performance.

It could be that some of the variation in post-nap recovery in our nappers could be explained by a subset of our subjects being nap-familiar while some not being habitual nappers. Future research should make sure to survey sleep history more extensively to include nap history (nap frequency, length, and quality) in order to investigate whether performance is differentially impacted by nap history for this generalized perceptual learning task.

It is also important to recognize that there has been no research to date to explicitly test how sleep inertia affects performance on generalized auditory perceptual learning performance. Most existing studies focus on the benefits of sleep consolidation in such tasks, rather than on performance immediately upon awakening.

There is a wealth of evidence to suggest that sleep inertia affects different cognitive tasks in different ways, depending on (1) the cognitive domain involved (e.g., attention, memory, etc.), (2) the complexity of the task, and (3) whether the task relies on recently consolidated memories or real-time processing. For example, tasks that rely on sustained attention and reaction time — such as the Psychomotor Vigilance Task — show particularly strong impairments in the first few minutes following awakening (Jewett et al., 1999), while more complex executive functions like

planning or flexible reasoning may remain disrupted even after basic alertness is restored (Horne & Moseley, 2011; Silva & Duffy, 2008; Tassi & Muzet, 2000).

In the visual domain, Mednick et al. (2003) found that participants tested immediately after a nap did not exhibit performance gains in a texture discrimination task, whereas those tested after a 60-minute delay did. This suggests that sleep inertia can mask the benefits of sleep-dependent learning, even in low-level perceptual tasks.

Given that generalized perceptual learning tasks often require higher-order cognitive processes, such as flexible application of learned representations, it is plausible that sleep inertia could have a more pronounced effect on performance in these tasks. However, empirical evidence specifically addressing this is currently lacking.

This raises important questions about the time course of sleep inertia, its relationship to memory stabilization, and how it interacts with nap history. Future research should include high-temporal-resolution testing after naps, with measurements taken at multiple intervals (e.g., 5, 15, 30, and 60 minutes post-nap) to chart the trajectory of recovery for generalized auditory perceptual learning. Including subjective measures of alertness as well as nap-familiarity could help assess individual differences in sleep inertia severity.

Also, circadian factors should be considered (Xia & Storm, 2017). In the current study, participants napped at 3:00 PM, which falls within the post-lunch circadian dip in alertness — a period generally conducive to sleep. However, the specific timing of a nap (e.g., early vs. late afternoon) can interact with an individual's overnight sleep history, nap history, and internal circadian clock (based on habitual sleep-wake timing) in ways that influence both sleep architecture and the efficacy of consolidation.

Prior research demonstrates that circadian phase modulates sleep propensity, architecture, and inertia, such that naps taken earlier in the afternoon (e.g., ~13:00–14:00) tend to include more SWS, while naps taken later (e.g., ~15:00 or beyond) often result in an increased proportion of REM sleep and a reduced proportion of SWS (Dijk & Czeisler, 1995; Hayashi et al., 1999; Campbell et al., 2005). Given that Chapter 5 demonstrated that the presence of both REM and SWS is critical for the consolidation of generalized perceptual learning, having a nap at 15:00 seems most advantageous to consolidation.

Moreover, both recent sleep history (e.g., how much sleep one has had over the past several nights) and nap history (e.g., frequency of daytime napping) influence the homeostatic pressure for SWS and REM during a nap. Individuals who are chronically sleep-restricted or who frequently nap may show altered homeostatic–circadian interactions, which can shift the relative balance or latency of NREM and REM stages during a nap (Monk et al., 1997; Campbell & Murphy, 2007). In the context of a 15:00 nap, this could mean that sleep-deprived individuals show more SWS "rebound" and truncated lighter stages (e.g., N1 or N2), while well-rested individuals or habitual nappers may exhibit a more REM-heavy nap, again, potentially altering the type of learning benefits gained.

Finally, cognitive performance upon waking is also shaped by circadian phase and can be further modulated by nap timing. Although early afternoon naps often coincide with peak sleep propensity, they may also heighten the risk of sleep inertia, especially for non-habitual nappers or individuals waking from SWS (Achermann et al., 1995; Tassi & Muzet, 2000). While a 30-minute delay between waking and testing — as used in the current study — is generally sufficient to allow for partial cognitive recovery, particularly for procedural tasks, the degree of sleep inertia may still vary depending on sleep stage upon awakening and individual nap characteristics. Taken together,

these findings underscore the importance of accounting for both circadian timing and prior sleep/napping history when interpreting nap-based consolidation effects.

Building on the results of Chapter 5, future studies should systematically manipulate the presence or absence of SWS and REM sleep using targeted protocols (such as in Bellesi, Riedner, Garcia-Molina, Cirelli, & Tononi, 2014). This could include waking participants before REM onset or delaying SWS onset using environmental or auditory stimulation. Such methods would help clarify whether both stages are necessary for consolidation, or if one can compensate for the other. Additionally, researchers may explore whether pharmacological or non-invasive stimulation techniques — such as acoustic stimulation to boost slow waves, or transcranial stimulation to enhance spindle density — can synergistically enhance memory consolidation, particularly when both SWS and REM are enhanced in tandem.

Given that consolidation efficacy varied widely across participants, future work should aim to design adaptive sleep interventions that account for individual sleep history. For instance, nap duration or structure could be adjusted for those who are sleep-restricted or experience chronic short sleep. Exploring non-invasive stimulation techniques during naps could enhance consolidation-relevant features like spindles or SWS. These approaches may be particularly beneficial for populations with atypical sleep architecture, such as shift workers, older adults, or individuals with insomnia, who often do not experience robust or regular sleep cycles.

Finally, an important direction for future research is to assess the generalizability of these findings beyond generalized auditory perceptual learning. It remains unclear whether the same sleep-based consolidation effects extend to tasks involving visual processing, motor learning, or semantic memory. Studies should compare nap-based effects across different modalities to determine whether the mechanism uncovered here is specific to auditory generalization or applies

more broadly. Furthermore, it would be valuable to assess whether generalized learning tasks across domains benefit uniquely from a combination of SWS and REM sleep, or whether this additive benefit is a general principle of memory stabilization for generalized perceptual learning.

In light of the findings from Experiment 1 and 2, we propose a hybrid view in which multiple mechanisms of sleep-dependent consolidation operate in parallel but are selectively recruited depending on task demands. We see that for generalized auditory perceptual learning that: (1) Short naps can support robust consolidation, even without evidence of multiple sleep cycles or high spindle activity. (2) Sleep architecture is modulated by prior sleep history. (3) Sleep inertia may not universally impair cognition, and the timing of recovery (e.g., delayed effects after napping) suggests that consolidation may unfold gradually and possibly continue into post-sleep waking periods. (4) The microstructural features of sleep influence performance on our generalized perceptual learning task, and that spindle-independent consolidation may exist for procedural or generalized learning. For hippocampal, episodic, or inferential learning, replay and spindles may dominate. For perceptual generalization, other processes — perhaps involving cortical plasticity and attentional modulation — may be more relevant.

These results carry broad implications for our understanding of memory, the design of cognitive interventions, and public health. They suggest that even brief, strategically timed naps may benefit learning, particularly for individuals who are sleep restricted. Furthermore, the nuanced relationships between sleep stages and learning outcomes underscore the need for precision in sleep-based interventions, whether behavioral, pharmacological, or neuromodulatory.

These findings collectively demonstrate that even a brief nap — consisting of a single sleep cycle — can support the consolidation of generalized auditory perceptual learning. This challenges long-held assumptions about the required duration and architecture of sleep for memory

consolidation. Moreover, it highlights the complex interplay between sleep stages, individual sleep history, and task structure. By isolating the behavioral and physiological mechanisms that contribute to consolidation, this work provides a strong foundation for future research aimed at understanding how sleep dynamically supports performance in the context of auditory perceptual learning.

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Appendix A: Demographic Questionnaire

How old are you? _____ years

Are you: ☐Male ☐Female ☐Other

Are you: ☐Left-handed ☐Right-handed

Are you a student? ☐Yes ☐No

If yes, what year?

What is your major/ what department are you in?

Appendix B: Language Experience Questionnaire

Age: _____

Gender: _____

Primary Language: _____

1. Do you speak any secondary languages? YES NO

If so, please list them below, along with your level of proficiency for each:

_____ Basic Proficient Semi-Fluent Fluent

_____ Basic Proficient Semi-Fluent Fluent

_____ Basic Proficient Semi-Fluent Fluent

_____ Basic Proficient Semi-Fluent Fluent

2. In what country were you born? _____

3. Please list the different areas in which you have lived, including the country, region/state, and number of years. Please write your answers chronologically (earliest to most recent).

_____ _____

_____ _____

_____ _____

_____ _____

_____ _____

_____ _____

(City, State/Province/Region, Country) (Number of years)

Appendix C: Sleep Questionnaire

1. What time do you usually go to sleep?
2. Do you fall asleep easily?
3. How deeply do you sleep (Light, Medium, Deep)?
4. What time do you usually wake up?
5. Do you usually feel well rested?
6. Have you ever sought medical attention for a sleep disorder?
7. Do you have any disabilities that disrupt your sleep?
8. Are you currently taking any medications to help you sleep?
9. Are you taking any other medications? (excluding oral contraceptives)
10. Do you have a history of substance abuse or diagnosed major mental illness?
11. How many caffeinated beverages do you drink each day?

Appendix D: Sleep Log

Evening of: _____

1. Time you went to bed: _____
2. Approximately how long did it take you to fall asleep? _____
3. Time that you got out of bed in the morning: _____
4. Did you feel well-rested when you awoke? Yes No
5. Approximately how many times did you awake last night? _____
    a. What was the total amount of time that you were awake (approximately)? _____
6. What is the total amount of time that you slept? _____

Evening of: _____

1. Time you went to bed: _____
2. Approximately how long did it take you to fall asleep? _____
3. Time that you got out of bed in the morning: _____
4. Did you feel well-rested when you awoke? Yes No
5. Approximately how many times did you awake last night? _____
    a. What was the total amount of time that you were awake (approximately)? _____
6. What is the total amount of time that you slept? _____